Effect of Chemoprophylactic Treatment on the Dynamical Spread of Malaria.

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Abstract: In this paper, a mathematical model for the transmission of malaria is developed and analyzed. We establish the basic reproduction number R_O for the model. The analysis shows that the disease free equilibrium is globally asymptotically stable whenever the associated threshold quantity R_O is less than unity i.e. $R_O < 1$. Moreover, we show that there exists a unique endemic equilibrium whenever the associated threshold quantity Ro exceeds unity i.e. $R_O > 1$. The numerical analysis shows that as the treatment rate of the exposed class increases the population size of the exposed reduces as they move out of the class and the population of the infected class reduces while the population size of the recovered class increases, which means that treatment of exposed class reduces the number of the people that will progress into infectious class which thereby reduces the spread of the malaria.

Keyword: Disease free equilibrium, Reproduction number, Stability, Transmission

1. INTRODUCTION

Malaria is a deadly disease caused by Plasmodium parasite. About 3.2 billion people – almost half of the world`s population are at risk of malaria. In 2015, there were roughly 214 million malaria cases and an estimated 438000 malaria death with sub- Sahara Africa carrying disproportionately high share of the global malaria burden. In 2015, the region was home of 89% of malaria cases and 91% of malaria death. Young children, pregnant women, and non- immune travelers from malaria – free areas are the most vulnerable to the disease. [22, 24]. About 30,000 travellers from industrialized countries were reported to contract malaria each year and between 1-4% of travellers who acquired plasmodium falciparum malaria died [16]

There are four parasite species that cause malaria in humans which include Plasmodium falciparum, Plasmodium vivax, Plasmodium malariae, Plasmodium ovale. In recent years, some human cases of malaria have also occurred with Plasmodium knowlesi - a species that infects animals (monkey). Plasmodium falciparum is the most deadly and accounts for 80% of malaria cases and 90% off death [23, 13]. Malaria is transmitted among human by female mosquitoes of genus anopheles. Female mosquitoes take blood meals to carry out egg production and such blood meals are the link between the human and the mosquito host in the parasite life [6].

The symptoms of malaria appear seven days or more (usually 7- 15 days) after being bitten by infectious mosquito. The first symptoms include; fever, headache, chills and vomiting. Children with severe malaria frequently develop one or more of the following symptoms; severe anaemia, respiratory distress in relation to metabolic acidosis, or cerebral malaria and in adults, multi-organ involvement is also frequent [24]. However, malaria is preventable and curable. Malaria can be treated in just 48 hours yet it can cause total complication if the diagnosis and treatment are delayed. The prevention is usually by the use of insecticides treated bed nets, spraying with residual insecticides and the best available treatment, particularly for P.falciparum malaria is Artemisinin-based Combination Therapy (ACT). Travellers to high risk plasmodium falciparum endemic areas need effective chemoprophylaxis. This treatment "chemoprophylaxis" can also be given to an individual at or on exposure stage.

Several works have been done on malaria and its transmission dynamics. Huo and Qiu [12] presented the stability of a mathematical model of malaria transmission with relapse. It was shown that the disease free equilibrium is globally asymptotically stable if $R_O \leq 1$, and the system is uniformly persistence if $R_O > 1$. Abdullahi et.al [1] proposed a new model for the spread of malaria with emphasis on the effectiveness of drug. Similarly, in the study by Tumwiine et.al [20], they concluded that due to new births and immunity loss to malaria, the susceptible class will always be refilled and the disease becomes more endemic.

In this paper, we formulate a new model for the spread of malaria. We assume that a fraction of newly infected individuals enter into the exposed class (slow progressor) and the remaining ones enter directly into the infected class at a faster rate (fast progressor). We also incorporate treatment into the exposed class in view to determine its impact on the entire population for the spread of the malaria.

2. MODEL FORMULATION

The population size $N_h(t)$ of human is sub – divided into sub – classes of individuals who are Susceptible $S_h(t)$, Exposed $E_M(t)$, Infected $I_M(t)$, and Recovered $R_M(t)$, So that;

$$N_h(t) = S_h(t) + E_M(t) + I_M(t) + R_M(t) \quad (1)$$

Also, the population size $N_V(t)$ of the vectors (mosquitoes) is sub – divided into susceptible mosquitoes $S_V(t)$, Exposed $E_V(t)$ and Infectious mosquitoes $I_V(t)$. So that;

$$N_V(t) = S_V(t) + E_V(t) + I_V(t) \quad (2)$$

The susceptible population is increased by recruitment of individuals into the population (either by birth or migration at the rate π_h). The population decreases by infection following effective contact with infectious mosquito (at the rate λ_M) and natural death (at the rate μ).Where an individual recovered, at some point in time, he losses the immunity (at the rate ϕ) and then becomes susceptible again. Thus;

$$\frac{dS_h}{dt} = \pi_h - \lambda_M S_h - \mu S_h + \phi R_M \quad (3)$$

where

$$\lambda_M = \frac{\beta_M a I_V}{N_V} \quad (4)$$

Where β_M is the transmission probability from mosquito to human, provided that there is a contact between the human and mosquito and ``a`` is the number mosquito bite that one human has per unit time.

A fraction '' ε '' of newly infected individuals move to the exposed class (slow progressor) while the remaining fraction 1- ε moves to the infectious class (fast progressor). The exposed population declines due to progression to infections class (at the rate κ_M), treatment (at the rate τ_1) and natural death (at the rateμ).Thus;

$$\frac{dE_M}{dt} = \varepsilon\lambda_M S_h - (\kappa_M + \mu)E_M - \tau_1 E_M \quad (5)$$

The population of infected individual increases by fast progressor of newly infected individual, progression from exposed class (at the rate κ_M). The population declines due to treatment (at the rate τ_2), those that recovered (at the rate r), natural death (at the rate μ) and disease induced death (at the rate δ_{IM}). Thus,

$$\frac{dI_M}{dt} = (1-\varepsilon)\lambda_M S_h + \kappa_M E_M - (\tau_2 + r + \delta_{IM} + \mu)I_M \quad (6)$$

The population of the recovered is generated by the treatment of exposed and infected individuals (at rate τ_1 and τ_2). It also comprises of those that recovered (at the rate r). The population decreases due to individual that losses immunity (at rate ϕ) and natural death (at the rate μ). Then,

$$\frac{dR_M}{dt} = \tau_1 E_M + \tau_2 I_M + r I_M - (\phi + \mu)R_M \quad (7)$$

Susceptible mosquitoes (S_V) are generated at a constants rate π_V (recruitment rate) and acquire malaria infection following effective contact with human infected with malaria (at a rate λ_V) .Where the force of infection λ_V is given by;

$$\lambda_V = \beta_V b\left(\frac{E_M + \eta_1 I_M}{N_h}\right) \quad (8)$$

Where β_V is the transmission probability of infection from human to mosquito, b is the number of human bites one mosquito has per unit time, N_h is the total population of human, η_1 is the modification parameter comparing the transmissibility of infectious individuals in relationship to exposed individual. Newly infected mosquitoes move to exposed class and they are assumed to suffer natural death (at a rate μ_V). Hence,

$$\frac{dS_V}{dt} = \pi_V - \lambda_V S_V - \mu_V S_V \quad (9)$$

The exposed mosquito consists of newly infected mosquitoes and their population diminishes by progression to infectious class (at the rate σ_V) and natural death of the mosquito (at the rate μ_V). Therefore;

$$\frac{dE_V}{dt} = \lambda_V S_V - (\sigma_V + \mu_V)E_V \quad (10)$$

The infectious mosquito has those that progresses from exposed class and reduces by the natural death of the mosquito at the rate μ_V). Hence;

$$\frac{dI_V}{dt} = \sigma_V E_V - \mu_V I_V \qquad (11)$$

In summary, combining the above formulations and assumptions together, we have the following system of differential equations.

$$\frac{dS_h}{dt} = \pi_h - \lambda_M S_h - \mu S_h + \phi R_M \qquad (12)$$

$$\frac{dE_M}{dt} = \varepsilon\lambda_M S_h - L_1 E_M \qquad (13)$$

$$\frac{dI_M}{dt} = (1-\varepsilon)\lambda_M S_h + \kappa_M E_M - L_2 I_M \qquad (14)$$

$$\frac{dR_M}{dt} = \tau_1 E_M + L_3 I_M - L_4 R_M \qquad (15)$$

$$\frac{dS_V}{dt} = \pi_V - \lambda_V S_V - \mu_V S_V \qquad (16)$$

$$\frac{dE_V}{dt} = \lambda_V S_V - L_5 E_V \qquad (17)$$

$$\frac{dI_V}{dt} = \sigma_V E_V - \mu_V I_V \qquad (18)$$

Where;

$L_1 = \kappa_M + \mu + \tau_1$, $L_2 = \tau_2 + r + \delta_{IM} + \mu$,

$L_3 = \tau_2 + r$, $L_4 = \phi + \mu$, $L_5 = \sigma_V + \mu_V$

Where

$$\lambda_M = \frac{\beta_M a I_V}{N_V} \qquad (19)$$

$$\lambda_V = \beta_V b\left(\frac{E_M + \eta_1 I_M}{N_h}\right) \qquad (20)$$

The model extends earlier models [2, 17] by assuming that a fraction of newly infected individuals enter into the exposed class (slow progress), and the remaining fraction into the infected class (fast progress). The model includes the treatment of the exposed class. The table I & II below give the description of the variables and parameters used in the model.

3. ANALYSIS OF THE MODEL

Lemma 1: The close set $D = D_h \times D_V \subset R_+^7$ is positive invariant for the model equation (12)-(18) with non-negative initial conditions in R_+^7

Proof 1: Consider the biologically- feasible region $D = D_h x D_V \subset R_+^7$ with

$$D_h = \left\{(S_h, E_M, I_M, R_M)\varepsilon R_+^4 : N_h \le \frac{\pi_h}{\mu}\right\}$$

$$\& \; D_V = \left\{(S_V, E_V, I_V)\varepsilon R_+^3 : N_V \le \frac{\pi_V}{\mu_V}\right\}$$

We shall show that D is positive invariance (i.e. all solutions in D remain in D for all time t>0).The rate of change of the total population of human and mosquitoes by adding the model;

$$\frac{dN_h}{dt} = \pi_h - \mu N_h - \delta_{IM} I_M \text{ and } \frac{dN_V}{dt} = \pi_V - \mu_V N_V$$

where $N_h = S_h + E_M + I_M + R_M$ and $N_V = S_V + E_V + I_V$

It follows that;

$$\frac{dN_h}{dt} \le \pi_h - \mu N_h \text{ and } \frac{dN_V}{dt} \le \pi_V - \mu_V N_V$$

A standard comparison theorem [14] can be used to show that $N_h(t) \le N_h(0)e^{-\mu t} + \frac{\pi_h}{\mu}\left(1 - e^{-\mu t}\right)$

and $N_V(t) \le N_V(0)e^{-\mu_V t} + \frac{\pi_V}{\mu_V}\left(1 - e^{-\mu_V t}\right)$.

In particular $N_h(t) \le \frac{\pi_h}{\mu}$ and $N_V(t) \le \frac{\pi_V}{\mu_V}$,

If $N_h(0) \le \frac{\pi_h}{\mu}$ and $N_V(0) \le \frac{\pi_V}{\mu}$ .Therefore, all solution of the model with initial condition in D remains there for $t > 0$. This implies that D is positively – invariant. In this region, the model can

be considered as been epidemiologically and mathematically well posed.

3.1. Disease Free Equilibrium (DFE)

The model equation (12) - (18) has a disease free equilibrium (DFE), which is gotten by setting all the right hand sides of the equations in the model to zero, which is given by ;

$$E_0 = \left(S_h, E_M, I_M, R_M, S_V, E_V, I_V\right)$$

$$= \left(\frac{\pi_h}{\mu}, 0, 0, 0, \frac{\pi_V}{\mu_V}, 0, 0\right) \qquad (20)$$

Using next generation matrix [21], the non-negative matrix F (new infection terms) and non-singular matrix V (other transferring terms) of the model are given, respectively by;

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\varepsilon\beta_M a\pi_h\mu_V}{\mu\pi_V} \\ 0 & 0 & 0 & 0 & \frac{(1-\varepsilon)\beta_M a\pi_h\mu_V}{\mu\pi_V} \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_V b\pi_V\mu}{\mu_V\pi_h} & \frac{\beta_V b\eta_1\pi_V\mu}{\mu_V\pi_h} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} L_1 & 0 & 0 & 0 & 0 \\ -\kappa_M & L_2 & 0 & 0 & 0 \\ -\tau_1 & -L_3 & L_4 & 0 & 0 \\ 0 & 0 & 0 & L_5 & 0 \\ 0 & 0 & 0 & -\sigma_V & \mu_V \end{bmatrix}$$

The associated reproduction number Ro for malaria model is given by $R_O = \rho\left(FV^{-1}\right)$, where ρ is the spectral radius of the dominant eigen value of the next generation matrix FV^{-1}. Hence;

$$R_0 = \frac{\sqrt{L_1L_2L_5\mu_V ab\sigma_V\beta_M\beta_V(\varepsilon L_2 + \varepsilon\eta_1\kappa_M + \eta_1L_1 - \eta_1L_1\varepsilon)}}{L_1L_2L_5\mu_V} \qquad (21)$$

The threshold quantity R_O is the basic reproduction number of the model equation above, which is the average number of new case of an infection caused by one typical infected individual/mosquito in a population consisting of susceptible only. [9]

3.2. Global stability of the Disease Free Equilibrium

Theorem 2:

The disease free equilibrium of malaria model given by (20) is globally asymptotically stable if $R_O < 1$,

Proof:

We will use comparison theorem [14] to prove the global stability.

The rate of change of variables representing the infected components of equation (12) – (18) can be re-written as;

$$\begin{pmatrix} \frac{dE_M}{dt} \\ \frac{dI_M}{dt} \\ \frac{dE_V}{dt} \\ \frac{dI_V}{dt} \end{pmatrix} = (F - V)\begin{pmatrix} E_M \\ I_M \\ E_V \\ I_V \end{pmatrix} - \begin{pmatrix} \varepsilon\lambda_M S_h \\ (1-\varepsilon)\lambda_M S_h \\ \lambda_V S_V \\ 0 \end{pmatrix} \qquad (22)$$

Then,

$$\begin{pmatrix} \frac{dE_M}{dt} \\ \frac{dI_M}{dt} \\ \frac{dE_V}{dt} \\ \frac{dI_V}{dt} \end{pmatrix} \le (F - V)\begin{pmatrix} E_M \\ I_M \\ E_V \\ I_V \end{pmatrix}$$

According to Castillo - Chavez et. al [4] and Driesssche and Watmough[21], all the eigen values of the matrix F – V have negative real parts. It follows that the linearized differential inequality system above is stable whenever $R_o < 1$ .Consequently, by comparison theorem [14]. We have that $E_M = I_M = R_M = E_V = I_V = 0$, $\to (0,0,0,0,0)$ as $t \to \infty$ .Substituting $E_M = I_M = R_M = E_V = I_V = 0$ into (12),we have that $S_h(t) \to S_h(0)$ and $S_V(t) \to S_V(0)$ as $t \to \infty$. Hence, we have a positive invariant region. It follows that disease free equilibrium is globally asymptotically stable whenever $R_0 < 1$.

3.3. Existence of endemic equilibrium point of malaria model

Here, we try to find the condition for the existence of equilibrium for which malaria is endemic in the population. Consider the malaria model equation (12) – (18)

Let the associated reproduction number of the model equation (12) - (18) above be R_o^1 given by;

$$R_0 = \frac{\sqrt{L_1L_2L_5\mu_V ab\sigma_V\beta_M\beta_V(\varepsilon L_2 + \varepsilon\eta_1\kappa_M + \eta_1L_1 - \eta_1L_1\varepsilon)}}{L_1L_2L_5\mu_V} \quad (23)$$

Let $E_H^{**} = \left(S_h^{**}, E_M^{**}, I_M^{**}, R_M^{**}, S_V^{**}, E_V^{**}, I_V^{**}\right)$ represents any arbitrary endemic equilibrium of model equations (12) - (18).

Let λ_V and λ_M at the endemic steady state be denoted by λ_V^{**} and λ_M^{**} given by;

$$\lambda_M^{**} = \frac{\beta_M a I_V^{**}}{N_V^{**}} \quad (24)$$

$$\lambda_V^{**} = \beta_V b\left(\frac{E_M^{**} + \eta_1 I_M^{**}}{N_h^{**}}\right) \quad (25)$$

where $N_h^{**} = S_h^{**} + E_M^{**} + I_M^{**} + R_M^{**}$

and $N_V^{**} = S_V^{**} + E_V^{**} + I_V^{**}$

Solving the equations of the model at steady state and re - writing the values of E_M, I_M, R_M in terms of $\lambda_M^{**}S_h^{**}$ and re – writing the values E_V, I_V in terms of $\lambda_V^{**}S_V^{**}$, we have;

$$S^{**} = \frac{\pi_h + R_M^{**}}{\lambda_M^{**} + \mu} \quad (26)$$

$$E_M^{**} = \frac{\varepsilon\lambda_M^{**}S_h^{**}}{L_1} = P_1\lambda_M^{**}S_h^{**} \quad (27)$$

$$I_M^{**} = \frac{(1-\varepsilon)\lambda_M S_h^{**}L_1 + \kappa_M\varepsilon\lambda_M^{**}S_h^{**}}{L_1L_2}$$

$$I_M^{**} = P_2\lambda_M^{**}S_h^{**} \quad (28)$$

$$R_M^{**} = \frac{\tau_1\varepsilon\lambda_M^{**}S_h^{**}L_2 + L_3\left[(1-\varepsilon)\lambda_M^{**}S_h^{**}L_1 + \kappa_M\varepsilon\lambda_M^{**}S_h^{**}\right]}{L_1L_2L_4}$$

$$R_M^{**} = P_3\lambda_M^{**}S_h^{**} \quad (29)$$

$$S_V^{**} = \frac{\pi_V}{\lambda_V^{**} + \mu_V} \quad (30)$$

$$E_V^{**} = \frac{\lambda_V^{**}S_V^{**}}{L_5} = P_4\lambda_V^{**}S_V^{**} \quad (31)$$

$$I_V^{**} = \frac{\sigma_V\lambda_V^{**}S_V^{**}}{L_5\mu_V} = P_5\lambda_V^{**}S_V^{**} \quad (32)$$

Where; $P_1 = \frac{\varepsilon}{L_1}$, $P_2 = \frac{(1-\varepsilon)L_1 + \kappa_M\varepsilon}{L_1L_2}$,

$$P_3 = \frac{\tau_1\varepsilon L_2 + L_3\left[(1-\varepsilon)L_1 + \kappa_M\varepsilon\right]}{L_1L_2L_4}, \quad P_4 = \frac{1}{L_5},$$

$$P_5 = \frac{\sigma_V}{L_5\mu_V}$$

Substituting (27) - (29) into (25) gives;

$$\lambda_V^{**}\left(1 + (P_1 + P_2 + P_3)\lambda_M^{**}\right) = \beta_V b(P_1 + \eta_1P_2)\lambda_M^{**}$$

Let $P_1 + P_2 + P_3 = P_7$

$$\lambda_V^{**} = \frac{\beta_V b(P_1 + \eta_1 P_2)\lambda_M^{**}}{1 + P_7\lambda_M^{**}} \qquad (33)$$

Substituting (31) & (32) into (24) gives;

$$\lambda_M^{**}\left(1 + (P_4 + P_5)\lambda_V^{**}\right) = \beta_M a P_5 \lambda_V^{**}$$

Let $P_4 + P_5 = P_6$

$$\lambda_M^{**}\left(1 + P_6\lambda_V^{**}\right) = \beta_M a P_5 \lambda_V^{**} \qquad (34)$$

Putting (33) into (34), we have;

$$\left(1 + P_6\lambda_V^{**}\right)\left(1 + P_7\lambda_M^{**}\right) = \beta_M\beta_V abP_5(P_1 + \eta_1 P_2)$$

$$\left(1 + P_6\lambda_V^{**}\right)\left(1 + P_7\lambda_M^{**}\right) = R_o^{\,2}$$

Hence $1 + P_6\lambda_V^{**} = R_o^{\,2}$ or $1 + P_7\lambda_M^{**} = R_o^{\,2}$

So, $\lambda_V^{**} = \frac{R_o^{\,2} - 1}{P_6} > 0$, whenever $R_o > 1$

or $\lambda_M^{**} = \frac{R_o^{\,2} - 1}{P_7} > 0$, whenever $R_o > 1$

Therefore, there exists an endemic equilibrium whenever $R_o > 1$

4. NUMERICAL SIMULATION

In this section, we carry out numerical simulation of the model system (12) - (18).We take the following initial conditions;

$S_h(0) = 8000$ $E_M(0) = 6500$, $I_M(0) = 5000$, $R_M(0) = 3000$, $S_V(0) = 700$, $E_V(0) = 500$, $I_V(0) = 400$. The values of the parameters used are taken from literatures. The table of the parameter values used is as shown table II

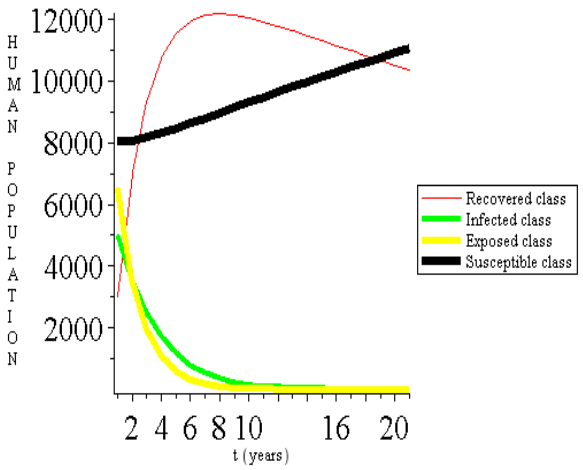

Fig. 1: Graph of the total human population against time

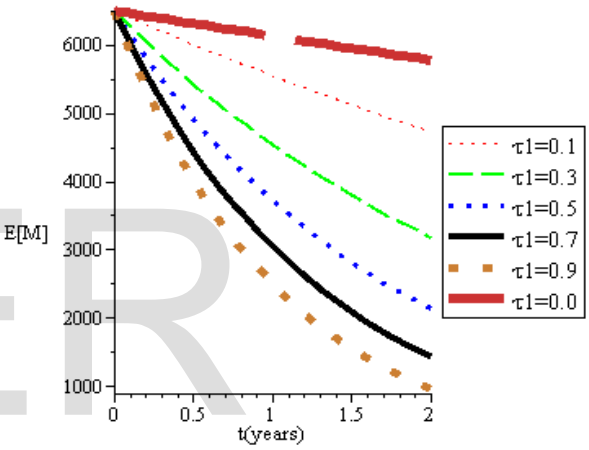

Fig 2: Graph of the exposed class against time at different values of treatment rate

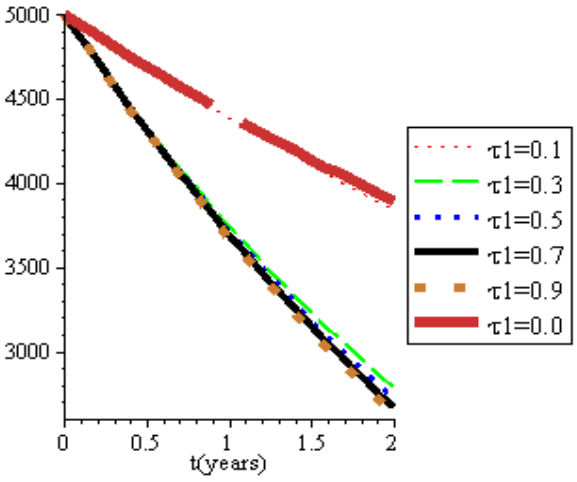

Fig.3: Graph of infected class against time at different treatment rate.

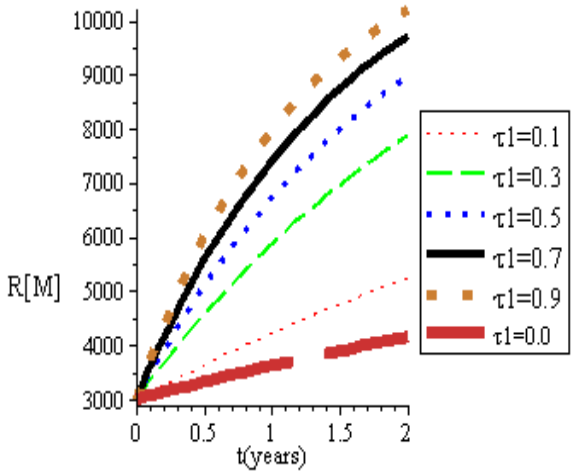

Fig 4: Graph of the Recovered class against time at different treatment rate.

5. DISCUSSION OF RESULTS

From the fig. 1, the susceptible human population increases with time due to the loss of the immunity of the recovered class which returns to the susceptible class. The population of the infected also decreases. Moreover, the population of the exposed individual reduces because they leave the class and there is increase in the number of people that recovered because of the treatment, which later decreases due to loss of immunity.

Fig.2 shows that in absence of treatment i.e. $\tau_1 = 0.0$, high number of people would be exposed to malaria disease. It shows that treatment has a pronounced effect on the exposed class i.e. as we increase the treatment, the population of the exposed class decreases because the exposed individual will leave the class to join other class.

Fig. 3 shows that there is reduction in the number of individuals that are infected but more noticeable when the treatment rate increased to 0.3. This implies that treatment of the exposed class reduces the number of individuals that will be infectious.

Fig. 4 shows the effect of treatment on the recovered individuals. It shows that when the treatment of the exposed individual increases, it also increases the recovered individual, which means early treatment of the exposed individuals plays a vital role in the dynamical spread of malaria, i.e. chemoprophylaxis prevents infection from developing into clinical disease.

Table I: Table of variables.

VARIABLE	DESCRIPTION
$S_h(t)$	Susceptible individuals
$E_M(t)$	Exposed individuals
$I_M(t)$	Infected individuals
$R_M(t)$	Recovered individuals
$S_V(t)$	Susceptible vectors (mosquitoes)
$E_V(t)$	Exposed vectors (mosquitoes)
$I_V(t)$	Infected vectors (mosquitoes)

Table II: Table of parameters and their values.

PAR.	DESCRIPTION	VALUES	REF.
π_h	Recruitment rate into the human population	0.000051	[5]
μ	Natural death rate of human	0.0004	[25]
ϕ	Rate of loss of immunity	0.0146	[7]
ε	Slow progressor	0.6	Assumed
η	Modification parameter	0.01	Assumed
κ_M	Progression rate for human	0.0588	[3,15]
τ_1	Treatment rate for exposed individuals	Variable	Variable
τ_2	Treatment rate for infected individuals	0.143	Assumed
r	Recovery rate	0.005	[8]
δ_{IM}	Death due to disease	0.05	[18]
π_V	Recruitment rate of the vectors(mosquitoes)	0.071	[3,12]
μ_V	Natural death rate of mosquitoes	0.04	[8]
σ_V	Progression rate for mosquitoes	0.1	[7]
β_M	Transmission probability from mosquito to human	0.02	[7]
β_V	Transmission probability from human to mosquito	0.09	[12,18]
a	Number of mosquito bites per unit time	0.5	Assumed
b	Number of human bitten per unit time	0.6	[7]
λ_M	Force of infection from mosquito to human		
λ_V	Force of infection from human to mosquito		

6. CONCLUSION

We presented and analyzed a mathematical model for the transmission of malaria. We investigated the impact of the treatment on the transmission of the malaria. We obtained the basic reproduction number R_o, which is the average number of new cases of an infection caused by one typical infected individual/ mosquito in a population consisting of susceptible only. We proved that the disease free equilibrium is globally asymptotically stable whenever $R_o < 1$. Moreover, we tried to show that there exists a unique endemic equilibrium whenever $R_o > 1$. In conclusion, the numerical simulation shows that as treatment rate of the exposed class increases, the population size of the exposed individuals reduces while that of the recovered class increases. Therefore, the study suggests that people that are exposed to malaria should get treatment on time so as to reduce the number of people that will progress into infectious class which thereby reduces the spread of malaria. Also new travellers in malaria endemic region should take chemoprophylactic treatment in order to curb the transmission of malaria.

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