Fahad Al Basir*, Arnab Banerjee and Santanu Ray

Exploring the effects of awareness and time delay in controlling malaria disease propagation

Abstract: In this article, a mathematical model has been derived for studying the dynamics of malaria disease and the influence of awareness-based interventions, for control of the same, that depend on 'level of awareness'. We have assumed the disease transmission rates from vector to human and from human to vector, as decreasing functions of 'level of awareness'. The effect of insecticides for controlling the mosquito population is influenced by the level of awareness, modelled using a saturated term. Organizing any awareness campaign takes time. Therefore a time delay has been incorporated in the model. Some basic mathematical properties such as nonnegativity and boundedness of solutions, feasibility and stability of equilibria have been analysed. The basic reproduction number is derived which depends on media coverage. We found two equilibria of the model namely the disease-free and endemic equilibrium. Disease-free equilibrium is stable if basic reproduction number ($\mathcal{R}_0$) is less than unity ($\mathcal{R}_0 < 1$). Stability switches occur through Hopf bifurcation when time delay crosses a critical value. Numerical simulations confirm the main results. It has been established that awareness campaign in the form of using different control measures can lead to eradication of malaria.

Keywords: basic reproduction number ($\mathcal{R}_0$); disease awareness; Hopf bifurcation; media campaign; numerical stability analysis; time delay.

1 Introduction

In most of the poor, rural and semirural areas of the world malaria is one of the major causes of mortality [1]. About 231 million cases of malaria were reported in the year 2017 alone by World Health Organization (WHO), occurring in 106 countries, of which, around 81% occurred in African Regions resulting in about 91% death of the infected persons [2]. In 2018, globally there were 228 million cases of which about 91% was in African continent. There was an estimated 405,000 deaths, around 67% (272,000) of which were in children aged under five years. The cases reported in the south-east Asian region was around 3.4% of the total reported [3].

Among the different types of protozoan parasite *Plasmodium*, *Plasmodium falciparum* and *Plasmodium vivax* are the most common infection causing types, in Africa and South East Asia [4]. The digenetic parasite completes its life cycle via two hosts—human and—female *Aenopheles* sp. mosquito. Gametocytes of the parasite are ingested by the vector while feeding on an infected human. This gametocyte undergoes sporogonic cycle inside the mosquito and is then transmitted or injected to a healthy individual (during uptake of blood meal) in the form of sporozoites (the infective stage) [5, 6]. Effective control of the infection include controlling the spread of disease through various methodologies at different levels or stages of life cycles of both parasite and vector.

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Over the past decade, several control interventions have been explored to reduce transmission of this disease. These measures include, but are not limited to, insecticide-treated nets, indoor spraying, intermittent preventive treatment in pregnant women and infants, larval control and other vector control mediations. Most of these control strategies have proven to be some of the most effective since malaria typically affects rural and poor human populations [7].

One major obstacle in implementing control strategies for any kind of disease regulation is public adherence to their persisting beliefs [8]. This is particularly common in tropical and subtropical areas of countries where malaria is more prevalent. Various misconceptions regarding the disease prevails among the common mass. Well organized dissipation of correct information might be able to urge people in modifying their behavior to minimize the risk of infection thus helping in reshaping the future course of an epidemic [9–11]. A lot of effort is made to spread awareness among the people to prevent vector borne diseases. In this regard, the US president designated April 25 as the Malaria Awareness Day to induce awareness among people. In India, female health volunteers are involved for making people aware of the disease and its control methodology [12, 13]. Exploring proper control strategies is of paramount importance in order to prevent the spread of this disease.

Simple formulation of control strategies alone is not enough to control the spread of any disease. Proper awareness build-up is also required in order for successful implementation of the same. Increased awareness (through media campaigns and other methods) can be reflected in decreasing rate of transmission and can also be attributed to increased mosquito-population-regulation procedures. Aware people not only enforce preventive measures themselves, but also make efforts to reduce mosquito breeding sites [14, 15]. These control or preventive measures include simplistic methods like the use of bed nets while sleeping, mosquito repellents, etc., or others like larvae destruction with larvicides, use of larvivorous fish, genetically manipulated insect vectors and so on [16–18].

Mathematical models have been in use for understanding disease dynamics and effects of different intervention tactics on such subtleties [19–21]. Several authors have previously used models showing dynamics of malaria transmission considering host and vector population. Host and vector populations were divided respectively into three (susceptible, infectious/infected and recovered) and two (susceptible and infective) subclasses [4, 22–24]. In Ref. [7], authors have additionally considered two subpopulations—susceptible and infective, along with vector bias effect and also optimal effect of different control measures.

Models for malaria control include a wide array of different methodologies ranging from various vector control strategies to comparing effectiveness of different disease control procedures. For example, use of medicated insect nets and spraying [25] to the administration of different vaccines exerting control [26]. In Ref. [27], a deterministic model was developed to assess the effects of recurrent malaria – reinfection and relapse on the transmission dynamics of the disease. An agent-based model was introduced using a susceptible-infectious-susceptible (SIS) system of humans and mosquitoes to predict malaria epidemiological scenarios [28]. A climate-based malaria model which involves both vertical and horizontal transmissions of the engineered Serratia AS1 bacteria in mosquito population was studied in Ref. [29]. Ref. [30] estimated the degree of transmission incorporating a loss of innate immunity in the infected individuals. This was followed [33, 31] by including the relapse dynamics of the disease among the infected individuals. Interdependencies among the parasite, host and their environment was studied earlier [32]. Trend analysis of the disease from field survey and intervention in disease spread was also investigated upon [33].

Dissemination of research is a key regulator toward growth of awareness among individuals—imbibing them with working knowledge of managing and possibly eradicating the disease [34]. Knowledge gained through awareness campaigns can decay over time, and the delay in acquiring information and implementing the same can affect the transmission dynamics of any infectious disease including malaria. Furthermore, delay can also come into effect during transmission of the disease, expression in the host and so on. Previous models have considered [35] scenarios of explicit transmission delay (incubation in host and vector) or for the transmission of two varieties of malaria with time lags [36]. Transmission model considering two different delays was studied by Ref. [37]. An SEIS deterministic differential equation model considered the time lapse between egg laying and adult mosquito eclosion [38]. Time delay also arises in controlling malaria with awareness, for example, there may be delay in reporting infected cases to the health organization. This fact

should be taken into consideration for model formulation suitably incorporating time delays. Models of this kind have been considered in previous literature [39–42].

In Ref. [8], a model for controlling vector-borne disease through media awareness campaign was proposed which assumed that arrangement of such programs should be in proportion to number of infected people. Furthermore, the human population were divided into three groups, viz. susceptible, infected and aware population. A separate population $M(t)$ representing the number of media campaign was taken into account to check the importance of media campaigns. But authors have considered disease transmission rates as constant. Transmission rated, from infective vector to human and infected human to vector, should depend on the level of awareness [27, 28, 40, 43].

In this article, we assumed the malaria disease transmission rates as function of 'level of awareness'. Also the control measures are assumed as awareness induced saturated functions. Also, level of awareness has been assumed as a model population. Both human and mosquito populations are divided into two sub-populations namely, susceptible and infected humans and susceptible and infective vectors. Number of infected cases, global factors (namely TV, radio, social media) etc. can also increase the level awareness. Infection transmission rates are assumed as function of level of awareness, $A(t)$. Also, level of awareness in form of control increases the death rates of mosquito vector. Finally, a time delay has been considered, signifying the time taken in reporting of infected cases or implementing the control measures.

The article is organized as follows. In the next section, delay model for malaria disease dynamics and possible control using public awareness has been proposed. Some preliminary results namely nonnegativity, boundedness, existence of equilibria and characteristic equation of the model have been provided in Section 3. Stability analysis of equilibria have been carried out, with possible occurrence of Hopf bifurcation, using qualitative theory (Sections 4 and 5). Analytical results are confirmed through numerical simulations (Section 6). Finally, a discussion concludes the paper with a future outline of the present work (Section 7).

2 Mathematical model derivation

We formulate the mathematical model using the following assumptions. We have followed the mathematical model proposed by Agusto et al. [24] and derive the model for the effects awareness and time delay on the transmission of malaria infection.

We have considered human and mosquito population. The human population is divided into two classes namely the susceptible $S_h(t)$ and infected $I_h(t)$ human. The mosquito population is also divided into two classes, susceptible $S_v(t)$ and infected $I_v(t)$ mosquito. We have also considered level of awareness $A(t)$ as a separate population.

We have used *SIS* type model to capture the dynamics of malaria transmission in a human population as immunity to malaria is not fully acquired and it decays with time. Without new contacts individuals may lose immune memory and may become infected again. For a mosquito population, we have used *SI* type model assuming that mosquito does not recuperate from malaria parasites and also neither the malaria parasites do not damage the mosquito population and nor recover from infection.

The host population is divided into two compartments, susceptible (S_h) and infected (I_h), with a total population (N_h) given by $N_h = S_h + I_h$. Analogously, the vector population is divided into two compartments, susceptible (S_v) and infected (I_v), with a total population (N_v) given by $N_v = S_v + I_v$. All newborn individuals are assumed to be susceptible and no infected individuals are assumed to come from outside of the community.

Let λ_h be the constant growth of human population either by birth or immigration. The whole human population is subject to the natural mortality with a constant rate d; λ_v be the constant growth rate of susceptible mosquito population.

The force of infections for susceptible humans (α) and susceptible vectors (β) are given by

$$\alpha = \frac{p_1 \alpha_0}{N_h}, \quad \beta = \frac{p_2 \beta_0}{N_h} \tag{1}$$

where p_1 and p_2 are the transmission probability per bite from infectious mosquitoes to humans and from infected humans to mosquitoes, respectively, and α_0 and β_0 are the maximum transmission in absence of awareness. All awareness induced interventions affect the spread of malaria from being aware that this disease is actually troubling people. It is assumed that being aware people will take all necessary protections for personal defense and successfully escape the chances of getting infected.

In the modelling process, it is assumed that media campaigns increase the level of awareness regarding personal protection as well as the way of controlling mosquito population.

We denote by $A(t)$ the level of population awareness of the disease, its dynamics can be assumed by the following differential equation

$$\frac{dA}{dt} = \omega_0 + \omega I_h - \theta A.$$

(2)

Here, we assume that awareness level increases at a rate ω_0 from some global sources such as radio, TV campaign [10] and the level of awareness decreases at a rate θ due to fading of memory.

The disease transmission rate from infective mosquito to susceptible human will decrease with increase in the level of awareness [40]. Thus we modify α as a function of the level of awareness,

$$\alpha = \frac{p_1 \alpha_0}{N_h (1 + A)}$$

(3)

Similarly, rate of transmission β from infected human to susceptible mosquito is modified as follows,

$$\beta = \frac{p_2 \beta_0}{N_h (1 + A)} .$$

(4)

Increasing the level of awareness, people will use insecticides to kill mosquitoes, modeled via the term $\gamma(A)S_v$ and $\gamma(A)I_v$ and $\gamma(A)$ is defined as

$$\gamma(A) = \frac{\gamma_0 A}{1 + A} ,$$

(5)

where γ_0 is the maximum rate of insecticide usage. Noticing the fact that for lower levels of awareness the use of insecticide will be linear, but if the awareness is high, it saturates at some level, because when everyone is already aware, the use of insecticide cannot increase further [43].

Further, the constants r and δ denote the recovery rate and disease-induced death rate of human population. For mosquito population, μ denote the natural death rate.

We have assumed that there is a time delay in organizing the awareness campaign due to the delay in reporting the number of infected human in health organization. Here, $\tau > 0$ is taken as the time delay parameter.

With the above assumptions, the following mathematical model is derived:

$$\frac{dS_h}{dt} = \lambda_h - \alpha(A)S_h I_v - dS_h + rI_h ,$$

$$\frac{dI_h}{dt} = \alpha(A)S_h I_v - (d + \delta)I_h - rI_h ,$$

$$\frac{dS_v}{dt} = \lambda_v - \beta(A)I_h S_v - \mu S_v - \gamma(A)S_v ,$$

$$\frac{dI_v}{dt} = \beta(A)I_h S_v - \mu I_v - \gamma(A)I_v ,$$

$$\frac{dA}{dt} = \omega_0 + \omega I_h(t - \tau) - \theta A.$$

(6)

Let C denote the Banach space of continuous functions $\phi : [-\tau, 0] \to \mathbb{R}_+^5$ equipped with the sup-norm,

$$\|\phi\| = \sup_{-\tau \le \zeta \le 0} \{|\phi_1(\zeta)|, |\phi_2(\zeta)|, |\phi_3(\zeta)|, |\phi_4(\zeta)|, |\phi_5(\zeta)|\} ,$$

Table 1: Biological meanings of variables/parameter used in the model [23, 24].

Variables/parameters	Descriptions	Values
S_h	Number of susceptible human	—
I_h	Number of infected human	—
S_v	Number of susceptible mosquito	—
I_v	Number of infected mosquito	—
A	Level of awareness on malaria	—
α_0	Disease transmission from infected mosquito to susceptible human	0.0005
β_0	Infection rate from infected human to susceptible mosquito	0.0005
λ_h	Recruitment rate of susceptible human	40
λ_v	Recruitment rate of susceptible mosquito	1000
p_1 Probability of disease transmission from mosquito	1	
p_2 Probability of disease transmission from human to mosquito	1	
μ	Natural death rate of mosquito	0.12
r	Recovery rate of infected human due to medication	0.025
d	Natural death rate of human	0.002
δ	Disease-induced death rate for human population	0.001

where, $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5) \in C([-\tau, 0], \mathbb{R}^5)$. For biological reasons, populations always have non-negative values if they survive, therefore, the initial function for model (6) is taken as below:

$$S_h(\zeta) = \phi_1(\zeta), \ I_h(\zeta) = \phi_2(\zeta), \ S_v(\zeta) = \phi_3(\zeta), \ I_v(\zeta) = \phi_4(\zeta),$$
$$M(\zeta) = \phi_5(\zeta), \ \text{and} \ \phi_i(\zeta) \geq 0, \ \zeta \in [-\tau, 0], \ \phi_i(0) > 0, \ i = 1, 2, 3, 4, 5. \tag{7}$$

In the following section, the system (6) will be analyzed with qualitative theories using initial conditions (7).

3 Preliminary results

Before proceeding with the analysis of model (6), we have to established some of its properties.

3.1 Non-negativity of solutions

From the fundamental theory of functional differential equations [44], it can be shown that the solution $(S_h(t), I_h(t), S_v(t), I_v(t), A(t))^T$ of model (6) with the initial condition (7) exists and is unique on $[0, +\infty)$.

Theorem 1. *Every solution of* (6) *with initial conditions* (7) *are non-negative for all $t > 0$.*

Proof. Using the method of steps, we show the non-negativity of solutions of system (6).

Considering the first equation of system (2), we can write,

$$\frac{S_h}{dt} \geq -(\alpha(A)S_h I_v + dS_h)$$

for some constant $T > 0$ and for all $t \in (0, T]$. This implies,

$$S_h(t) = S_h(0)\exp\left[-\left(\int_0^t (\alpha(A)S_h(\xi)I_v(\xi) + dS_h(\xi))d\xi\right)\right].$$

Using the standard comparison principle, we see that $S_h(t) \geq 0$ for all $t \in (0, T]$. Similarly, the non-negativity of the population I_h, S_v, I_v and A can be shown for all $t \in (0, T]$.

The above argument can be repeated to deduce the non-negativity of S_h, I_h, S_v, I_v and A on the interval $t \in (T, 2T]$ and so on the successive intervals $t \in (nT, (n+1)T]$, $n = 2, 3, \ldots$ to include all positive times. $\quad\square$

3.2 Boundedness

To ensure the model remains biologically plausible, all model populations have to remain bounded during their time evolution.

Theorem 2. *Every solution of* (6) *with initial conditions* (7) *are bounded in the region defined by the set,*

$$\mathbb{B} = \left\{ (S_h, I_h, S_v, I_v, A) \in C\left([-\tau, 0], \mathbb{R}^5\right) : 0 \le S_h + I_h \le \frac{\lambda_h}{d}, \right.$$

$$\left. 0 \le S_v + I_v \le \frac{\lambda_v}{\mu + \gamma_0}, \quad 0 \le A \le \frac{\omega_0 d + \omega\lambda_h}{d\theta} \right\}. \tag{8}$$

Proof. To prove the boundedness of $S_h(t)$ and $I_h(t)$, let

$$F(t) = S_h(t) + I_h(t)$$

Then,

$$\frac{dF(t)}{dt} = \frac{dS_h}{dt} + \frac{dI_h}{dt}$$
$$< \lambda_h - d(S_h + I_h)$$

Using the standard comparison principle, we get

$$\lim_{t \to \infty} \sup F(t) \le \frac{\lambda_h}{d}. \tag{9}$$

Hence, the $S_h(t)$ and $I_h(t)$ are bounded for all $t > 0$.

$$\lim_{t \to \infty} \sup S_v(t) + I_v(t) \le \frac{\lambda_v}{\mu + \gamma_0}. \tag{10}$$

Using the bound of I_h, the last equation of (6),

$$\frac{dA}{dt} \le \omega_0 + \frac{\omega\lambda_h}{d} - \theta A,$$

which gives

$$\lim_{t \to \infty} \sup A(t) \le \frac{\omega_0 d + \omega\lambda_h}{d\theta}. \tag{11}$$

Finally, using (9)–(11), we obtain the set $\mathbb{B}$ as given in (6). $\quad\square$

3.3 Characteristic equation

In this section, the characteristic equation at any equilibrium point is determined for the local stability of the delayed system (6). Linearizing the system (6) at any equilibria $E(S_h, I_h, S_v, I_v, A)$, we get

$$\frac{dX}{dt} = JX(t) + KX(t - \tau), \tag{12}$$

where $J = [J_{ij}]$, $K = [K_{ij}]$ are the following 5×5 matrices.

$$J = \left[J_{ij}\right]_{5\times5} =$$

$$\begin{bmatrix} -\alpha I_v - d & r & 0 & -\alpha S_h & -\alpha' S_h I_v \\ \alpha I_v & -r - \delta - d & 0 & -\alpha S_h & \alpha' S_h I_v \\ 0 & -\beta S_v & -(\mu + \gamma + \beta I_h) & 0 & -\beta' S_v I_h - \gamma' S_v \\ 0 & \beta S_v & \beta I_h & -(\mu + \gamma) & \beta' S_v I_h - \gamma' I_v \\ 0 & 0 & 0 & 0 & -\theta \end{bmatrix},$$

$$K_{ij} = \left[K_{ij}\right]_{5\times5} = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \omega & 0 & 0 & 0 \end{bmatrix}.$$

In the above matrix,

$$\alpha' = -\frac{\alpha_0}{(1+A)^2}, \quad \beta' = -\frac{\beta_0}{(1+A)^2}, \quad \gamma' = \frac{\gamma_0}{(1+A)^2}.$$

The characteristic equation of system (6) can be obtained using

$$\triangle(\xi) = \left|\xi I - J - e^{-\xi\tau} K\right| = 0, (I \text{ is the } 5\times5 \text{ identity matrix}),$$

which gives the following equation

$$\psi(\xi, \tau) = \xi^5 + \sigma_1\xi^4 + \sigma_2\xi^3 + \sigma_3\xi^2 + \sigma_4\xi + \sigma_5 \\ e^{-\xi\tau}(\sigma_6\xi^3 + \sigma_7\xi^2 + \sigma_8\xi + \sigma_9) = 0. \tag{13}$$

where $\sigma_i, i = 1, 2, \ldots, 9$ are given in Appendix.

3.4 Existence of equilibria

There are three possible equilibria for system (6), namely disease-free equilibrium, $E_0\left(\frac{\lambda_h}{d}, 0, \frac{\lambda_v}{\mu}, 0, \frac{\omega_0}{\theta}\right)$, and the endemic equilibrium, $E^\star = (S_h^\star, I_h^\star, S_v^\star, I_v^\star, A^\star)$, where

$$S_h = \frac{\lambda_h + rI_h^\star}{\alpha^\star I_v^\star + d}, \quad A_h^\star = \frac{\omega_0 + \omega I_h^\star}{\theta}, \quad S_v^\star = \frac{\lambda_v}{\mu + \gamma^\star + \beta^\star I_h^\star},$$

$$I_v^\star = \frac{\beta^\star I_h^\star S_v^\star}{\mu + \gamma^{\star\star}}, \quad I_h^\star = \frac{\alpha^\star \lambda_v \beta^\star \lambda_h - d(d + \delta + r)(\mu + \gamma^\star)^2}{(d + \gamma^\star)\alpha^\star \lambda_v \beta^\star + d(d + \delta + r)(\mu + \gamma^\star)}.$$

Biologically feasible endemic equilibrium exists if the following condition holds:

$$\alpha^\star \beta^\star \lambda_v \lambda_h > d(d + \delta + r)(\mu + \gamma^\star)^2 \Rightarrow \frac{\alpha^\star \beta^\star \lambda_v \lambda_h}{d(d + \delta + r)(\mu + \gamma^\star)^2} > 1. \tag{14}$$

4 Stability of equilibria and basic reproduction number $\mathcal{R}_0$

At the disease-free steady state E_0, the eigenvalues are $-d < 0$, $-\mu - \overline{\gamma} < 0$, $-\theta$ and rest of the eigenvalues satisfy the equation:

$$\xi^2 + (r + \delta + d + \mu + \overline{\gamma})\xi + \left[(r + d + \delta)(\mu + \overline{\gamma}) - \frac{\overline{\alpha}\overline{\beta}\lambda_h\lambda_v}{d(\mu + \gamma)}\right] = 0.$$

Here,

$$\overline{\gamma} = \frac{\gamma_0 A_0}{1 + A_0}, \quad \overline{\beta} = \frac{\mu \beta_0}{\lambda_v (1 + A_0)}, \quad \overline{\alpha} = \frac{d\alpha_0}{\lambda_h (1 + A_0)}, \quad \text{where}, \quad A_0 = \frac{\omega_0}{\theta} .$$

Now, we have determined the *basic reproduction number* for the system (6) as:

$$\mathcal{R}_0 = \frac{\overline{\alpha} \overline{\beta} \lambda_h \lambda_v}{d (\mu + \overline{\gamma})^2 (r + d + \delta)} . \tag{15}$$

Using (15) and (14), we have the following theorem.

Theorem 3. *Disease-free equilibrium E_0 of the model (6) is stable for $\mathcal{R}_0 < 1$, and unstable for $\mathcal{R}_0 > 1$, and ensuring the existence of endemic equilibrium E^*. Consequently, a forward bifurcation at $\mathcal{R}_0 = 1$.*

Remark 1.

(i) The basic reproduction number is derived using the method as established in [45].

(ii) It can be noted that $\mathcal{R}_0$ does not contain ω, hence the level of awareness due to observation of infected cases is not sufficient to eradicate malaria propagation. Moreover, $\mathcal{R}_0$ is monotonically decreasing with increasing ω_0 and γ and Theorem *3* suggests that eradication of malaria is possible if $\mathcal{R}_0 < 1$. The available means to achieve this is by increasing the rate of global awareness ω_0 and also use of insecticide γ.

(iii) The minimum awareness level such that we can rely on awareness for malaria eradication can be determined from Theorem *3*, and it is given below:

$$\omega_0^{\min} = \frac{\theta}{\mu + \gamma_0} \left[\sqrt{\frac{\alpha_0 \beta_0 p_1 p_2 \lambda_h \lambda_v}{r + d + \delta}} - \mu \right] \tag{16}$$

Disease-free equilibrium is stable when $\omega_0 > \omega_0^{\min}$, and the endemic equilibrium exists when $\omega_0 < \omega_0^{\min}$.

(iv) Delay has no effect on the disease-free equilibrium E_0, hence, the stability analysis of E_0, for $\tau > 0$ is not provided.

5 Stability of endemic equilibrium E^* and Hopf bifurcation

Two cases may be raised: (A) when $\tau = 0$ and (B) when $\tau > 0$. We discuss elaborately the cases below.

(A) For $\tau = 0$, the characteristic Eq. (13) takes the form as,

$$\xi^5 + \sigma_1 \xi^4 + (\sigma_2 + \sigma_6)\xi^3 + (\sigma_3 + \sigma_7)\xi^2 + (\sigma_4 + \sigma_8)\xi + (\sigma_5 + \sigma_9) = 0. \tag{17}$$

Denoting $a_1 = \sigma_1, a_2 = \sigma_2 + \sigma_6, a_3 = \sigma_3 + \sigma_7, a_4 = \sigma_4 + \sigma_8, a_5 = \sigma_5 + \sigma_9$ and using Routh-Hurwitz criterion, all roots of the characteristic equation have negative real parts if the following conditions hold

$$a_5 > 0, \quad a_1 a_2 - a_3 > 0, \quad a_3 (a_1 a_2 - a_3) - a_1 (a_1 a_4 - a_5) > 0$$

$$\text{and} \quad (a_1 a_2 - a_3)(a_3 a_4 - a_2 a_5) + (a_1 a_4 - a_5)(a_5 - a_1 a_4) > 0. \tag{18}$$

Thus endemic equilibrium E^* is stable for $\tau = 0$ if the conditions in (18) hold. The following theorem ensures the occurrence of Hopf bifurcation at the endemic equilibrium.

Theorem 4. *The interior equilibrium* E^*

(i) is stable if the conditions in (18) hold and becomes unstable otherwise.

(ii) undergoes Hopf-bifurcation at the generic parameter θ for its critical value θ^* if either of the conditions below is satisfied.

i. $\phi(\theta^*) = 0$ and $\left.\frac{d\phi}{d\theta}\right|_{\theta=\theta^*} \neq 0$, where

$$\phi(\theta) = (a_3 - a_1 a_2)(a_5 a_2 - a_3 a_4) - (a_5 - a_1 a_4)^2,$$

with

$$\varphi = \frac{a_5 - a_1 a_4}{a_3 - a_1 a_2} > 0, \quad a_3 - a_1\varphi \neq 0,$$

ii. $a_5 = a_1 a_4$, $a_3 = a_1 a_2$, $a_4 < 0$, $a_1 a_3 \neq 0$,

$$\left[a_1'\varphi^2 + (a_1 a_2' - a_3')\varphi - (a_1 a_4' - a_5')\right]\Big|_{\theta=\theta^*} \neq 0.$$

where a_i' ($i = 1, 2, \dots 5$) represent the derivatives of a_i ($i = 1, 2, \dots 5$) with respect to the genic bifurcation parameter θ and

$$\varphi = \frac{1}{2}\left(a_2 + \sqrt{a_2^2 - 4a_4}\right) > 0.$$

Outline of the proof of Theorem 4 can be found in [43].

(B) For $\tau > 0$, the characteristic equation at the endemic equilibrium takes the following form,

$$\psi(\xi, \tau) = \xi^5 + \sigma_1\xi^4 + \sigma_2\xi^3 + \sigma_3\xi^2 + \sigma_4\xi + \sigma_5 \tag{19}$$

$$e^{-\xi\tau}\left(\sigma_6\xi^3 + \sigma_7\xi^2 + \sigma_8\xi + \sigma_9\right) = 0.$$

Equation (19) is a transcendental equation containing infinitely many roots. For the stability we need to show that all the roots have negative real parts. For the existence of Hopf bifurcation, there should exist at least one pair of purely imaginary roots.

Without loss of generality, we assume that $\pm i\eta$ ($\eta > 0$) be the pair of pure imaginary roots of Eq. (19). In this case, we put $\xi = i\eta$ in (19). Separating real and imaginary parts, we have

$$\sigma_1\eta^4 - \sigma_3\eta^2 + \sigma_5 = (\sigma_7\eta^2 - \sigma_9)\cos(\eta\tau) + (\sigma_6\eta^3 - \sigma_8\eta)\sin(\eta\tau),$$

$$\eta^5 - \sigma_2\eta^3 + \sigma_4\eta = -(\sigma_7\eta^2 - \sigma_8\eta)\sin(\eta\tau) + (\sigma_6\eta^3 - \sigma_8\eta)\cos(\eta\tau). \tag{20}$$

Squaring and addition both sides of (20), the following equation is derived in η:

$$\eta^{10} + m_1\eta^8 + m_2\eta^6 + m_3\eta^4 + m_4\eta^2 + m_5 = 0, \tag{21}$$

where

$$\begin{aligned}
m_1 &= \sigma_1^2 - 2\sigma_2, \\
m_2 &= \sigma_2^2 - \sigma_6^2 - 2\sigma_1\sigma_3 + 2\sigma_4, \\
m_3 &= \sigma_3^2 - \sigma_7^2 + 2\sigma_6\sigma_8 - 2\sigma_2\sigma_4 + 2\sigma_1\sigma_5, \\
m_4 &= \sigma_4^2 - 2\sigma_3\sigma_5 + 2\sigma_7\sigma_9 - \sigma_8^2, \\
m_5 &= \sigma_5^2 - \sigma_9^2.
\end{aligned} \tag{22}$$

Letting $\eta^2 = l$, Eq. (21) becomes

$$H(l) = l^5 + m_1 l^4 + m_2 l^3 + m_3 l^2 + m_4 l + m_5 = 0, \tag{23}$$

Now, we define the followings for later use,

$$a_1^* = -\frac{6}{25}m_1^2 + \frac{3}{5}m_2, \quad b_1^* = -\frac{6}{25}m_1m_2 + \frac{2}{5}m_3 + \frac{8}{125}m_1^3,$$

$$c_1^* = -\frac{3}{625}m_1^4 + \frac{3}{125}m_1^2m_2 - \frac{2}{25}m_1m_3 + \frac{1}{5}m_4,$$

$$a_2^* = -\frac{1}{3}m_1^* - 4m_3^*, \quad b_2^* = -\frac{2}{27}a_1^{*3} + \frac{8}{3}a_1^*c_1^* - b_1^{*2},$$

$$\Delta_0 = a_1^* - 4c_1^*, \quad d_0 = \sqrt[3]{-\frac{b_2^*}{2} + \sqrt{\Delta_1}} + \sqrt[3]{-\frac{b_2^*}{2} - \sqrt{\Delta_1}} + \frac{a_1^*}{3},$$

$$\Delta_2 = -d_0^* - a_1^* + \frac{2b_1^*}{\sqrt{d_0^* - a_1^*}}, \quad \Delta_3 = -d_0^* - a_1^* - \frac{2b_1^*}{\sqrt{d_0^* - a_1^*}}.$$

(24)

Applying the results on the distribution of roots for an equation of degree five as derived in [46], we have the following lemma.

Lemma 1. *For the polynomial equation* (23)*, the following results hold:*

(a) If $m_5 < 0$, then equation (23) *will have at least one positive root.*
(b) Suppose $m_5 \geq 0$ and $b_1^* = 0$. Now if
    (i) $\Delta_0 < 0$, then equation (23) *has no positive real root,*
    (ii) $\Delta_0 \geq 0$ and $a_1^* \geq 0$, $c_1^* > 0$, then (23) *has no positive real root,*
    (iii) if (i) and (ii) are not satisfied, then (23) *has positive real root if and only if there exists at least one positive $l^* \in \{l_1, l_2, l_3, l_4\}$ such that $H(l^*) \leq 0$, where*

$$l_i = \delta_i - \frac{m_1}{5}, \quad i = 1, 2, 3, 4,$$

and

$$\delta_1 = \sqrt{\frac{-a_1^* + \sqrt{\Delta_0}}{2}} \quad \delta_2 = -\sqrt{\frac{-a_1^* + \sqrt{\Delta_0}}{2}}$$

$$\delta_3 = \sqrt{\frac{-a_1^* - \sqrt{\Delta_0}}{2}} \quad \delta_4 = -\sqrt{\frac{-a_1^* - \sqrt{\Delta_0}}{2}}$$

(c) Again suppose that $m_5 \geq 0$, $b_1^* \neq 0$ and $d_0^* > b_1^*$ then if
    (i) $\Delta_2 < 0$ and $\Delta_3 < 0$ then (23) *has no positive real root;*
    (ii) assume (i) is not satisfied, then (23) *has positive real root if and only if there exists at least one positive $l^* \in \{l_1, l_2, l_3, l_4\}$ such that $H(l^*) \leq 0$, where*

$$l_i = \delta_i - \frac{m_1}{5}, \quad i = 1, 2, 3, 4,$$

and

$$\delta_1 = \frac{-\sqrt{d_0^* - a_1^*} + \sqrt{\Delta_2}}{2} \quad \delta_2 = \frac{-\sqrt{d_0^* - a_1^*} - \sqrt{\Delta_2}}{2}$$

$$\delta_3 = \frac{-\sqrt{d_0^* - a_1^*} + \sqrt{\Delta_3}}{2} \quad \delta_4 = \frac{-\sqrt{d_0^* - a_1^*} - \sqrt{\Delta_3}}{2}$$

(d) *Assume that $\Delta_0 \geq 0$ and $b_1^\star \neq 0$, then (23) has positive real root if and only if $b_1^\star/4\,(a_1^\star - d_0^\star) + \frac{1}{2}d_0^\star = 0$ and $\bar{l} > 0$
    and $H(\bar{l}) \leq 0$, where $\bar{l} = b_1^\star/2\,(a_1^\star - d_0^\star) - \frac{1}{5}m_1$.*

Without loss of generality, we have assumed that Eq. (23) has r positive roots with $r \in \{1, 2, 3, 4, 5\}$, denoted by
$l_k, k = 1, 2, \ldots r$. Then Eq. (21) has r positive roots, say $\eta_k = \sqrt{l_k}$, $k = 1, 2, \ldots r$. Therefore, $i \pm \eta_k$ is a pair of purely
imaginary roots of (19). Now, using Eq. (20), we can derive

$$\sin(\tau\eta) = \frac{(\eta^5 - \sigma_2\eta^3 + \sigma_4\eta)(-\sigma_6\eta^3 + \sigma_9)}{(-\sigma_6\eta^3 + \sigma_9)^2 + (\sigma_6\eta^3 - \sigma_8\eta)^2}$$
$$-\frac{(\sigma_1\eta^4 - \sigma_3\eta^2 + \sigma_5)(-\sigma_6\eta^3 + \sigma_8\eta)}{(-\sigma_6\eta^3 + \sigma_9)^2 + (\sigma_6\eta^3 - \sigma_8\eta)^2} = \gamma(\eta). \tag{25}$$

If Eq. (21) has at least one positive root, say η_0, then Eq. (19) will have a pair of purely imaginary roots $\pm i\eta_0$
corresponding to the delay τ. Without loss of generality, we assume that Eq. (21) has 10 positive real roots,
which are denoted as $\eta_1, \eta_2, \ldots, \eta_{10}$, respectively. For every fixed η_k ($k = 1, 2, \ldots, 10$), the corresponding critical
values of time delay τ are

$$\tau_n^{(j)} = \frac{1}{\eta_k}\arcsin \gamma(\eta) + \frac{2\pi j}{\eta_k}, \quad j = 0, 1, 2, \ldots$$

Let

$$\tau_0 = \min\{\tau_k^{(j)}\}, \quad \eta_0 = \eta_k|_{\tau=\tau_0}, \quad k = 1, 2, \ldots, r, \quad j = 0, 1, 2, \ldots. \tag{26}$$

Taking the derivative of (19) with respect to τ, it is easy to obtain

$$\left(\frac{d\xi}{d\tau}\right)^{-1} = -\frac{5\xi^4 + 4\sigma_1\xi^3 + 3\sigma_2\xi^2 + 2\sigma_3\xi + \sigma_4}{\xi(\xi^5 + \sigma_1\xi^4 + \sigma_3\xi^2 + \sigma_4\xi + \sigma_5)}$$
$$-\frac{3\sigma_6\xi^2 + 2\sigma_7\xi + \sigma_8}{\xi(\sigma_6\xi^3 + \sigma_7\xi^2 + \sigma_8\xi + \sigma_9)} - \frac{\tau}{\xi}. \tag{27}$$

Thus, we have

$$\text{sign}\left[\frac{dRe\{\xi(\tau)\}}{d\tau}\right]_{\tau=\tau_k^{(j)}} = \text{sign}\left[Re\left(\frac{d\xi}{d\tau}\right)\right]_{\xi=i\eta_0} \tag{28}$$

Using (21) and after some calculation, it can be shown taking $\xi = i\eta_0$ that

$$\text{sign}\left[\frac{dRe\{\xi(\tau)\}}{d\tau}\right]_{\tau=\tau_k^{(j)}} = \text{sign}\left[\frac{H'(l_k)}{\eta_0^2(\sigma_6\eta_0^2 - \sigma_8)^2 + (-\sigma_7\eta_0^2 + \sigma_9)^2}\right] \tag{29}$$

We conclude that $\frac{dRe(\xi(\tau))}{d\tau}\Big|_{\tau=\tau_k^{(j)}}$ and $H'(l_k)$ have the same sign.
    We summarize the above discussion in the following theorem.

Theorem 5. *Let l_0 and η_0, τ_0 be defined by (26), then the followings are true.*

1. *If the following conditions:*

    (i) $m_5 < 0$;
    (ii) $m_5 \geq 0$, $b_1^\star = 0$, $\Delta_0 \geq 0$ and $a_1^\star < 0$ or $c_1^\star \leq 0$ and there exists $l^\star \in \{l_1, l_2, l_3, l_4\}$ such that $l^\star > 0$ and $H(l^\star) \leq 0$,
    (iii) $m_5 \geq 0$, $b_1^\star \neq 0$ $d_0^\star > b_1^\star$ and $\Delta_2 \geq 0$ or $\Delta_3 \geq 0$ and there exist $l^\star \in \{l_1, l_2, l_3, l_4\}$ such that $l^\star > 0$ and $H(l^\star) \leq 0$,

(iv) $\Delta_0 \geq 0$ and $b_1^\star \neq 0$, then (23) has positive real root if and only if $b_1^\star / 4(a_1^\star - d_0^\star) + \frac{1}{2}d_0^\star = 0$ and $\bar{l} > 0$ and $H(\bar{l}) \leq 0$, where $\bar{l} = b_1^\star / 2(a_1^\star - d_0^\star) - \frac{1}{5}m_1$,

are all not satisfied then equilibrium E^* is locally asymptotically stable for any $\tau \geq 0$.

2. If one of the conditions given in (a) is satisfied, then equilibrium E^* is locally asymptotically stable for $\tau \in [0, \tau_0)$.

3. If one of the conditions given in (a) holds and $H(\bar{l}_0) \neq 0$, then E^* undergoes Hopf bifurcation as τ passes through the critical value τ_0.

6 Numerical stability analysis and simulations

In this section, in order to observe how the parameters affect the stability properties of the equilibria, we numerically solve the model system and compute the characteristic eigenvalues in Matlab.

In Figure 1, we illustrate how actual dynamics of the system (6) changes depending on awareness rate ω_0. Figure 1(a) and (b) shows the system approaches the stable disease-free or endemic steady states for $\mathcal{R}_0 < 1$ or $\mathcal{R}_0 > 1$, respectively. From Figure 1(a), it is clear that there exists a minimum value of ω_0 below which infection persists.

It can be noted that according to Theorem 3, the stability of the disease-free equilibrium does not depend on the time delay τ, rather on the basic reproduction number $\mathcal{R}_0$ only. So if we can keep the value of $\mathcal{R}_0$ below 1, the disease-free steady state would be stable for any $\tau > 0$. At $\mathcal{R}_0 = 1$, a transcritical bifurcation occurs, and the system becomes endemic. In Figure 2, the impact of local awareness ω on infected cases is shown. As ω increases infected humans decrease.

Figure 3 illustrates how stability of the endemic steady state depends on the disease transmission rates α_0 and β_0. We observe that the product of transmission rate α_0 from vector to human and β_0 from human to vector
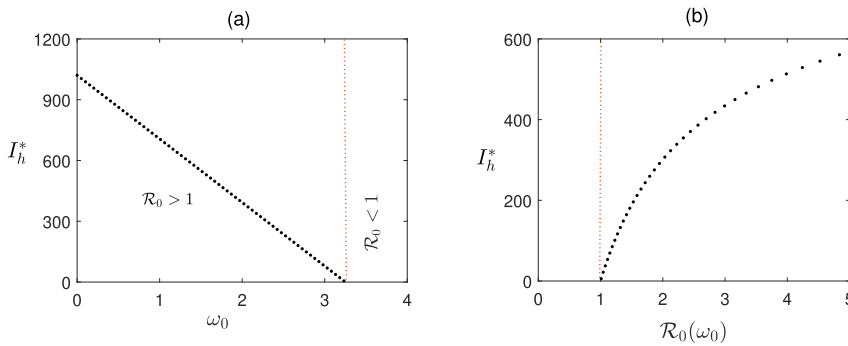
Figure 1: Transcritical bifurcation: Effect of ω_0 (global awareness) on the size of the epidemic and stability of disease-free equilibrium E_0. Values of the parameters are taken from Table 1.
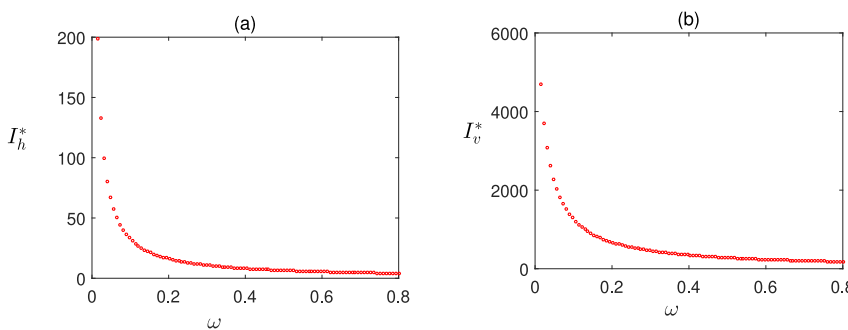
Figure 2: Effect of ω (local awareness) on the size of the epidemic. Values of the parameters are taken from Figure 1.
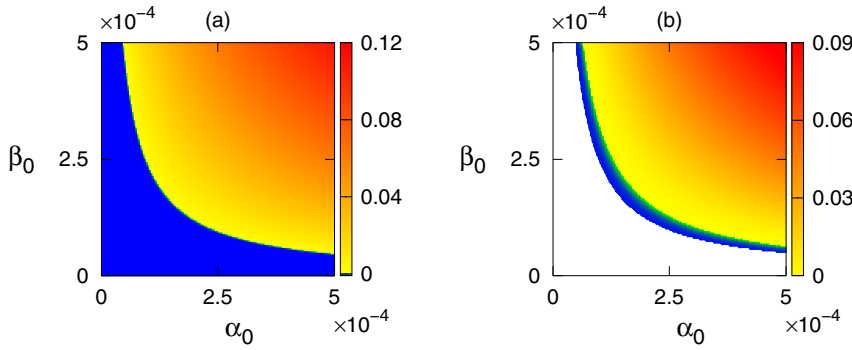
Figure 3: Region of stability of equilibria in $\alpha_0 - \beta_0$ parameter plane: (a) disease-free, (b) endemic equilibrium. Rest of the parameters are taken from Figure 1. Color code denotes the $\max[Re\,\xi]$ at steady state. In white region of both the figures, endemic equilibrium E^* is not feasible.

has to exceed some threshold value for the endemic steady state to be feasible, and for smaller values of this product the endemic state is stable but then also loses its stability.

Figure 4 shows that the endemic equilibrium only exists for a limited range of disease transmission rates, and it is stable for higher rates and unstable for smaller α_0. Increasing the local awareness rate ω leads to a destabilization of the endemic steady state, but surprisingly, increasing a global awareness rate ω_0 or a local awareness rate ω actually results in stabilizing an endemic steady state, whilst increasing these rates above certain values makes the endemic steady state unfeasible, in which case the disease-free steady state is stable.

In Figure 5, choosing parameters in the range where $\mathcal{R}_0 > 1$ and increasing the time delays τ, resulting in the system approaching endemic steady state in an oscillatory manner, with the amplitude of oscillations increasing with the time delay. Once the time delay exceeds the critical value τ_0, determined by Theorem 5, the endemic steady state becomes unstable via Hopf bifurcation, and the system exhibits stable periodic solutions. Figure 6 further illustrates the bifurcation diagram of endemic steady state E^* taking τ as the bifurcation parameter. Minima and maxima of this solution suggests that the amplitude of oscillations itself increases with τ.

Figure 7 illustrates how the critical value τ_0 depends on different parameters. If disease transmission rate α_0 increases, Hopf bifurcation can be observed for lower value of τ. It also suggests that critical value τ_0 increases as the global awareness level ω_0 increases. Delay-induced oscillation can be reduced if both disease transmission rate is lowered and increasing the level of awareness.

Effect of global awareness ω_0 is plotted in Figure 8. The periodic oscillation disappears for higher rate of global awareness. Thus the awareness campaign through radio, TV etc. is very important for malaria disease control and also for mosquito population control.
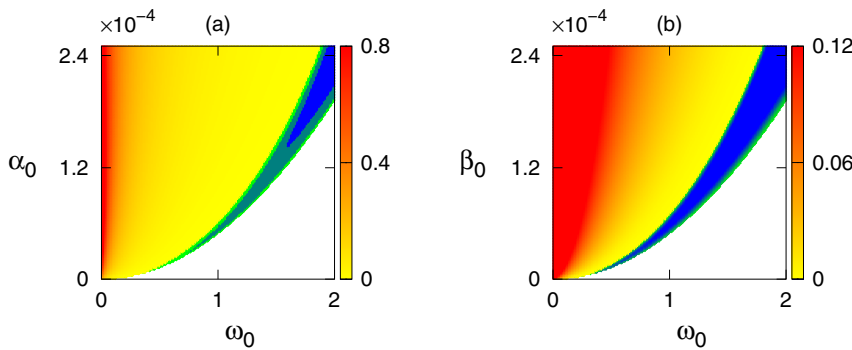
Figure 4: Region of stability of equilibria in $\omega_0 - \beta_0$ and $\omega_0 - \alpha_0$ parameter plane: (a) disease-free, (b) endemic equilibrium. Rest of the parameters are taken from Figure 1. Color code denotes the $\max[Re\,\xi]$ at E^*. In white region of both the figures, endemic equilibrium E^* is not feasible.
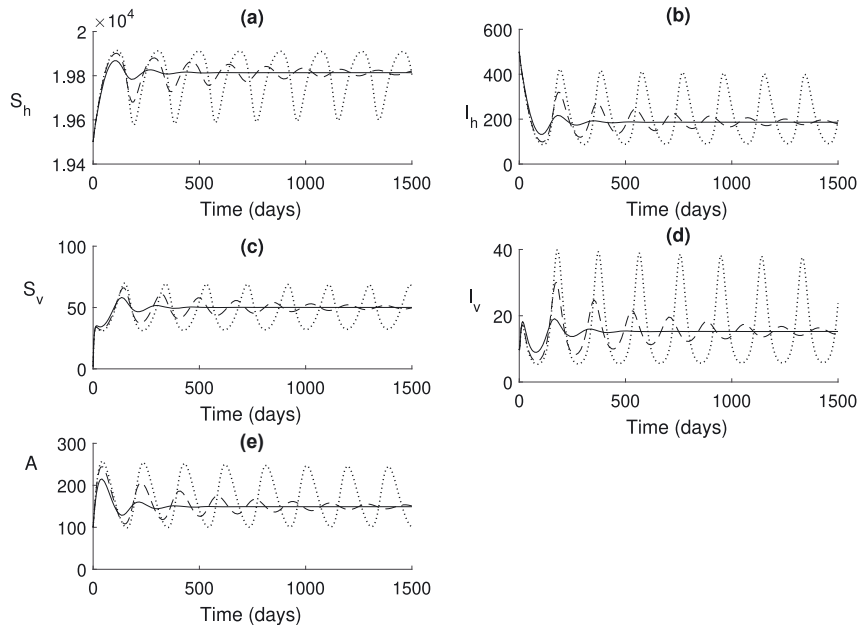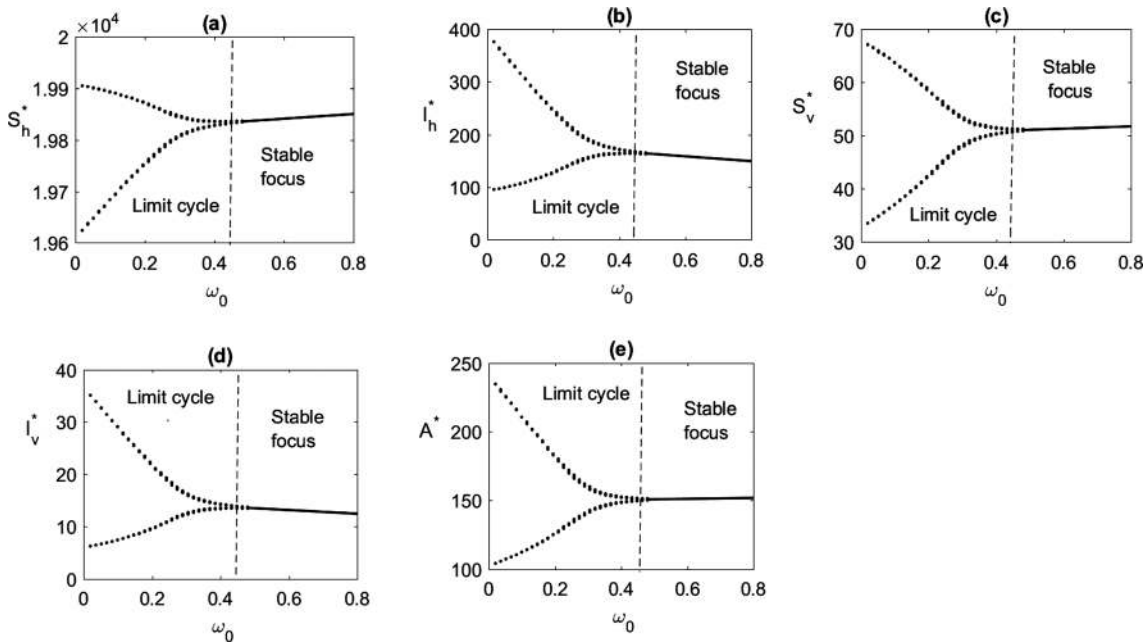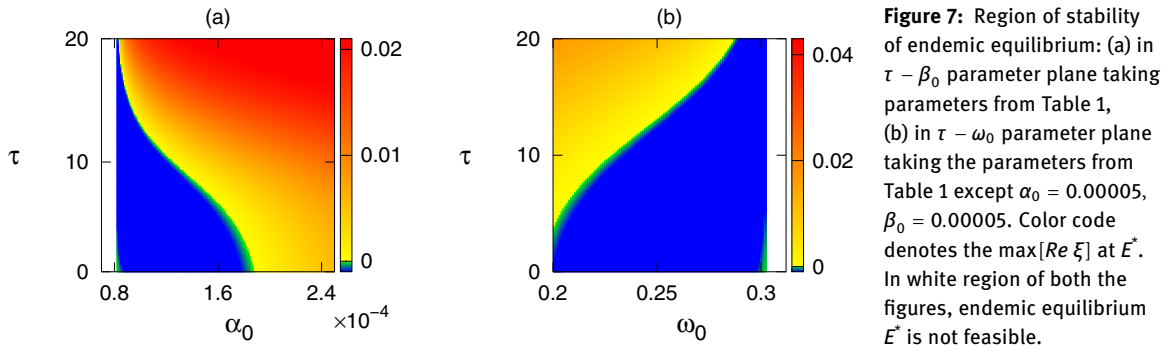
Figure 5: Numerical solution of model (6) populations for different values of delay, τ. Parameters are taken from Table 1. Solid line is for $\tau = 0$, dashed line for $\tau = 12$ and dotted line is for $\tau = 18$.
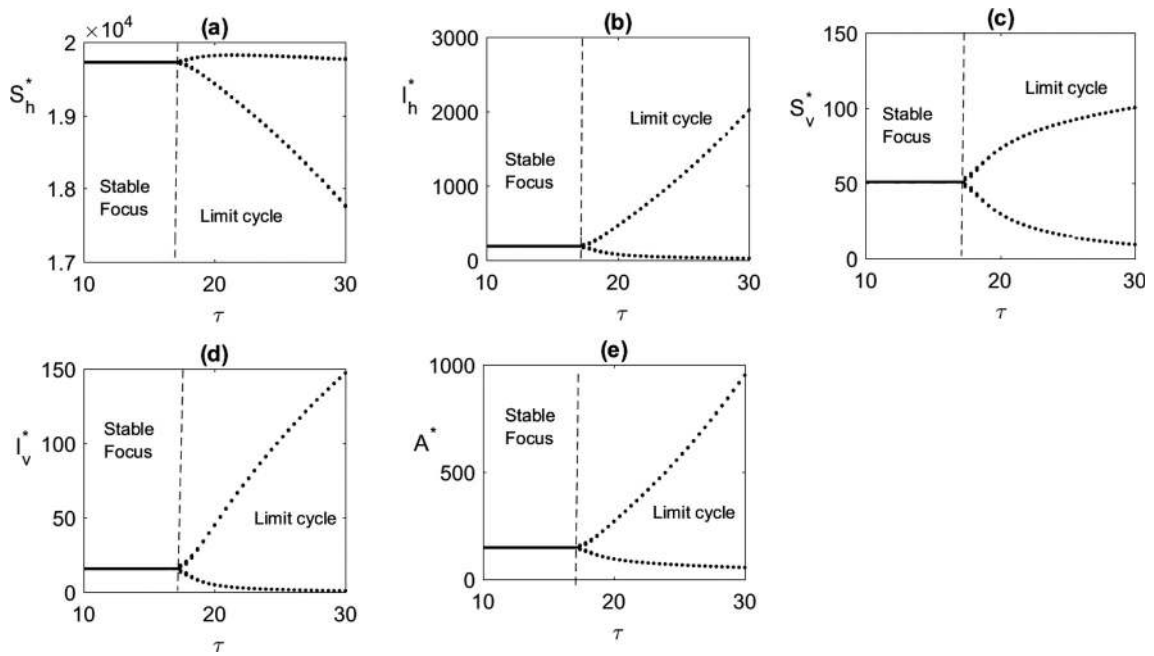
Figure 6: Bifurcation diagram is plotted taking τ as the main parameter. Parameters are taken from Table 1.

In Figure 9, we have shown the effect of insecticide on the delayed system. Periodic oscillation disappears and the endemic equilibrium becomes stable when γ_0 crosses the critical value $\gamma_0^* = 0.5827$. This shows that insecticide spraying is sensitive for malaria disease control.

Figure 7: Region of stability of endemic equilibrium: (a) in $\tau - \beta_0$ parameter plane taking parameters from Table 1, (b) in $\tau - \omega_0$ parameter plane taking the parameters from Table 1 except $\alpha_0 = 0.00005$, $\beta_0 = 0.00005$. Color code denotes the $\max[Re\ \xi]$ at E^*. In white region of both the figures, endemic equilibrium E^* is not feasible.

Figure 8: Bifurcation diagram is plotted taking ω_0 (global awareness) as the main parameter. Values of the parameters are taken from Table 1.

7 Discussion

Successful control of any vector-borne disease follows several factors including, but not limited to well-planned eradication strategies, targeted control measures to prevent spread of disease and also increase of awareness among the general mass. While inception of eradication programs require commitment from the higher authorities—namely policy makers—similar burden also falls on the common people to gather knowledge and increase awareness among themselves. This awareness is generated via effective expansion and improvement of case management, and through successive spread of the information [47].

In this article, the impact of awareness-based intervention for malaria control has been studied. For this, a mathematical model is derived considering SIS type sub-model for humans and SI sub-model for mosquitoes and the level of awareness as a separate population. We have assumed the disease transmission rates, vector to human and human to vector as decreasing function of the level of awareness, $A(t)$. Model analysis shows two feasible equilibria namely the disease-free and endemic equilibrium. Disease-free equilibrium is stable until
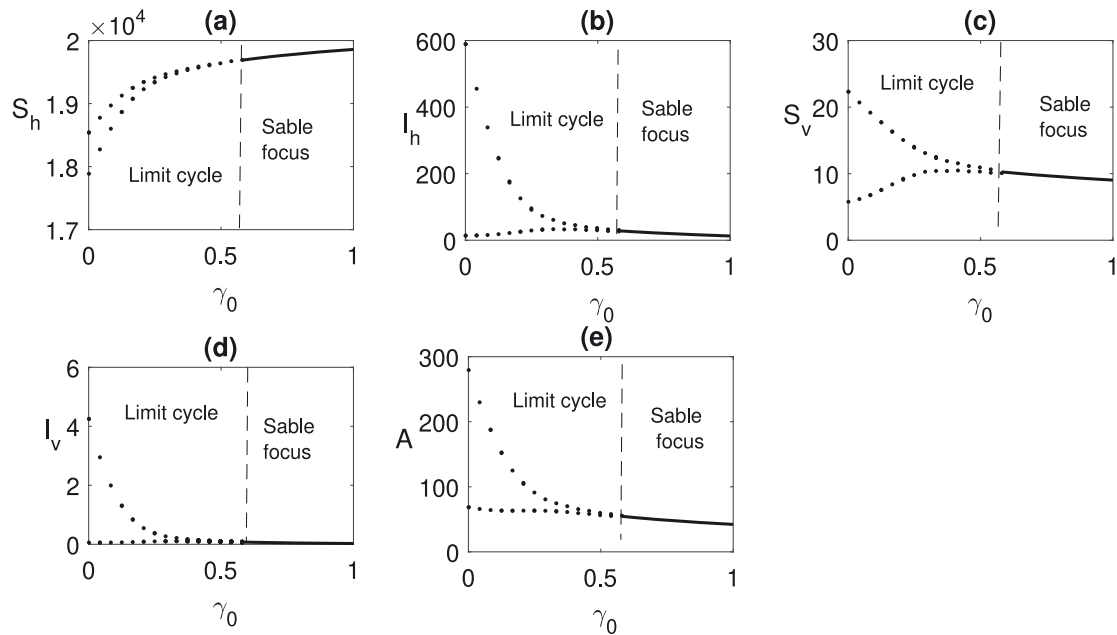
Figure 9: Bifurcation diagram is plotted taking γ_0 (rate of insecticide spraying) as the main parameter. Values of the parameters are taken from Table 1.

the value of the basic reproduction number, $\mathcal{R}_0 < 1$, but as the value of $\mathcal{R}_0$ exceeds 1, it becomes unstable, and numerically we see that endemic equilibrium exists for $\mathcal{R}_0 > 1$, which is a standard result in epidemiology. There exists a minimum awareness level $\omega_0^{\min}$ such that we can rely on awareness for malaria eradication (Remark 1). The endemic equilibrium, whenever exists, is locally asymptotically stable when the delay τ is below the threshold value τ_0. It becomes unstable through Hopf bifurcation if the delay parameter crosses the critical value τ_0. We have seen that the critical value of τ depends on other parameters [10] namely global awareness rate ω_0 and awareness activity rate γ_0 (Figures 6–8).

The model analysis suggests that level of awareness among people has enough potential to mobilize higher fraction of the population. Increasing the awareness through radio, TV etc., the abundance of mosquitoes will be reduced in the environment and hence disease can be controlled. Moreover, it is found that media coverage has a substantial effect on the basic reproduction number, $\mathcal{R}_0$. Also, by continuous efforts and effectual media coverage with tolerable delay disease can be eradicated.

While spread of successful control strategies adds to the awareness about a disease, it is not uncommon that this knowledge is diluted over certain periods of time. In these cases, debates may arise that shift the focus from the actual course of action that needs to be selected. These shortcomings should be included in the mathematical models to find their relevance.

Appendix

The coefficients of Eq. (19) are given below,

$$\sigma_1 = -\left(J_{11} + J_{22} + J_{44} + J_{33} + J_{55}\right),$$

$$\sigma_2 = +J_{12}J_{21} - J_{11}J_{22} - J_{11}J_{33} - J_{22}J_{33} + J_{23}J_{42} - J_{11}J_{44}$$
$$- J_{22}J_{44} - J_{33}J_{44} - J_{11}J_{55} - J_{22}J_{55} - J_{33}J_{55} - J_{44}J_{55},$$

$$\sigma_3 = -J_{12}J_{21}J_{33} + J_{11}J_{22}J_{33} + J_{13}J_{21}J_{42} - J_{11}J_{23}J_{42}$$
$$- J_{23}J_{33}J_{42} + J_{23}J_{32}J_{43} - J_{12}J_{21}J_{44} + J_{11}J_{22}J_{44} + +J_{22}J_{33}J_{55}$$
$$J_{11}J_{33}J_{44} + J_{22}J_{33}J_{44} - J_{12}J_{21}J_{55} + J_{11}J_{22}J_{55} + J_{11}J_{33}J_{55}$$
$$- J_{23}J_{42}J_{55} + J_{11}J_{44}J_{55} + J_{22}J_{44}J_{55} + J_{33}J_{44}J_{55},$$

$$\sigma_4 = -J_{13}J_{21}J_{33}J_{42} + J_{11}J_{23}J_{33}J_{42} + J_{13}J_{21}J_{32}J_{43} - J_{11}J_{23}J_{32}J_{43}$$
$$- J_{22}J_{33}J_{44}J_{55} + J_{12}J_{21}J_{33}J_{44} - J_{11}J_{22}J_{33}J_{44}$$
$$- J_{23}J_{33}J_{45}K_{52} + J_{12}J_{21}J_{33}J_{55} - J_{11}J_{22}J_{33}J_{55} + J_{11}J_{23}J_{42}J_{55}$$
$$+ J_{23}J_{33}J_{42}J_{55} - J_{23}J_{32}J_{43}J_{55} + J_{12}J_{21}J_{44}J_{55} - J_{11}J_{22}J_{44}J_{55}$$
$$- J_{11}J_{33}J_{44}J_{55} - J_{13}J_{21}J_{42}J_{55},$$

$$\sigma_5 = J_{13}J_{21}J_{33}J_{42}J_{55} - J_{11}J_{23}J_{33}J_{42}J_{55} - J_{13}J_{21}J_{32}J_{43}J_{55}$$
$$+ J_{11}J_{23}J_{32}J_{43}J_{55} - J_{12}J_{21}J_{33}J_{44}J_{55} + J_{11}J_{22}J_{33}J_{44}J_{55},$$

$$\sigma_6 = -J_{14}K_{52},$$

$$\sigma_7 = J_{11}J_{14}K_{52} + J_{14}J_{21}K_{52} + J_{14}J_{33}K_{52} + J_{14}J_{44}K_{52} + J_{23}J_{45}K_{52}$$

$$\sigma_8 = J_{11}J_{14}J_{33}K_{52} - J_{14}J_{21}J_{33}K_{52} + J_{23}J_{34}J_{43}K_{52} - J_{11}J_{14}J_{44}K_{52}$$
$$- J_{14}J_{21}J_{44}K_{52} - J_{14}J_{33}J_{44}K_{52} + J_{13}J_{21}J_{45}K_{52} - J_{11}J_{23}J_{45}K_{52},$$

$$\sigma_9 = J_{13}J_{21}J_{34}J_{43}K_{52} - J_{11}J_{23}J_{34}J_{43}K_{52} + J_{11}J_{14}J_{33}J_{44}K_{52}+$$
$$J_{14}J_{21}J_{33}J_{44}K_{52} - J_{13}J_{21}J_{33}J_{45}K_{52} + J_{11}J_{23}J_{33}J_{45}K_{52}.$$

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