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ASSESSING THE IMPACT OF CONTROL INTERVENTIONS AND AWARENESS ON MALARIA: A MATHEMATICAL MODELING APPROACH

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Abstract. Malaria is a vector-borne contagious disease which remains a public health burden for decades. It is highly endemic in Sub-Saharan African, and an estimated number of two hundred and twenty-eight million cases was reported in 2018 around the world. We develop and examine a deterministic model which describes the transmission dynamics of malaria between mosquito and human populations and examine the impacts of control interventions with their level of awareness on its control. The malaria-free equilibrium of the model is shown to be locally asymptotically stable if the threshold quantity $\mathcal{R}_0 < 1$. We study the stability of the endemic equilibrium and the conditions for the existence of backward bifurcation are presented. A sensitivity analysis was done to measure the outcome of the control intervention parameters on the reproduction number. The result shows that residual spray and bed-net usage are the most important parameter on the reproduction number. A numerical simulation was carried out and the result shows that combining bed-net usage and residual spray will reduce the burden of malaria faster. Particularly, results suggest that awareness and proportion of bed-net usage and residual spray should be priorities and increased to at least 75% for the possibilities of eliminating malaria.

Keywords: malaria; residual spray; bed-net usage; treatment; awareness; sensitivity analysis.

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1. INTRODUCTION

Among the deadliest diseases with a highly challenging health burden in the tropical regions is malaria. It is a vector-borne transmissible disease that is highly endemic in Sub-Saharan Africa, especially in improvised and low hygienic environments [1, 2, 3]. Despite continuous research about malaria for the past decades, it remains a major public health burden for which it was declared endemic in one hundred and nine countries in 2008 [4]. An estimated number of two hundred and twenty-eight million cases were reported in 2018 around the world. As stated in the world malaria report released by the World Health Organization (WHO) in 2019, about four hundred and five thousand deaths were recorded [2, 5]. Approximately three hundred to five hundred million cases occur globally annually, with over one million deaths yearly. The burden of malaria is tremendous in the Sub-Saharan African region such that eighty percent of these cases and ninety percent of these deaths occur in this region [6, 7, 8]. About 78% of deaths occurs in children below age five [1]. Malaria is caused by an infection with the protozoan parasite of genus *Plasmodium*. In humans, five different species of *Plasmodium* can cause infection, namely *Plasmodium falciparum*, *Plasmodium malariae*, *Plasmodium ovale*, *Plasmodium vivax*, and *Plasmodium knowlesi* [1, 9, 10]. It is transferred to humans through the bite of an infected female *Anopheles* mosquito [1]. After an effective bite, the parasite multiplies in the human liver and bloodstream to develop into an infectious form. After the incubation period of the disease (which is within 9-14 days), human begins to show symptoms. The symptoms characterized by malaria include; rise in body temperature, headache, cold, shivering, pain, anemia, fatigue, and vomiting among other symptoms [2, 11]. To present, there is no effective vaccine against malaria. However, it is preventable and curable. Treatments such as the use of anti-malaria drugs have been in use for decades. Though, some existing anti-malaria medications are losing their effectiveness as a result of the drug resistance evolved in the parasite [2, 12]. Some control techniques have been employed to prevent the infection of malaria in the human population, such as insecticide-treated mosquito nets (ITNs), indoor residual spraying (IRS) and bed-nets use [4, 11]. The occurrence of malaria has been increasing lately as a result of parasite drug resistance and mosquito insecticide resistance [9, 13], thus causing the disease to remain endemic in many regions.

Many researchers have employed mathematical models from different fields to investigate the spread of infectious diseases in a given population using diverse approaches (see [14, 15, 16, 17, 18, 19, 20]). Modeling of malaria has helped in understanding the transmission dynamics of this disease, including appropriate control strategies to mitigate it. Sir Ross published the first model in 1911 to demonstrate the development of malaria [2, 21]. Over the years, this model has been used in the past due to its simple nature. However, as the burden of malaria increases over the decades, numerous researchers have modified the existing model of Sir Ross, and have developed models by introducing different factors, parameters, and variables to further understand its dynamics spread in the population. For example, the model by Sir Ross's was improved by incorporating; the latent period of infection [2, 22]; the heterogeneity of human and mosquito [23, 24]; immunity factor [2, 25, 26]; susceptibility to malaria in the host population [27, 28]; a model with exposed human and exposed mosquito [12, 29] and recovered human [23, 30], among many other study. Furthermore, some modeler has developed models that integrate the effect of climate change (such as temperature and rainfall), and seasonality [31, 32, 33, 34, 35]. In addition, some researchers have investigate the transmission dynamics of malaria within-host level [36, 37, 38, 39, 40]. A mathematical model has been utilized in the decision-making process of intervention programs for the prevention and control of malaria in the populace. Thus, a huge number of researchers have employed mathematical models to predict effective control of malaria using optimal control theory. These studies assess the optimal interventions strategies required for effective control of malaria. Examples of these studies includes [11, 41, 42, 43, 44, 45]. Although numerous researches have been performed on the spread and control of malaria, however, it remains a health burden in some regions, especially Sub-Saharan Africa. Thus, continuous efforts must be encouraged in modeling the dynamics of this disease and its control in the endemic regions. Among many models that have been developed, we discuss the methods, results, and limitation of few studies that stands as the foundation to the model proposed in this work.

In [30], the authors proposed a five compartmental deterministic model to describe the transmission dynamics of malaria between the mosquito and human populations. The model allows the transmission from the recovered humans due to incomplete immunity to reinfection. In this

study, the authors employ both the standard incidence and the mass action incidence malaria model to evaluate the effect of incomplete immunity to reinfection in the spread of the disease in the human population. The result from this study shows that the standard incidence model shows the phenomenon of backward bifurcation as a result of the reinfection of individuals who recovered from malaria. Furthermore, the result shows that this phenomenon can be eliminated by using the mass action incidence instead of the standard incidence function. Thus, the global dynamics of malaria disease with reinfection is determined by the threshold quantity reproduction number. In addition, numerical simulations result suggests that increasing the rate of incomplete protection of recovered humans and decreasing the life expectancy of mosquitoes, will increase the region of backward bifurcation. It must be noted that the model developed and analyzed in [30] did not consider the exposed humans and the aquatic stage of the mosquito population. Also, it is not always true that a recovered individual must be re-infected by an infected mosquito before progressing to the infected human population. Immune human individuals progress to the susceptible population following the loss of their immunity [11, 29].

A study on the effect of bed-net usage on malaria commonness is presented in [46]. The authors formulated and analyzed the basic susceptible – infectious (SI) model, consisting of human and mosquito populations, to examine the effect of bed-net use on the spread of malaria infection in the population. The model incorporates the effect of human behavior such as the lack of effective usage of bed-net, on the spread of the disease. Results from the model analysis show the existence of backward bifurcation. This implies that reducing the reproduction number only is not sufficient in eliminating the disease, except when the initial cases of malaria infection in both populations are insignificant. Furthermore, results illustrate that bed-net usage decreases the reproduction number. Specifically, results reveal that if seventy-five percent of the human population effectively uses the bed-net, then malaria may be eradicated. The limitation of this model includes the absence of the exposed individuals who can transfer the malaria infection to mosquitoes when they come for their blood meal. Also, since the presence of backward bifurcation nullifies the guarantee that reduction of the threshold quantity below unity will eliminate the disease, it is important to encourage additional strategies like indoor residual spraying, and early treatment of infected individuals to lessen the burden of malaria in

the population. In addition, a model that accounts for the control of immature mosquitoes in the aquatic stage will help in studying the effective control of malaria in the population.

2. MODEL FORMULATION

Motivated by the model presented in [30, 41, 46], we present a deterministic model to investigate the impact of awareness about preventive measures and treatment care on the spread of malaria in a population. Malaria is a disease that is preventable, treatable, and curable in the human population. Among many other preventative measures against malaria is the use of insecticides treated nets (henceforth refer to as bed-nets); use of residual spray; intermittent preventive treatment; and use of repellent that contains diethyltoluamide [41, 47]. However, if an individual is infected with malaria, the use of anti-malaria medications has been shown to regulate malaria in humans [48]. Sadly, the incidence of malaria is increasing due to drug and mosquito insecticide resistance in regions where malaria is endemic [9, 13]. Preventive and treatment healthcare has shown to lessen the burden of malaria in the endemic regions [48, 49], thus increasing the chance of eradicating malaria in these regions, it is important to increase the awareness or educational campaign about prevention and treatment strategies against this disease. Individuals' awareness about malaria and its mode of transmission will allow the human population to take precautionary measures such as personal prevention against mosquito bites, and control of mosquito population. As a result of this awareness, it is expected that the human population embraces prevention and treatment of malaria, thus leading to a decrease in the cases of malaria. In this study, we incorporate a saturated function for the level of awareness on bed-nets usage, residual spray, and treatment of infected humans in the model to study the outcome of preventive and treatment measures enhanced by awareness on the dynamic transmission of malaria among humans. We denote the awareness about the use of bed-nets as A_1 , awareness about residual spray as A_2 and awareness about the mode of treatment for malaria disease as A_3 , such that $\{A_1, A_2, A_3\} \in A$.

Transmission of malaria can only occur between two interacting hosts namely human and mosquito, thus we group the interacting hosts into human and mosquito populations. The human population is further sub-group into susceptible S_h , exposed E_h , infectious I_h , and recovered R_h ,

based on their disease status. There are mainly four stages in the development of mosquitoes namely; egg, larva, pupa, and adult stage. However, for simplicity purposes, we classify the mosquito population into the immature and mature stage, such that (egg, larva, and pupa) are classified as immature mosquito population denoted by M_m . Furthermore, we sub-divide the mature mosquito population into susceptible S_m , exposed E_m , and infectious I_m . Hence, the total human and mosquito populations at time $t \geq 0$ are given as $N_h(t) = S_h + E_h + I_h + R_h$, and $N_m(t) = M_m + S_m + E_m + I_m$ respectively. The susceptible human population is produced through birth or immigration at the recruitment rate π_h , followed by the loss of immunity of recovered humans at a rate ω . All human populations are reduced by natural mortality with a constant rate μ_h . The susceptible human population is more reduced by the force of infection rate $\lambda_h(A_1 + A_2)$ (defined in 4), following an effective bite by an infected mosquito, thus susceptible humans moved to the exposed human population after infection. The exposed human population decreased by the progression rate of exposed individuals to the infectious population at a rate σ_h . The infectious population is generated by the progression of exposed humans to their infectious state at a rate σ_h and is reduced by disease-induced death (death caused by malaria) at a rate δ_h , and recovery of infectious individuals at a rate $\tau_h(A_3)$. The recovery rate of infectious humans is model as a function of awareness such that

$$(1) \quad \tau_h(A_3) \equiv \tau_h(q) = \tau_h + \frac{\tau_{max}qA_3}{1 + A_3}, \quad 0 \leq q \leq 1, \quad 0 \leq A_3 \leq 1$$

where the recovery rate of individuals is denoted by τ_h , and $\tau_{max}q$ is the recovery rate due to awareness. Lastly, on the human population, the recovered human population is produced by the recovery rate of infectious humans. The recovered human population is depopulated as a result of the loss of immunity at a rate ω . The mosquito population is grouped into four sub-populations namely immature, susceptible, exposed, and infectious mosquito populations. The immature mosquito population is generated by mosquito egg deposition at a rate $\pi_m(A_2)$. This population is reduced due to the development of immature mosquitoes at a rate ϕ and mosquito death at rate $\mu_m(A_2)$. We model the egg deposition rate as a function of awareness such that

$$(2) \quad \pi_m(A_2) \equiv \pi_m(p) = \pi_{max} - \frac{(\pi_{max} - \pi_{min})pA_2}{1 + A_2}, \quad 0 \leq p \leq 1, \quad 0 \leq A_2 \leq 1$$

where π_{max} and π_{min} are the maximum and minimum egg deposition rates of mosquitoes. Residual spraying is expected to reduce the recruitment of mosquitoes to its minimum rate such that if $p = 0$, the egg deposition rate of mosquito will remain in its maximum value π_{max} . The susceptible mosquito population is created by the maturation rate of immature mosquitoes. All the mature mosquitoes (susceptible, exposed, and infectious) are reduced by mosquito mortality with a rate $\mu_m(A_1 + A_2)$. After effective contact with an infected human, the susceptible mosquito population is further reduced by the force of infection rate $\lambda_m(A_1 + A_2)$ (defined in 4), and thus move to the exposed mosquito population. This population is decreased by the movement rate of exposed mosquitoes to the infectious population at a rate σ_m . The infectious mosquito population is generated by the progression of exposed mosquitoes to their infectious state at a rate σ_m . The awareness compartment A is populated by a saturated function $F(I_h)$, given by $F(I_h) = \frac{a_0 I_h}{a_1 + a_2 I_h}$, where a_0 , a_1 , and a_2 are information growth rate. This class is reduced by fading of memory about awareness, or human sentiment about awareness information at a rate a_3 . The saturated function $F(I_h)$ depends on the infectious human population density since the awareness about the disease and the need to protect individuals is proportional to the number of infected humans. This kind of function has been used in [50] to model the role of information in disease prevalence.

Following the above model formulation descriptions and assumptions, the deterministic model used in studying the dynamics of malaria in this study is given as

$$\begin{aligned}
 \frac{dS_h}{dt} &= \pi_h + \omega R_h - \lambda_h(A_1 + A_2)S_h - \mu_h S_h \\
 \frac{dE_h}{dt} &= \lambda_h(A_1 + A_2)S_h - (\mu_h + \sigma_h)E_h \\
 \frac{dI_h}{dt} &= \sigma_h E_h - (\tau_h(A_3) + \mu_h + \delta_h)I_h \\
 \frac{dR_h}{dt} &= \tau_h(A_3)I_h - (\mu_h + \omega)R_h \\
 (3) \quad \frac{dM_m}{dt} &= \pi_m(A_2) - (\phi + \mu_m(A_2))M_m \\
 \frac{dS_m}{dt} &= \phi M_m - \lambda_m(A_1 + A_2)S_m - \mu_m(A_1 + A_2)S_m \\
 \frac{dE_m}{dt} &= \lambda_m(A_1 + A_2)S_m - \sigma_m E_m - \mu_m(A_1 + A_2)E_m
 \end{aligned}$$

$$\begin{aligned}\frac{dI_m}{dt} &= \sigma_m E_m - \mu_m (A_1 + A_2) I_m \\ \frac{dA}{dt} &= \frac{a_0 I_h}{a_1 + a_2 I_h} - a_3 A\end{aligned}$$

with the initial conditions $S_h(0) > 0, E_h(0) \geq 0, I_h(0) \geq 0, R_h \geq 0, A(0) \geq 0, M_m > 0, S_m > 0, E_m(0) \geq 0$, and $I_m \geq 0$. The description of the model variables and parameters are presented in Table 1 and Table 2 respectively, while the schematic illustration is provided in Figure 1.

Following the approach in [46] and [51], we define the forces of infection $\lambda_h(A_1 + A_2)$ and $\lambda_m(A_1 + A_2)$ as a function of the level of awareness such that

$$(4) \quad \lambda_h(A_1 + A_2) = \frac{\beta_{hm} \varepsilon(A_1 + A_2) I_m}{N_h}, \quad \lambda_m(A_1 + A_2) = \frac{\beta_{mh} \varepsilon(A_1 + A_2) (\eta E_h + I_h)}{N_h}$$

where β_{hm} is the likelihood that a susceptible individual will be infected due to a bite by infectious mosquitoes, β_{mh} is the likelihood that a susceptible mosquito will be infected as a result of contact with infectious human, and $\varepsilon(A_1 + A_2)$ is the contact rate of humans and mosquitoes, which is dependent on awareness about bed-net usage and residual spray respectively. The contact rate associated with awareness on bed-net usage $\varepsilon(A_1)$ is given by a decreasing function

$$\varepsilon(A_1) \equiv \varepsilon(b) = \varepsilon_{max} - \frac{(\varepsilon_{max} - \varepsilon_{min}) b A_1}{1 + A_1}, \quad 0 \leq b \leq 1, \quad 0 \leq A_1 \leq 1$$

where b represents the proportion of bed-net usage, and ε_{min} and ε_{max} are the minimum and maximum mosquito biting rate respectively. Note that bed-net usage is expected to decrease the contact rate to its minimum such that if the proportion of bed-net usage $b = 0$, then transmission would be at its maximum level ε_{max} . In addition, the saturated function for the awareness of bed-net usage reduces the contact rate as the awareness increases. Similarly, the contact rate associated with awareness on the use of outdoor or indoor residual spray $\varepsilon(A_2)$ is given by a decreasing function

$$\varepsilon(A_2) \equiv \varepsilon(p) = \varepsilon_{max} - \frac{(\varepsilon_{max} - \varepsilon_{min}) p A_2}{1 + A_2}, \quad 0 \leq p \leq 1, \quad 0 \leq A_2 \leq 1$$

where ε_{min} and ε_{max} are the minimum and maximum mosquito biting rate respectively, and p is the proportion of residual spray. Note that residual spraying is expected to reduce the contact rate to its minimum such that if the proportion of residual spray $p = 0$, then transmission would be at its maximum level (ε_{max}). The saturated function for the awareness of residual spraying

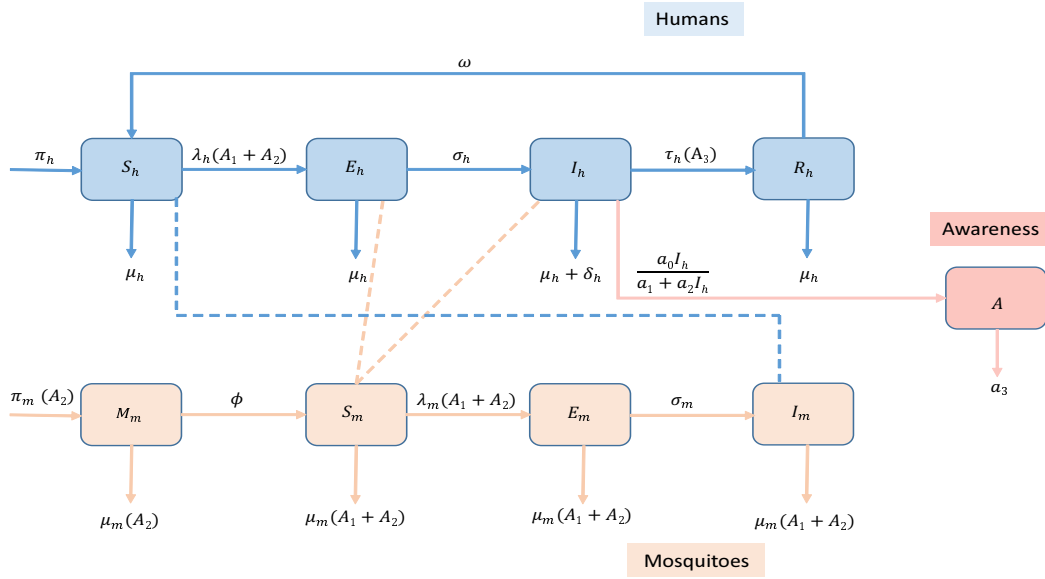


Figure 1: The schematic illustration of the malaria model (3).

reduces the contact rate as the awareness increases. We define the mosquito death $\mu_m(A_1 + A_2)$ as a function of awareness such that, $\mu_m(A_1)$ and $\mu_m(A_2)$ are the mosquitoes death rate as a result of awareness about bed-net usage and residual spray respectively. Mosquitoes hunting for blood meal can die as a result of contact with the insecticide on treated net. Thus, we model the mosquito's death rate as a result of bed-net usage as

$$(5) \quad \mu_m(A_1) \equiv \mu_m(b) = \mu_m + \frac{\mu_{max} b A_1}{1 + A_1}, \quad 0 \leq b \leq 1$$

Similarly, the mosquito's death rate as a result of residual spray is given as

$$(6) \quad \mu_m(A_2) \equiv \mu_m(p) = \mu_m + \frac{\mu_{max} p A_2}{1 + A_2}, \quad 0 \leq p \leq 1$$

where μ_m is the natural death rate of mosquito, while $\mu_{max} b$ and $\mu_{max} p$ are the death rate as a result of insecticide on bed-nets and residual spray respectively.

Variable	Description
S_h	Susceptible humans
E_h	Exposed humans
I_h	Infectious humans
R_h	Recovered humans
M_m	Immature mosquitoes
S_m	Susceptible mosquitoes
E_m	Exposed mosquitoes
I_m	Infectious mosquitoes
$\{A_1, A_2, A_3\} \in A$	Level of awareness (Bed-nets, Residual Spray, and Treatment respectively)

Table 1: Description of the state variables of the model (3).

2.1. Properties of the model: (Positivity and boundedness). The fundamental properties of model (3) will be examined in this section. For the model (3) to be epidemiologically meaningful, it is necessary to show that its state variables are positive for all time $t > 0$ and that Ω is bounded. Thus, we claim the following

Theorem 1. *The solutions of system (3) with positive initial conditions $S_h(0)$; $E_h(0)$; $I_h(0)$; $R_h(0)$; $M_m(0)$; $S_m(0)$; $E_m(0)$; $I_m(0)$; $A(0)$, will remain positive for all time $t > 0$.*

Proof. Let $t_1 = \sup\{t > 0 : S_h(0) > 0, E_h(0) > 0, I_h(0) > 0, R_h(0) > 0, M_m(0) > 0, S_m(0) > 0, E_m(0) > 0, I_m(0) > 0, A(0) > 0 \in [0, t]\}$. Hence, $t_1 > 0$. The first equation of the system (3) is written as

$$(7) \quad \frac{dS_h}{dt} = \pi_h + \omega R_h - \lambda_h(A_1 + A_2)S_h - \mu_h S_h \geq \pi_h - \lambda_h(A_1 + A_2)S_h - \mu_h S_h$$

By using the integrating factor method, the above expression is further given as

$$\frac{d}{dt} \left(S_h(t) \exp \left[\mu_h t + \int_0^t \lambda_h(A_1 + A_2)(x) dx \right] \right) \geq \pi_h \exp \left[\mu_h t + \int_0^t \lambda_h(A_1 + A_2)(x) dx \right]$$

Hence,

$$S_h(t_1) \exp \left[\mu_h t_1 + \int_0^{t_1} \lambda_h(A_1 + A_2)(x) dx \right] - S_h(0) \geq \int_0^{t_1} \pi_h \left(\exp \left[\mu_h y + \int_0^y \lambda_h(A_1 + A_2)(x) dx \right] \right) dy$$

Parameter	Description
π_h	Recruitment rate of humans
ω	Immunity waning rate of humans
σ_h	Disease progression rate from exposed to infectious human
σ_m	Disease progression rate from exposed to infectious mosquito
τ_h	Recovery rate of infectious humans
$\tau_{max}q$	Recovery rate of infectious humans due to awareness on treatment care
η	Disease modification parameter
μ_h	Natural death rate of humans
δ_h	Death rate due to disease for humans
β_{hm}	Probability of effective transmission from human to mosquito
β_{mh}	Probability of effective transmission from mosquito to human
ϵ_{max}	Maximum mosquito biting rate
ϵ_{min}	Minimum mosquito biting rate
b	Proportion of bed-net usage
p	Proportion of residual spray indoor or outdoor
π_{max}	Maximum egg deposition rate
π_{min}	Minimum egg deposition rate
ϕ	Maturation rate of immature mosquitoes
μ_m	Natural death rate of mosquitoes
$\mu_{max}b$	Mosquitoes death rate as a result of bed-net usage
$\mu_{max}q$	Mosquitoes death rate as a result of residual spray
a_0, a_1, a_2	Awareness information growth rate
a_3	Fading of awareness memory

Table 2: Description of the parameters of the model (3).

so that,

$$\begin{aligned}
S_h(t_1) &\geq S_h(0) \exp \left[-\mu_h t_1 - \int_0^{t_1} \lambda_h(A_1 + A_2)(x) dx \right] \\
&+ \exp \left[-\mu_h t_1 - \int_0^{t_1} \lambda_h(A_1 + A_2)(x) dx \right] \times \int_0^{t_1} \pi_h \left(\exp \left[\mu_h y + \int_0^y \lambda_h(A_1 + A_2)(x) dx \right] \right) dy > 0.
\end{aligned}$$

In the same way, it can be shown that $E_h(t) > 0$, $I_h(t) > 0$, $R_h(t) > 0$, $M_m(t) > 0$, $S_m(t) > 0$, $E_m(t) > 0$, $I_m(t) > 0$, and $A(t) > 0$ for all time $t > 0$. \square

Furthermore, for the malaria model (3) to be mathematically and epidemiologically meaningful, it is necessary to analyze system (3) in a biologically feasible region $\Omega = \Omega_h \times \Omega_m \in \mathcal{R}_+^5 \times \mathcal{R}_+^4$ such that

$$\Omega_h = \left\{ (S_h, E_h, I_h, R_h, A) \in \mathcal{R}_+^5 : S_h + E_h + I_h + R_h \leq \frac{\pi_h}{\mu_h}, \quad A \leq \frac{a_0 \pi_h}{a_3(a_1 \mu_h + a_2 \pi_h)} \right\}$$

and

$$\Omega_m = \left\{ (M_m, S_m, E_m, I_m) \in \mathcal{R}_+^4 : M_m + S_m + E_m + I_m \leq \frac{\pi_m(A_2)}{\mu_m} \right\}$$

where $\mu_m = \min \{ \mu_m(A_2), \mu_m(A_1 + A_2) \}$. Using the standard technique (see [50, 52]), the feasible region Ω can be shown to be positively invariant. Hence, all the solutions are in the feasible region Ω where the malaria model (3) is said to be mathematically and epidemiologically well-posed [53, 52]. We claim the following result in the theorem below

Theorem 2. *The biological feasible region $\Omega = \Omega_h \cup \Omega_m \subset \mathcal{R}_+^5 \times \mathcal{R}_+^4$ is positively invariant for the malaria model (3) with non-negative initial conditions in \mathcal{R}_+^9 .*

3. MODEL ANALYSIS

Here, we investigate the existence and stability of the steady states, and the nature of bifurcation exhibited by system (3) is examined. The model presented in (3) has two steady states namely; the disease-free equilibrium (henceforth refer to as malaria-free equilibrium), and the endemic equilibrium. The malaria-free steady-state solution describes the population without malaria infection, while the endemic equilibrium steady-state solution exists at any positive prevalence of malaria in the population.

3.1. Existence and stability of the malaria-free equilibrium (MFE). The malaria-free equilibrium of the system (3), represented by \mathcal{M}_0 is obtained as

$$\begin{aligned} \mathcal{M}_0 &= (S_h^*, E_h^*, I_h^*, R_h^*, M_m^*, S_m^*, E_m^*, I_m^*, A^*) \\ (8) \quad &= \left(\frac{\pi_h}{\mu_h}, 0, 0, 0, \frac{\pi_m(A_2)}{(\phi + \mu_m(A_2))}, \frac{\phi \pi_m(A_2)}{\mu_m(A_1 + A_2)(\phi + \mu_m(A_2))}, 0, 0, 0 \right) \end{aligned}$$

We compute the reproduction number \mathcal{R}_0 to study the stability of the model. Using the approach and notations in [52, 54], the matrix F (new infections) and matrix V (transition terms) are respectively given as

$$F = \begin{pmatrix} 0 & 0 & 0 & \beta_{hm}\varepsilon(A_1 + A_2) \\ 0 & 0 & 0 & 0 \\ \frac{\eta\beta_{mh}\varepsilon(A_1+A_2)S_m^*}{S_h^*} & \frac{\beta_{mh}\varepsilon(A_1+A_2)S_m^*}{S_h^*} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ -\sigma_h & k_2 & 0 & 0 \\ 0 & 0 & \mu_m(A_1 + A_2) + \sigma_m & 0 \\ 0 & 0 & -\sigma_m & \mu_m(A_1 + A_2) \end{pmatrix}$$

where $k_1 = \mu_h + \sigma_h$, and $k_2 = \tau_h(A_3) + \delta_h + \mu_h$. The next generation matrix (NGM) with large domain $K_L = FV^{-1}$ is given below as

$$(9) K_L = \begin{pmatrix} 0 & 0 & \frac{\beta_{hm}\sigma_m\varepsilon(A_1+A_2)}{(\mu_m(A_1+A_2)+\sigma_m)\mu_m(A_1+A_2)} & \frac{\beta_{hm}\varepsilon(A_1+A_2)}{\mu_m(A_1+A_2)} \\ 0 & 0 & 0 & 0 \\ \frac{\beta_{mh}\varepsilon(A_1+A_2)S_m^*(\eta k_2 + \sigma_h)}{k_1 k_2 S_h^*} & \frac{\beta_{mh}\varepsilon(A_1+A_2)S_m^*}{k_2 S_h^*} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

It is obvious from the model equation (3) that, there are only two states-at-infection among the four infected states. This can also be seen by looking at matrix F and observing that the entire second and fourth row contains zeros. Hence, the NGM K for the small domain is therefore two-dimensional. Thus, using the approach of [55] with an auxiliary matrix E , the NGM K is obtained as

$$(10) K = E^T K_L E = E^T F V^{-1} E = \begin{pmatrix} 0 & \frac{\beta_{hm}\sigma_m\varepsilon(A_1+A_2)}{(\mu_m(A_1+A_2)+\sigma_m)\mu_m(A_1+A_2)} \\ \frac{\beta_{mh}\varepsilon(A_1+A_2)S_m^*(\eta k_2 + \sigma_h)}{k_1 k_2 S_h^*} & 0 \end{pmatrix}$$

Thus, it follows that the reproduction number for the system (3), which is the spectral radius of K given by $\mathcal{R}_0 = \rho(K)$, is obtained as

$$(11) \quad \mathcal{R}_0 = \sqrt{\mathcal{R}_h \mathcal{R}_m} = \sqrt{\frac{\beta_{mh} \beta_{hm} \sigma_m (\eta k_2 + \sigma_h) \varepsilon^2 (A_1 + A_2) S_m^*}{k_1 k_2 S_h^* \{(\mu_m (A_1 + A_2) + \sigma_m) \mu_m (A_1 + A_2)\}}}$$

where

$$\mathcal{R}_h = \frac{\beta_{mh} \varepsilon (A_1 + A_2) S_m^* (\eta k_2 + \sigma_h)}{k_1 k_2 S_h^*}, \quad \mathcal{R}_m = \frac{\beta_{hm} \sigma_m \varepsilon (A_1 + A_2)}{(\mu_m (A_1 + A_2) + \sigma_m) \mu_m (A_1 + A_2)}.$$

The reproduction number \mathcal{R}_0 is a threshold quantity that characterizes the average number of new secondary infections generated by a single infected individual during an infectious period, in a completely susceptible population [41, 54]. Consequently, the threshold quantity given in equation (11) represents the average number of malaria infections that one malaria-infected individual can reproduce in an entirely susceptible population. Using Theorem 2 in [56], the local stability of the malaria-free equilibrium \mathcal{M}_0 is summarized in the theorem below.

Theorem 3. *The malaria-free equilibrium \mathcal{M}_0 , of the model (3) is locally asymptotically stable in the biological feasible region Ω if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. To establish Theorem 3, we obtain the Jacobian matrix of system (3) at malaria free-equilibrium \mathcal{M}_0 as

$$(12) \quad \mathcal{J}(\mathcal{M}_0) = \begin{pmatrix} -\mu_h & 0 & 0 & \omega & 0 & 0 & 0 & -k_8 & 0 \\ 0 & -k_1 & 0 & 0 & 0 & 0 & 0 & k_8 & 0 \\ 0 & \sigma_h & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_h(A_3) & -k_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_4 & 0 & 0 & 0 & 0 \\ 0 & -k_5 \eta & -k_5 & 0 & \phi & -k_6 & 0 & 0 & 0 \\ 0 & k_5 \eta & k_5 & 0 & 0 & 0 & -k_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma_m & -k_6 & 0 \\ 0 & 0 & \frac{a_0}{a_1} & 0 & 0 & 0 & 0 & 0 & -a_3 \end{pmatrix}$$

Such that $k_1 = \mu_h + \sigma_h$, $k_2 = \tau_h(A_3) + \delta_h + \mu_h$, $k_3 = \omega + \mu_h$, $k_4 = \phi + \mu_m(A_1 + A_2)$, $k_6 = \mu_m(A_1 + A_2)$, $k_7 = \mu_m(A_1 + A_2) + \sigma_m$, $k_8 = \beta_{hm} \varepsilon (A_1 + A_2)$ and $k_5 = \frac{S_m^* \beta_{mh} \varepsilon (A_1 + A_2)}{S_h^*}$. From (12),

we can show that all the eigenvalues of $\mathcal{J}(\mathcal{M}_0)$ are negative. The first five eigenvalues are obtained as, $-\mu_h, -k_3, -k_4, -k_6$ and $-a_3$. Thus, the remaining four eigenvalues are obtained from the sub-matrix \mathcal{S} , given as

$$(13) \quad \mathcal{S} = \begin{pmatrix} -k_1 & 0 & 0 & k_8 \\ \sigma_h & -k_2 & 0 & 0 \\ k_5\eta & k_5 & -k_7 & 0 \\ 0 & 0 & \sigma_m & -k_6 \end{pmatrix}$$

As stated by the Routh-Hurwitz criterion, the matrix \mathcal{S} will be real and negative if

- (i) $\text{Tr}(\mathcal{S}) < 0$
- (ii) $\text{Det}(\mathcal{S}) > 0$

From 13,

$$\text{Tr}(\mathcal{S}) = -(k_1 + k_2 + k_7 + k_6) < 0$$

and

$$\text{Det}(\mathcal{S}) = k_1 k_2 k_6 k_7 (1 - \mathcal{R}_0) > 0 \quad \text{if} \quad \mathcal{R}_0 < 1$$

All the eigenvalues of the matrix (12) are real and negative if $\mathcal{R}_0 < 1$, thus, the malaria-free equilibrium \mathcal{M}_0 is locally asymptotically stable and unstable otherwise. \square

Theorem 3 suggests that malaria can be controlled in the population whenever $\mathcal{R}_0 < 1$ if the initial sizes of the sub-population of system (3) are in the basin of attraction of \mathcal{M}_0 .

3.2. Existence of endemic equilibria and backward bifurcation. Here, we examine the possibilities of the existence of endemic equilibria and a backward bifurcation. A model is known to exhibit the phenomenon of backward bifurcation when a small positive unstable equilibrium appears while the disease-free equilibrium and a larger positive equilibrium are locally asymptotically stable when the threshold quantity is less than unity. In other words, this phenomenon is possible when the stable disease-free equilibrium coexists with a stable endemic equilibrium, under some given values for which the reproduction number is less than unity. The endemic equilibria denoted by $\mathcal{M}_1 = (S_h^{**}, E_h^{**}, I_h^{**}, R_h^{**}, M_m^{**}, S_m^{**}, E_m^{**}, I_m^{**}, A^{**})$ represents

the steady-state solution in the presence of malaria, and is obtained as

$$\begin{aligned}
S_h^{**} &= \frac{k_1 k_2 k_3 \pi_h}{k_1 k_2 k_3 [\lambda_h^{**}(A_1 + A_2) + \mu_h] - \omega \sigma_h \tau_h(A_3) \lambda_h^{**}(A_1 + A_2)} \\
E_h^{**} &= \frac{k_2 k_3 \pi_h \lambda_h^{**}(A_1 + A_2)}{k_1 k_2 k_3 [\lambda_h^{**}(A_1 + A_2) + \mu_h] - \omega \sigma_h \tau_h(A_3) \lambda_h^{**}(A_1 + A_2)} \\
I_h^{**} &= \frac{k_3 \pi_h \sigma_h \lambda_h^{**}(A_1 + A_2)}{k_1 k_2 k_3 [\lambda_h^{**}(A_1 + A_2) + \mu_h] - \omega \sigma_h \tau_h(A_3) \lambda_h^{**}(A_1 + A_2)} \\
(14) \quad R_h^{**} &= \frac{\pi_h \sigma_h \tau_h(A_3) \lambda_h^{**}(A_1 + A_2)}{k_1 k_2 k_3 [\lambda_h^{**}(A_1 + A_2) + \mu_h] - \omega \sigma_h \tau_h(A_3) \lambda_h^{**}(A_1 + A_2)} \\
M_m^{**} &= \frac{\pi_m(A_2)}{k_4}, \quad S_m^{**} = \frac{\phi \pi_m(A_2)}{k_4 [\lambda_m^{**}(A_1 + A_2) + k_6]} \\
E_m^{**} &= \frac{\phi \pi_m(A_2) \lambda_m^{**}(A_1 + A_2)}{k_4 k_7 [\lambda_m^{**}(A_1 + A_2) + k_6]}, \quad I_m^{**} = \frac{\phi \sigma_m \pi_m(A_2) \lambda_m^{**}(A_1 + A_2)}{k_4 k_6 k_7 [\lambda_m^{**}(A_1 + A_2) + k_6]} \\
A^{**} &= \frac{a_0 \pi_h \sigma_h k_3 \lambda_h^{**}(A_1 + A_2)}{a_3 \{ a_1 k_1 k_2 k_3 [\mu_h + \lambda_h^{**}(A_1 + A_2)] + \sigma_h \lambda_h^{**}(A_1 + A_2) [a_2 k_3 \pi_h - a_1 \omega \tau_h(A_3)] \}}
\end{aligned}$$

with the force of infections given as

$$(15) \quad \lambda_h^{**}(A_1 + A_2) = \frac{\beta_{hm} \varepsilon (A_1 + A_2) I_m^{**}}{N_h^{**}}, \quad \lambda_m^{**}(A_1 + A_2) = \frac{\beta_{mh} \varepsilon (A_1 + A_2) (\eta E_h^{**} + I_h^{**})}{N_h^{**}}$$

substituting equation 14 and the value of $\lambda_m^{**}(A_1 + A_2)$ into (15) and expanding in $\lambda_h^{**}(A_1 + A_2)$ results to the following polynomial equation

$$(16) \quad z_1 [\lambda_h^{**}(A_1 + A_2)]^2 + z_2 \lambda_h^{**}(A_1 + A_2) + z_3 = 0$$

where the polynomial coefficients z_i for $i = 1, \dots, 3$ are given as

$$(17) \quad z_1 = P_1 P_6 P_8 \pi_h, \quad z_2 = P_6^2 (P_8 + P_1 \mu_m \pi_h) - P_2 P_3 P_7, \quad z_3 = \mu_m (\pi_h k_1 k_2 k_3)^3 (1 - \mathcal{R}_0^2)$$

where: $P_1 = k_2k_3 + k_3\sigma_h + \sigma_h\tau_h(A_3)$, $P_2 = k_8\sigma_m\phi\pi_m(A_2)$, $P_3 = k_1k_2k_3 - \omega\sigma_h\tau_h(A_3)$, $P_4 = \pi_hk_1k_2k_3$, $P_5 = \pi_hk_3\beta_{mh}(\eta k_2 + \sigma_h)\varepsilon(A_1 + A_2)$, and $P_6 = P_1\pi_h\mu_m(A_1 + A_2) + P_5$. From the polynomial (16) above, the coefficient z_1 is always positive, and the constant term z_3 is negative or positive depending on the value of \mathcal{R}_0 . This implies that, if $\mathcal{R}_0^2 > 1$, z_3 is negative and if $\mathcal{R}_0^2 < 1$, then z_3 is positive. Thus, the following result hold.

Theorem 4. *The malaria model given by equation (3) has*

- (i) *exactly one unique endemic equilibrium if $z_3 < 0$ or $\mathcal{R}_0 > 1$,*
- (ii) *exactly one unique endemic equilibrium if $z_2 < 0$, and either $z_3 = 0$ or $z_2^2 - 4z_1z_3 = 0$,*
- (iii) *exactly two endemic equilibria if $z_3 > 0$, $z_2 < 0$ and $z_2^2 - 4z_1z_3 > 0$,*
- (iv) *no endemic equilibrium otherwise.*

It is obvious from case (i) of Theorem 4 that the malaria model (3) has a unique equilibrium point represented by \mathcal{M}_1 , whenever $\mathcal{R}_0 > 1$. In addition, case (iii) of Theorem 4 shows the possibility of backward bifurcation in the malaria model (3) when $\mathcal{R}_0 < 1$. To check for the possibility of backward bifurcation when $\mathcal{R}_0 < 1$, we set the discriminant $z_2^2 - 4z_1z_3 = 0$ and solve for the critical value of \mathcal{R}_0 denoted by \mathcal{R}_0^c , such that

$$(18) \quad \mathcal{R}_0^c = \sqrt{1 - \frac{z_2^2}{4z_1\mu_m(\pi_hk_1k_2k_3)^3}}$$

Hence, backward bifurcation will occur for the value \mathcal{R}_0^c such that $\mathcal{R}_0 < 1$. The result is summarized in the theorem below.

Theorem 5. *The malaria model (3) undergoes a backward bifurcation when case (iii) of Theorem 4 holds and $\mathcal{R}_0^c < \mathcal{R}_0 < 1$.*

Following the result above, the backward bifurcation phenomenon implies that the epidemiological condition of having the reproduction number less than unity to eradicate a disease although necessary is no longer enough for disease eradication. Thus, to effectively control malaria in the population, additional control measures will be needed to enable epidemic control. That is, the condition $\mathcal{R}_0 < \mathcal{R}_0^c < 1$ must be satisfied.

4. NUMERICAL RESULTS AND DISCUSSION

We examine the effect of control interventions (bed-net usage, residual spray, and treatment) and their level of awareness on the dynamics of malaria. To accomplish this, we performed a sensitivity analysis to investigate the impact of control interventions on the reproduction number. Furthermore, we simulate the proposed model (3) under different scenarios, using the baseline parameter values as given in Table 3, except otherwise stated.

4.1. Impact of interventions on \mathcal{R}_0 . Since the threshold quantity \mathcal{R}_0 given in (11) determines the control of malaria in the population (except for scenario where the bifurcation phenomenon occurs), we assess the impact of the interventions (bed-net usage, insecticide residual spray, and treatment) on the reproduction number \mathcal{R}_0 . To accomplish this, we use the normalized forward sensitivity indices to investigate the relationship of each parameter on \mathcal{R}_0 . Using the method in [59, 60], the normalized forward sensitivity index $\mathcal{X}_i^{\mathcal{R}_0}$ for each of the intervention parameter $\{b, p, q \in i\}$, is defined as

$$(19) \quad \mathcal{X}_i^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial i} \times \frac{i}{\mathcal{R}_0}$$

By using the formula presented in (19), the numerical values for the normalized forward sensitivity indices of the three intervention parameters are given in Table 3. It must be noted that, $\mathcal{X}_b^{\mathcal{R}_0} < 0$, $\mathcal{X}_p^{\mathcal{R}_0} < 0$, and $\mathcal{X}_q^{\mathcal{R}_0} < 0$. This implies that an increase in the respective intervention parameters will reduce the value of the reproduction number. For instance, increasing the number of individuals who use bed-net will reduce the reproduction number and vice versa. In Figure 2, we simulate the effect of each intervention on the reproduction number. Figure 2 shows a decrease in the reproduction number with increasing interventions as expected. However, Figure 2(c) shows that the proportion of treatment is less significant on the reproduction number. Thus, control interventions such as residual spray, and bed-net usage should be prioritized in reducing the burden of malaria in the population.

To examine the effect of control intervention and awareness on malaria burden, we obtained some contour plots for the reproduction number \mathcal{R}_0 , as a function of control interventions and their respective level of awareness. As shown in Figure 3, an increase in control interventions with the level of awareness reduces the reproduction number. Specifically, Figure 3(a) show

Parameters	Baseline value	Range	Dimension	Reference
π_h	$\left(\frac{10^3}{65 \times 365}\right)$	$\left(\frac{10^3}{80 \times 365} - \frac{10^3}{58 \times 365}\right)$	Day ⁻¹	[41, 26]
ω	0.0005275	$(5.5 \times 10^{-5} - 1.1 \times 10^{-2})$	Day ⁻¹	[41, 26]
σ_h	0.058824	$\left(\frac{67}{10^3} - \frac{2}{10}\right)$	Day ⁻¹	[57, 11, 2]
σ_m	0.05555	$\left(\frac{29}{10^3} - \frac{33}{10^2}\right)$	Day ⁻¹	[57, 11, 2]
τ_h	0.0092	$\left(\frac{14}{10^4} - \frac{17}{10^3}\right)$	Day ⁻¹	[41, 26]
$\tau_{max}q$	$\tau_h \times q$	$\left(\frac{14}{10^4} - \frac{17}{10^3}\right) \times q$	Day ⁻¹	Estimated
η	0.5	(0.1 – 1)	Dimensionless	[41]
μ_h	$\left(\frac{1}{65 \times 365}\right)$	$\left(\frac{1}{80 \times 365} - \frac{1}{58 \times 365}\right)$	Day ⁻¹	[41, 26]
δ_h	0.0003454	$(1 \times 10^{-15} - 4.1 \times 10^{-4})$	Day ⁻¹	[41, 26]
β_{hm}	$\frac{22}{10^3}$	$\left(\frac{1}{10^2} - \frac{27}{10^2}\right)$	Dimensionless	[58, 57]
β_{mh}	$\frac{48}{10^2}$	$\left(\frac{72}{10^3} - \frac{64}{10^2}\right)$	Dimensionless	[58, 57]
ϵ_{max}	0.5	(0.1 – 1)	Day ⁻¹	[58, 57]
ϵ_{min}	1×10^{-2}	(0 – 0.1)	Day ⁻¹	[58, 57]
b	0.250	$(0 \leq b \leq 1)$	Dimensionless	Variable
p	0.250	$(0 \leq p \leq 1)$	Dimensionless	Variable
q	0.250	$(0 \leq q \leq 1)$	Dimensionless	Variable
π_{max}	$\frac{10^4}{14}$	$\left(\frac{10^4}{21} - \frac{10^4}{14}\right)$	Day ⁻¹	[57, 58]
π_{min}	$\frac{10}{14}$	$\left(\frac{10}{21} - \frac{10}{14}\right)$	Day ⁻¹	[57, 58]
ϕ	0.343	(0.333 – 1)	Day ⁻¹	[41]
μ_m	$\frac{1}{18}$	$\left(\frac{1}{21} - \frac{1}{3}\right)$	Day ⁻¹	[41, 26]
$\mu_{max}b$	$\mu_m \times b$	$\left(\frac{1}{21} - \frac{1}{3}\right) \times b$	Day ⁻¹	Estimated
$\mu_{max}p$	$\mu_m \times p$	$\left(\frac{1}{21} - \frac{1}{3}\right) \times p$	Day ⁻¹	Estimated
a_0, a_1, a_2	0.03	0.01 – 0.05	Dimensionless	Assumed
a_3	0.01	0.00 – 0.02	Dimensionless	Assumed
$\{A_1, A_2, A_3\} \in A$	0.25	$(0 \leq A \leq 1)$	Dimensionless	Variable

Table 3: Parameter values of the malaria model.

Parameter	b	p	q
Sensitivity Index	-0.38807	-0.61054	-0.06805

Table 4: Sensitivity indices of the intervention parameters.

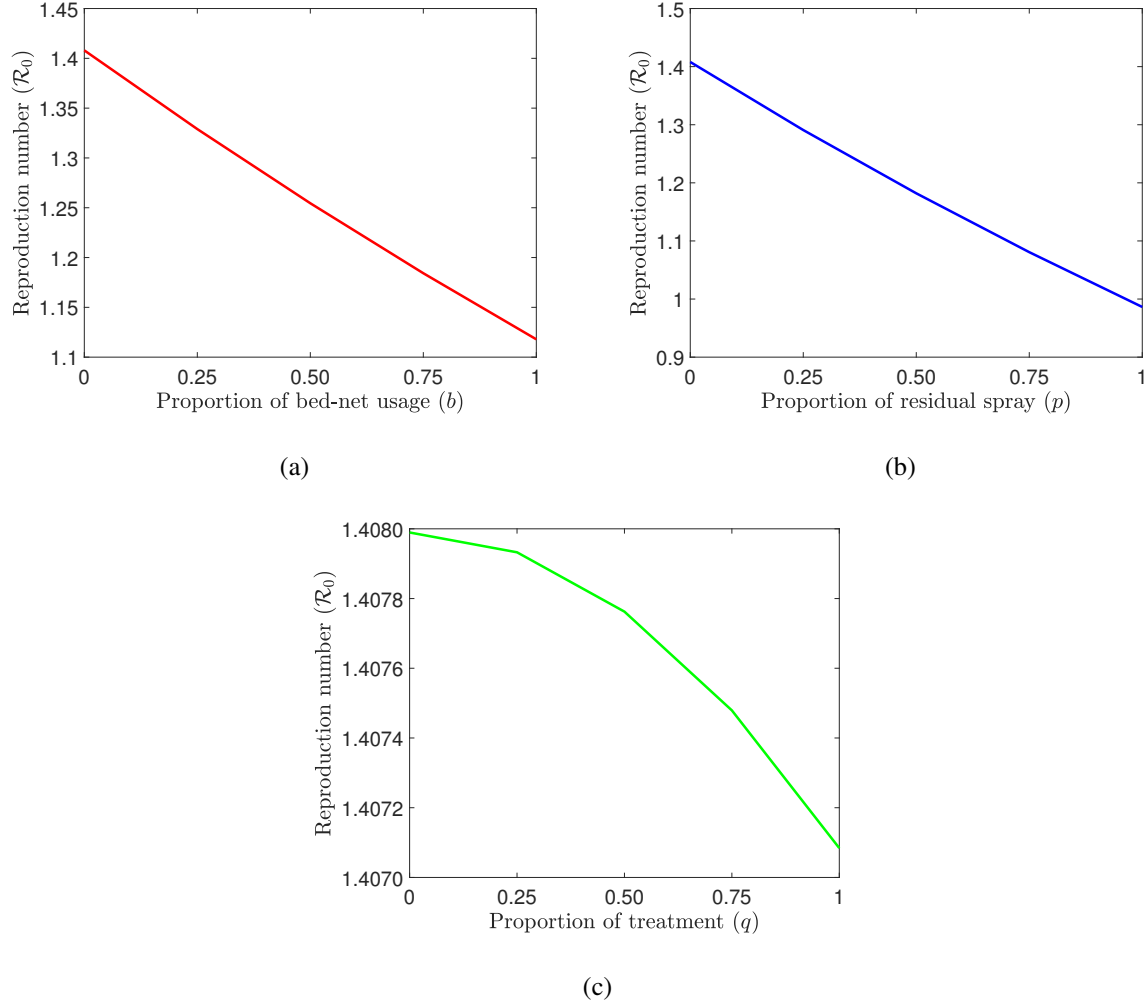


Figure 2: Reproduction number \mathcal{R}_0 of malaria model (3), with respect to (a) Proportion of bed-net usage (b); (b) Proportion of residual spray (p); and (c) Proportion of treatment (q). The parameter values used are given in Table 3 except for $\delta_h = 0.0003454 \times 10^3$, to facilitate $\mathcal{R}_0 = 1.41$.

that increase in the proportion of bed-net usage (b) and its level of awareness (A_1) decrease the reproduction number, while Figure 3(b) show that increase in the proportion of residual spray (p) and its level of awareness (A_2) decrease the reproduction number. It must be noted that in Figure 3(a) and Figure 3(b), as control interventions with their level of awareness converge to

one, the reproduction number reduces below unity. This implies that, if 100% of the population are aware and uses either of the control interventions strategies (bed-net or residual spray), malaria is expected to be eradicated in the population in the absence of backward bifurcation phenomenon. Figure 3(c) show that increase in the proportion of treatment (q) and its level of awareness (A_3) decreases the reproduction number. However, unlike the result from Figure 3(a) and Figure 3(b), as the proportion of treatment (q) and its level of awareness (A_3) converges to one, the reproduction number fails to reduce below unity. This means that, even if all infected individuals are treated for malaria, the disease will not be eliminated in the population. This result is expected since the model allows reinfection of recovered humans due to the loss of immunity.

4.2. Impact of interventions and awareness on infected population. We examine the behavior of infected human and mosquito populations under different scenarios to predict the elimination of malaria in the population. Since malaria exposed individuals can transfer the infection, we defined the total infected human population as the summation of exposed humans and infectious humans ($E_h + I_h$). Similarly, we considered the total infected mosquito population as the summation of exposed mosquitoes and infectious mosquitoes ($E_m + I_m$). To investigate the impact of control interventions and their level of awareness on the infected population, we simulate model (3) under three different scenarios. For the first scenario, we simulate the impact of single control intervention such as bed-net usage only, residual spray only, and treatment only. For the second scenario, we simulate the impact of double control intervention such as (bed-net usage and residual spray) only, (bed-net usage and treatment) only, and (residual spray and treatment) only. For the last scenario, we simulate the impact of all the control interventions (bed-net usage, residual spray, and treatment) on the infected population. Throughout the simulation, we assumed that the proportion of control interventions usage is equivalent to their level of awareness since realistically the proportion of control intervention usage is dependent on the level of awareness.

Figure 4 depicts the effect of single control interventions with their respective level of awareness on the infected human and mosquito population. From Figure 4(a) and Figure 4(b), the

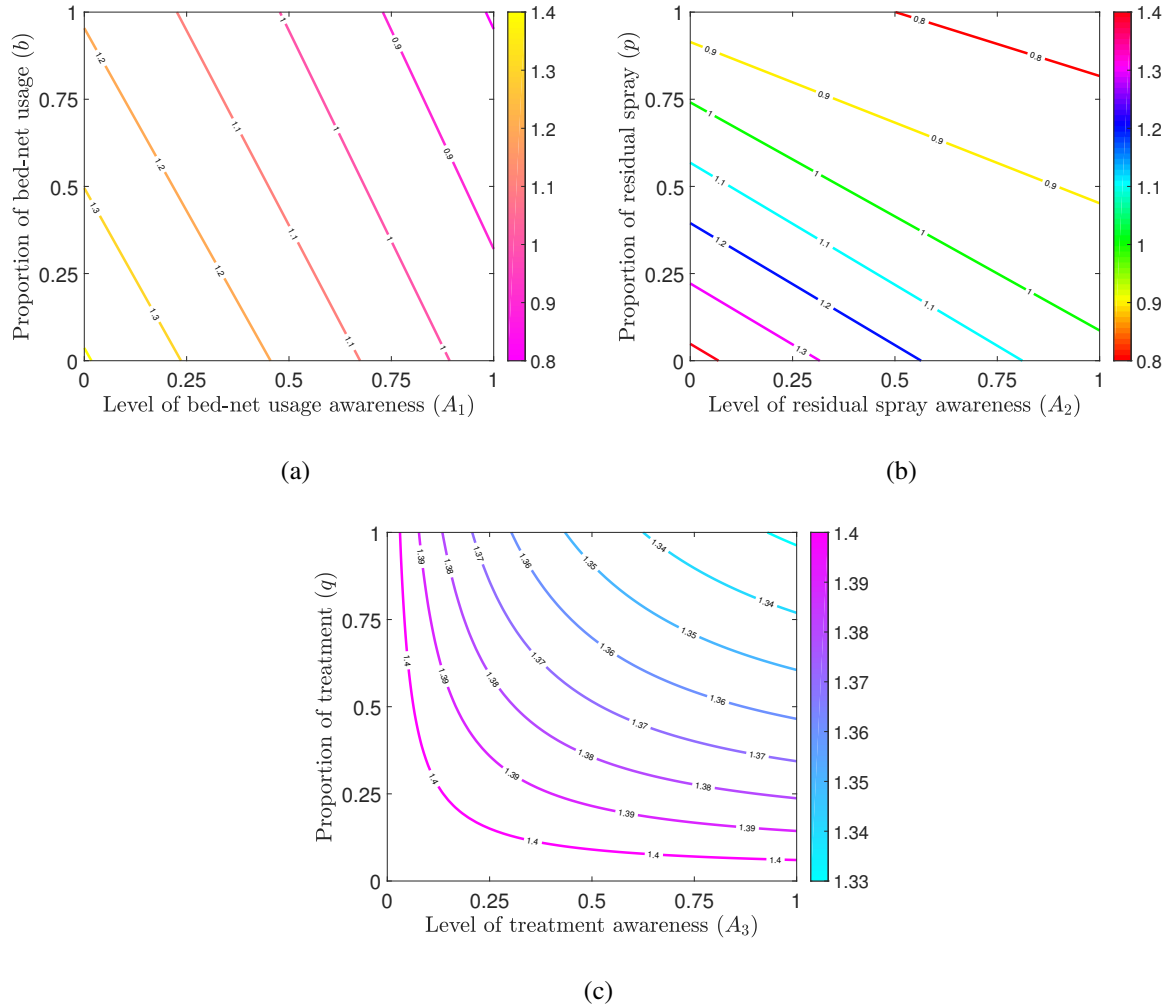


Figure 3: Contour plot of the reproduction number \mathcal{R}_0 of malaria model (3), (a) varying proportion of bed-net usage (b) with respect to level of bed-net usage awareness (A_1); (b) varying proportion of residual spray (p) with respect to level of residual spray awareness (A_2); (c) varying proportion of treatment (q) with respect to level of treatment awareness (A_3). Parameter values used are given in Table 3 except for $\delta_h = 0.0003454 \times 10^3$, to facilitate $\mathcal{R}_0 = 1.41$.

result shows that increase in bed-net usage and its level of awareness reduces the infected human and mosquito population respectively. Similarly, from Figure 4(c) and Figure 4(d) result shows that increase in residual spray and its level of awareness reduces the infected human and mosquito population respectively. In Figure 4(c) and Figure 4(d), it is obvious that residual spray reduces the burden of malaria faster than bed-net usage. This supports the result from the sensitivity analysis as presented in Table 4. From the result, it is noted that increasing the

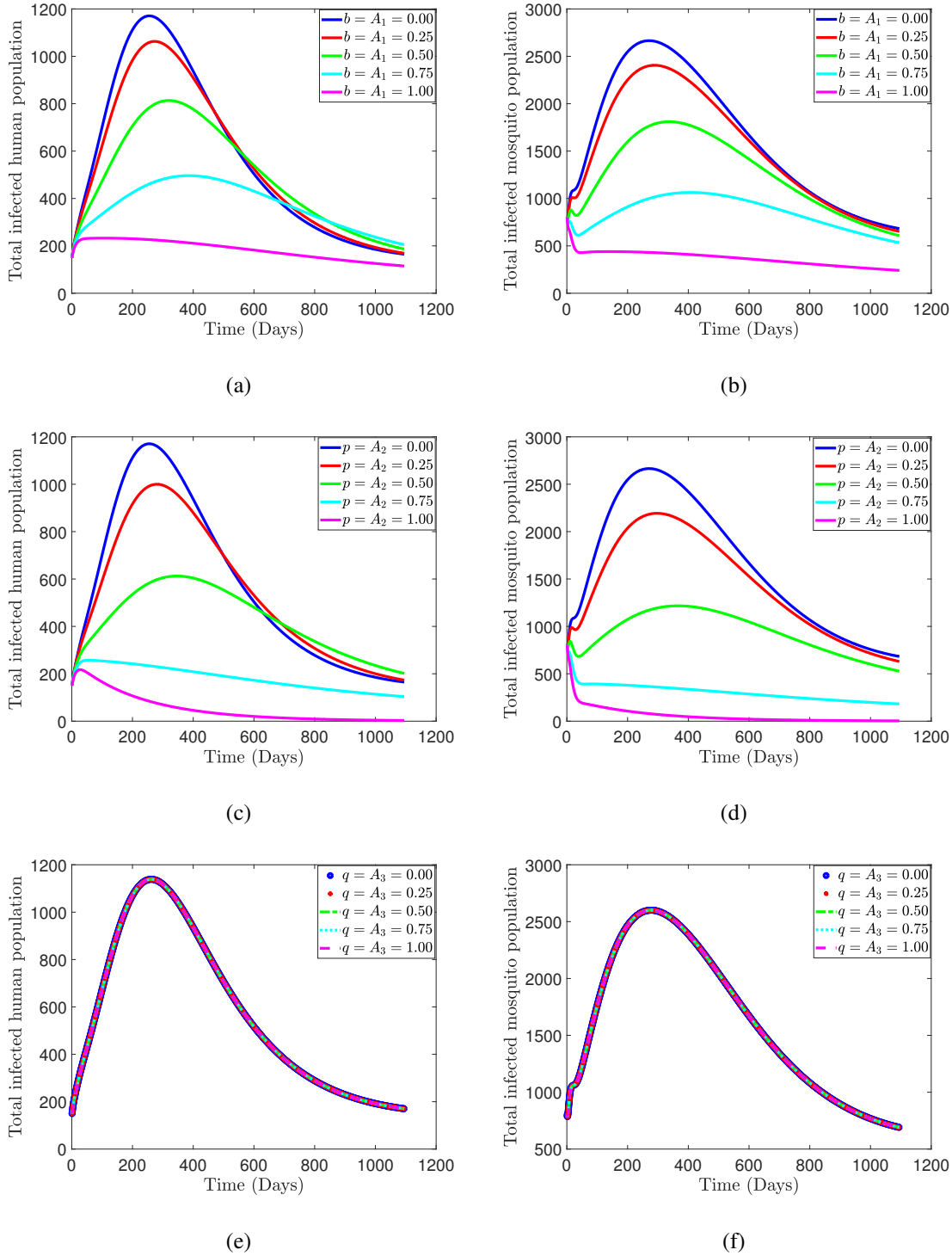


Figure 4: Simulations of the malaria model (3) showing the effects of intervention and respective to level of awareness on the total infected human population ($E_h + I_h$) and total infected mosquito population ($E_m + I_m$). (a,b) Bed-net usage only ($p = q = A_2 = A_3 = 0$); (c,d) residual spray only ($b = q = A_1 = A_3 = 0$); and (e,f) treatment only ($b = p = A_1 = A_2 = 0$). The parameter values used are as given in Table 3.

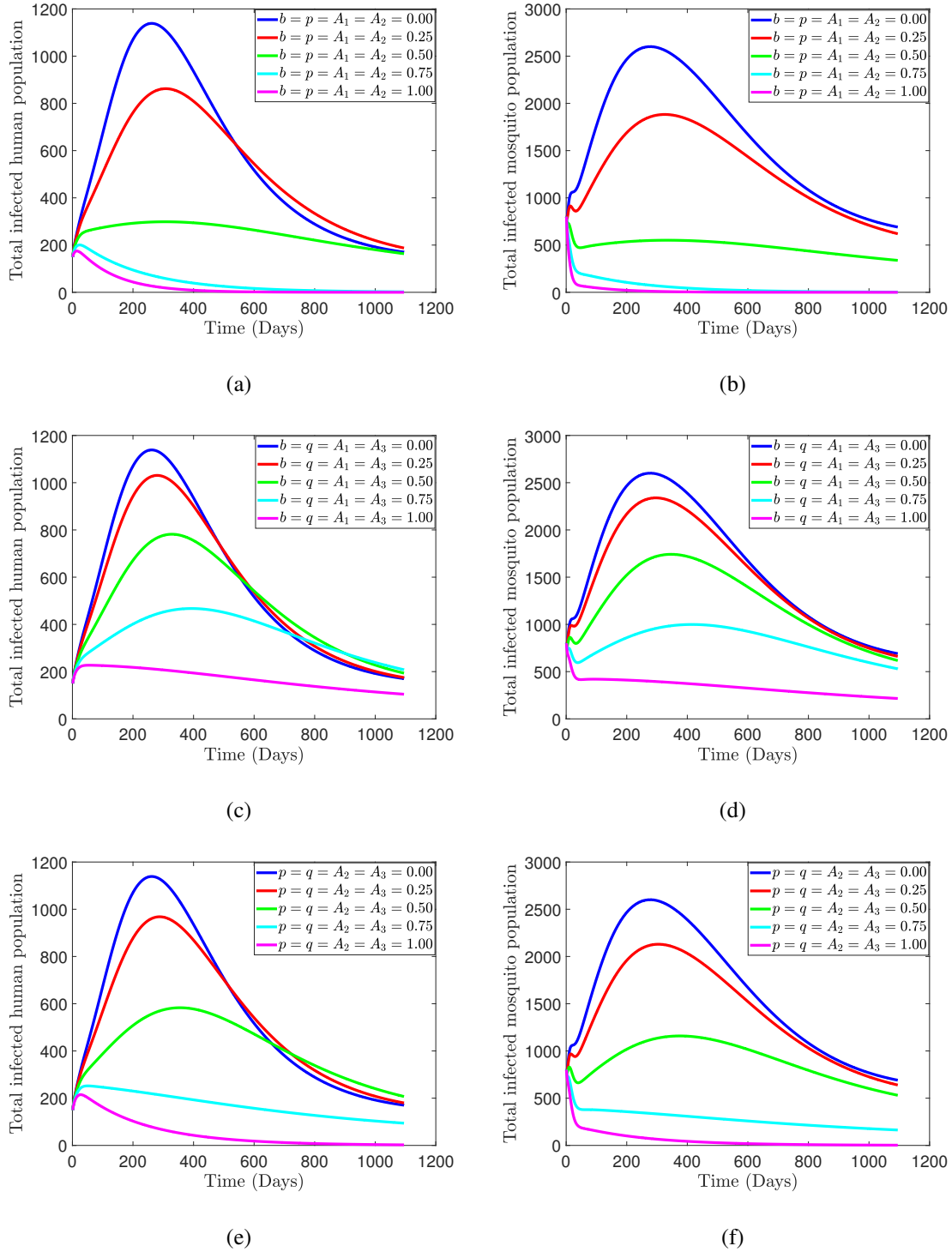


Figure 5: Simulations of the malaria model (3) showing the effects of combined intervention and respective to level of awareness on the total infected human population ($E_h + I_h$) and total infected mosquito population ($E_m + I_m$). (a,b) Bed-net usage and residual spray only ($q = A_3 = 0$); (c,d) bed-net usage and treatment only ($p = A_2 = 0$); and (e,f) residual spray and treatment only ($b = A_1 = 0$). The parameter values used are as given in Table 3.

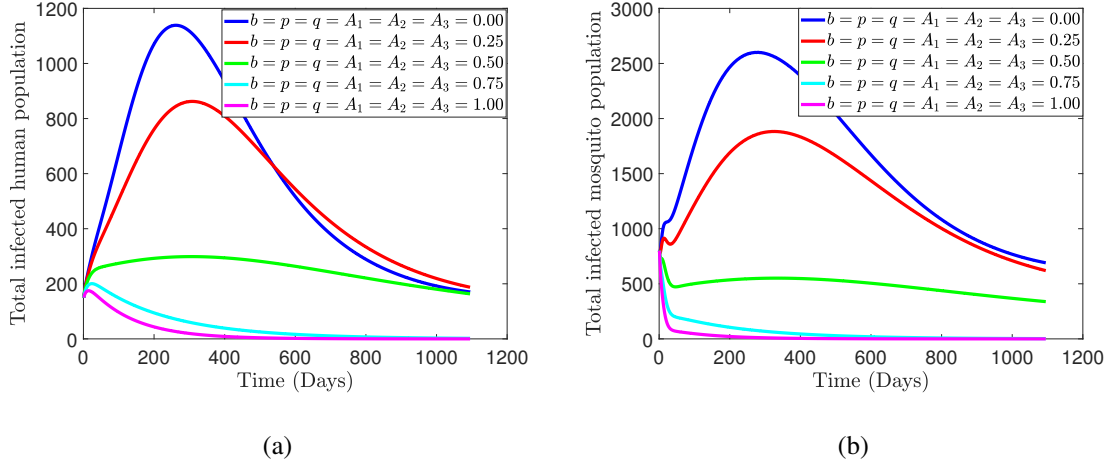


Figure 6: Simulations of the malaria model (3) showing the effects of all the intervention and respective awareness on the total infected human population ($E_h + I_h$) and total infected mosquito population ($E_m + I_m$). The parameter values used are as given in Table 3

proportion of bed-net usage or residual spray to 100% will effectively reduce the burden of malaria to the barest minimum. Since it is not realistic for the total human population to use a single control strategy, we simulate the impact of double control interventions on the infected population in Figure 5. We simulate the effect of treatment and its level of awareness on the total infected human and mosquito population in Figure 4(e) and Figure 4(f) respectively. The result shows that there is an insignificant effect of treatment on the total infected population. This result is similar to the one in Figure 3(c). Since bed-net usage and residual spray are preventive healthcare, the results from Figure 4 show that preventive healthcare is better than treatment healthcare. Thus, to reduce the burden of malaria, it is important to facilitate the use of preventive healthcare such as bed-net usage or residual spray among the populace.

In Figure 5 we simulate the effect of double control interventions with their respective level of awareness on the infected human and mosquito population. Overall, the result shows that combined control interventions reduce the total human and mosquito population faster than the use of single control intervention. Particularly, it is obvious from Figure 5(a) and Figure 5(b) that a combination of bed-net usage and the residual spray reduces the burden of malaria faster than any other double combined intervention. In Figure 6, we simulate the effect of all the control interventions with their respective level of awareness on the infected human and

mosquito population. It is noted that the result is alike to the one presented in Figure 5(a) and Figure 5(b). Thus, it is recommended that awareness and proportion of bed-net usage and residual spray should be priorities to mitigate the burden of malaria in the population. In addition, it is recommended that the proportion of bed-net usage and residual spray should be increased to at least 75% to eliminate malaria in the population.

5. CONCLUSIONS

Malaria is one of the deadliest diseases with highly challenging health issues in tropical regions. It is highly endemic in Sub-Saharan Africa, especially in an improvised and low-hygiene environment. Malaria is an infectious disease that is preventable, treatable, and curable in the human population, thus, understanding the influence of mitigation strategies such as bed-net usage, residual spray and treatment can help us inform public health policy. In this study, we developed a deterministic model to investigate the dynamical features of malaria in the population, and we assessed the impacts of control interventions with their level of awareness to effectively mitigate the burden of the disease. We obtained the malaria-free equilibrium and the endemic equilibrium of the model. The malaria-free equilibrium is shown to be locally asymptotically stable whenever the reproduction number \mathcal{R}_0 is less than unity, and unstable otherwise. Epidemiologically, this result implies that malaria can be effectively controlled in the population whenever the reproduction number is less than unity if the initial sub-populations of the infected compartments of the model system (3) are small enough. In other words, malaria can be effectively controlled in the population if the control strategies implemented can reduce and maintain the reproduction number below unity. We obtained the endemic equilibrium of the model, and the criteria for the existence of the phenomenon of bifurcation are investigated. The model is said to undergo a backward bifurcation phenomenon when the critical value of $\mathcal{R}_0^c < \mathcal{R}_0 < 1$. The existence of the backward bifurcation phenomenon suggests that reducing reproduction number \mathcal{R}_0 below unity is not enough to eliminate malaria, thus, a combination of control strategies may be needed to control the spread of malaria in the population. A sensitivity analysis was performed to examine the effect of bed-net usage, residual spray, and treatment on the reproduction number. The result shows that increase in any of the control interventions will decrease the reproduction number. Furthermore, the result shows that residual spray is the most

influential intervention in reducing the reproduction number as presented in Table 4. Following this result, we simulate the effect of control interventions and their level of awareness on the infected human and mosquito population, under three different scenarios. The overall result from the numerical simulations is that combining bed-net usage and residual spray as a preventive healthcare measure will reduce the burden of malaria faster. Particularly, results suggest that awareness and proportion of bed-net usage and residual spray should be priorities and increased to at least 75% for the possibilities of eliminating malaria. Thus, we recommend that malaria control programs should focus on increasing bed-net usage to reduce the bite of humans by an infected mosquito. In addition, the use of residual spray should be encouraged to reduce the mosquito population. All these can be achieved by increasing awareness about preventive care for malaria and increasing the distribution of bed-net and residual spray in regions where malaria is endemic.

CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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