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A MATHEMATICAL MODEL FOR CONTROL OF VECTOR BORNE DISEASES THROUGH MEDIA CAMPAIGNS

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Abstract. Vector borne diseases spread rapidly in the population. Hence their control intervention must work quickly and target large area as well. A rational approach to combat these diseases is mobilizing people and making them aware through media campaigns. In the present paper, a non-linear mathematical model is proposed to assess the impact of creating awareness by the media on the spread of vector borne diseases. It is assumed that as a response to awareness, people will not only try to protect themselves but also take some potential steps to inhibit growth of vectors in the environment. The model is analyzed using stability theory of differential equations and numerical simulation. The equilibria and invasion threshold for infection i.e., basic reproduction number, has been obtained. It is found that the presence of awareness in the population makes the disease invasion difficult. Also, continuous efforts by the media along with the swift dissemination of awareness can completely eradicate the disease from the system.

1. **Introduction.** Vector borne diseases like dengue, malaria, chikungunya, yellow fever, etc., cause enormous morbidity and mortality in tropical and subtropical regions. Globally, malaria is the most prevalent amongst all vector borne diseases. It is a serious health concern in various nations of Africa, South-East Asia, America, Europe and Western Pacific region. It kills about two-three million people annually, [32] mostly African residents and is second most lethal disease after HIV/AIDS in South Africa. Estimates reveal that two fifth of the world's population is at risk of contracting dengue, [31]. In the 2006 outbreak of chikungunya, around 1.25 million cases were reported from various states of India, [30]. Each year yellow fever affects about 0.2 million people and causes 30,000 deaths worldwide, [33]. These figures clearly demonstrate that the prevalence of vector borne diseases is escalating with an alarming rate.

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Vector borne diseases cannot be transmitted to humans directly but through blood-sucking arthropods called *vectors*. Basically, the transmission of vector borne diseases depends upon three factors: the pathogen, the vector and the host. Due to unavailability of effective vaccines, vector control is one of the primary interventions used for eradication of vector borne diseases. For this purpose mostly insecticides are used, which toxify breeding sites of the mosquitoes and aquatic habitats of the larva. Indoor residual spraying, space spraying, chemical larvicides and adulticides are also employed to alter the longevity of vectors. But their use has always been a matter of debate due to their hazardous effects on human health. Another problem associated with the use of insecticides is that vectors develop resistance via evolution after some generations. This has attributed to the re-emergence of various vector borne diseases in Africa, India and many other nations, [26, 28]. As continuous use of insecticides makes them less effective, it is not a stable solution for vector control. In view of this, World Health Organization(WHO) is emphasizing on Integrated Vector Management for control of vector borne diseases, instead of relying on a single method, [9]. Hence, continuous efforts are being made by researchers to gain profound insight about transmission and plausible strategies for control of vector borne diseases, [2, 10, 15, 16, 19, 21, 25, 27].

A major obstacle in the implementation of any strategy for disease control is public adherence to their perceived believes. In rural and semi-rural areas of countries where these diseases are more prevalent, various misconceptions prevail among people about vectors and their breeding sites, [1, 29]. But if well-informed, people may modify their behavior to minimize their risk of contracting infection and reshape the future course of an epidemic, [5]. Therefore, a lot of attention is being paid to make people aware of causes and preventive measures requisite for vector borne diseases so that their prevalence can be controlled. In this regard, the US president designated April 25 as *Malaria Awareness Day* to continuously foster awareness among people. Moreover, some NGO's and other non-profitable firms like "malaria no more" are also taking laudable participation in fight against ravaging vector borne diseases like malaria and dengue. In India, a comic book named "A Tale of Two Magic Potions" is being used as an innovative approach for making people aware of malaria and its control, [22].

Thus, creating awareness through media campaigns can serve as a commendable avenue for control of vector borne diseases. In our recent work, [23] we have shown that an epidemic outbreak can be controlled if people take obligatory precautions to protect themselves from contracting infection. However, to hold better command over the prevalence, awareness programs should be implemented within a plausible time because longer delay in their implementation may destabilize the system, [24]. Some modelers have also studied the impact of media, information and awareness programs on the spread of directly transmitted infectious diseases, [6, 7, 8, 12, 14, 17, 18]. But the transmission dynamics of vector borne diseases is different from directly transmitted diseases. Hence, the interplay between dissemination of awareness and transmission of vector borne diseases should be studied explicitly. Therefore, in the present paper a nonlinear mathematical model is proposed and analyzed, to assess the impact of creating awareness through media campaigns on vector borne diseases.

In the next section, model formulation along with its full description is provided. In Sections 3, some mathematical results regarding the existence of equilibria, basic reproduction number and stability behavior of equilibria obtained, are derived. In

Section 4, numerical simulation is carried out to check the feasibility of analytically obtained results. Finally, in the last section the principal findings of the study are summarized.

2. **The mathematical model.** Consider a habitat with two interacting populations; human (host) and mosquito (vector). The human population is increasing with a constant rate and mosquito population logistically. In epidemiology, it is well known fact that vectors are the potential transmitters of vector borne diseases. So, interaction of susceptible humans with infective mosquitoes will give rise to the emergence of infective cases in human population. Similarly, when a susceptible mosquito bites the infected human, mosquito will become infective. Hence, there is a criss-cross interaction between human and mosquito population, which makes the dynamics of the disease more complex. Now, when any vector borne disease emerges within the population, public health agencies will increase the media campaigns to propagate awareness about disease transmission and its control. Due to media campaigns, some people will become acquainted with the tools and techniques requisite for prevention of prevalent disease. Consequently the behavioral changes will occur among the population and some people will form a separate class. We have regarded these people as aware people. The aware people will not only modify their behavior to alter potential risk of acquiring infection but also attribute to vector control. They will use measures such as sleeping under impregnate bed nets, using mosquito repellents, etc., for personal protection. Moreover, they will make effectual efforts to reduce the breeding sites for mosquitoes such as dirty and stagnant water, by keeping their surroundings clean and hence reduce the growth of mosquitoes. It is noted here that in the modelling process our main focus is on studying the dynamics of most prevalent vector borne disease, i.e., malaria. Therefore, we have made the assumptions those are relevant to the spread of malaria and other diseases with similar dynamics.

In the modelling process, the total human population $N_H(t)$ at time t is divided in three compartments; susceptible human population $X_H(t)$, infective human population $Y_H(t)$ and aware human population $A_H(t)$, as per their status with respect to disease evolve and awareness. Let us assume that human population is increasing, either by birth or immigration, with a constant rate Λ. The entire human population is subject to natural mortality with a constant rate d_H. Consider that $N_V(t)$ denotes the total mosquito population at time t, which is also divided in two subclasses: susceptible mosquito population $X_V(t)$ and infective mosquito population $Y_V(t)$. The mosquito population is assumed to follow the logistic growth model with intrinsic growth rate r and carrying capacity K. It is assumed that the birth rate of mosquito population is density dependent whereas death rate is constant, [20]. Further, let $M(t)$ denotes the number of media campaigns at that time t. We have considered that these media campaigns will be executed according to the number of infective humans present in the community. Moreover, the diminution of media campaigns due to social and psychological barriers is also incorporated in the modeling process. Further, we have considered that not all the programs get diminished but a certain level of campaigns are always be maintained in the system because even if the infective individuals are not present in the system there is a possibility that some infective mosquito may be harboring the pathogens within them. Considering the progression of infection according to SIS model among humans and SI model among mosquitoes, the afore-discussed situation can mathematically

be depicted as follows:

$$\begin{aligned}
\frac{dX_H}{dt} &= \Lambda - \beta_{HV} X_H Y_V - \lambda X_H M - d_H X_H + \nu Y_H + \lambda_0 A_H,\\
\frac{dY_H}{dt} &= \beta_{HV} X_H Y_V - \nu Y_H - \alpha Y_H - d_H Y_H,\\
\frac{dA_H}{dt} &= \lambda X_H M - \lambda_0 A_H - d_H A_H,\\
\frac{dX_V}{dt} &= b_V N_V - r\frac{N_V^2}{K} - \beta_{VH} X_V Y_H - \phi A_H X_V - d_V X_V,\\
\frac{dY_V}{dt} &= \beta_{VH} X_V Y_H - \phi A_H Y_V - d_V Y_V,\\
\frac{dM}{dt} &= \mu Y_H - \mu_0 (M - M_0),
\end{aligned} \tag{1}$$

where, $X_H(0) > 0, Y_H(0) \geq 0, A_H(0) \geq 0, X_V(0) \geq 0, Y_V(0) > 0, M(0) \geq M_0$.

Here, the constant β_{HV} denotes the rate with which susceptible humans acquire infection from mosquitoes. We assume that human feeding rate, i.e., the number of bites per mosquito on a human per unit time is b and the transmission probability of infection from mosquito to human per bite is ρ_1, so that $\beta_{HV} = b\rho_1$. Similarly, consider that probability of transmission of infection from human to mosquito per bite is ρ_2 then, $\beta_{VH} = b\rho_2$ denotes the rate with which mosquitoes acquire infection from humans. Further, the constants ν and α denote the recovery rate and disease-induced death rate of human population. The constant λ represents the rate of dissemination of awareness among humans due to which they form a separate class i.e., aware class and λ_0 is the rate with which people in aware class lose awareness due to memory fading and some other social reasons. It is assumed that individuals in the aware class will take all necessary precautions for personal protection and successfully escape the chances of contracting infection. For mosquito population, b_V and d_V denote the natural birth and death rate respectively. It is noted here that the difference of natural birth and the natural death rate gives the intrinsic growth rate of mosquito population i.e., $r = b_V - d_V$. Also, the efforts made by aware individuals will reduce the abundance of mosquitoes with a rate ϕ. The constant μ represents the rate with which awareness campaigns are being executed and μ_0 represents the rate with which they are diminishing. Moreover, M_0 represents the baseline number of awareness programs maintained in the system. All the above constants are assumed to be positive. It is noted here that the proposed model is density dependent, which is a realistic assumption for vector borne diseases.

3. **Model analysis.** In the modelling process, we have used the fact that $N_H = X_H + Y_H + A_H$ and $N_V = X_V + Y_V$. So, the model system (1) can also be written in the following from,

$$\begin{aligned}
\frac{dY_H}{dt} &= \beta_{HV}(N_H - Y_H - A_H)Y_V - (\nu + \alpha + d_H)Y_H,\\
\frac{dA_H}{dt} &= \lambda(N_H - Y_H - A_H)M - (\lambda_0 + d_H)A_H,\\
\frac{dN_H}{dt} &= \Lambda - \alpha Y_H - d_H N_H,
\end{aligned} \tag{2}$$

$$\begin{aligned}\frac{dY_V}{dt} &= \beta_{VH}(N_V - Y_V)Y_H - (\phi A_H + d_V)Y_V,\\ \frac{dN_V}{dt} &= rN_V\left(1 - \frac{N_V}{K}\right) - \phi A_H N_V,\\ \frac{dM}{dt} &= \mu Y_H - \mu_0(M - M_0).\end{aligned}$$

Here, model system (2) is equivalent to model system (1), so in the further course of study we will analyze model system (2). The region of attraction for model system (2) is given by the set,

$$\begin{aligned}\Omega = \Big\{(Y_H, A_H, N_H, Y_V, N_V, M) \in \mathbb{R}^6_+ : 0 \le Y_H, A_H \le N_H \le \frac{\Lambda}{d_H},\\ 0 \le Y_V \le N_V \le K_R, 0 \le M \le M_R\Big\}, \qquad (3)\end{aligned}$$

where, $K_R = \dfrac{K\left(r - (\phi p\Lambda/d_H)\right)}{r}$ and $M_R = \dfrac{\mu(\Lambda/d_H) + \mu_0 M_0}{\mu_0}$,which attracts all solutions initiating inside the positive orthant.

Here, p is a dimensionless quantity defined as, $p = \dfrac{\lambda M_0}{\lambda M_0 + \lambda_0 + d_H}$ and it is apparent that $0 < p < 1$.

3.1. **Equilibrium states and their existence.** In order to obtain the equilibria of model system (2), we solve the following algebraic equations, which are obtained by setting growth rate of all the variables equal to zero.

$$\begin{aligned}\beta_{HV}(N_H - Y_H - A_H)Y_V - (\nu + \alpha + d_H)Y_H &= 0, &(4)\\ \lambda(N_H - Y_H - A_H)M - (\lambda_0 + d_H)A_H &= 0, &(5)\\ \Lambda - \alpha Y_H - d_H N_H &= 0, &(6)\\ \beta_{VH}(N_V - Y_V)Y_H - (\phi A_H + d_V)Y_V &= 0, &(7)\\ rN_V\left(1 - N_V/K\right) - \phi A_H N_V &= 0, &(8)\\ \mu Y_H - \mu_0(M - M_0) &= 0. &(9)\end{aligned}$$

The model system (2) exhibits three non-negative equilibria:

(i) Disease- and vector-free equilibrium (DVFE) $E_0(0, p\Lambda/d_H, \Lambda/d_H, 0, 0, M_0)$, this equilibrium is biologically not feasible because the extinction of vector population is not a usual phenomenon.

(ii) Disease-free equilibrium (DFE) $E_1(0, p\Lambda/d_H, \Lambda/d_H, 0, K_R, M_0)$, this equilibrium exists provided $r > \phi p(\Lambda/d_H)$ i.e., for existence of this equilibrium intrinsic growth rate of vector population must be higher than their deterioration rate.

(iii) Endemic equilibrium $E^*(Y_H^*, A_H^*, N_H^*, Y_V^*, N_V^*, M^*)$,
which exists under certain conditions. This equilibrium denotes the endemicity of disease in the system.

The existence of E_0 and E_1 is trivial. So, here we discuss the existence of E^* only. For this purpose, using equations (5), (6) and (9), we obtain

$$A_H = \frac{\lambda(\Lambda - (\alpha + d_H)Y_H)(\mu Y_H + \mu_0 M_0)}{d_H(\lambda(\mu Y_H + \mu_0 M_0) + \mu_0(\lambda_0 + d_H))} = g(Y_H)(\text{say}). \qquad (10)$$

Further, for $N_V \neq 0$, equation (8) yields

$$N_V = \frac{K}{r}(r - \phi g(Y_H)). \qquad (11)$$

From equations (7) and (11), we can obtain

$$Y_V = \frac{\beta_{VH} K(r - \phi g(Y_H)) Y_H}{r(\beta_{VH} Y_H + \phi g(Y_H) + d_V)}. \tag{12}$$

Finally using all these values in equation (4) for $Y_H \neq 0$, we get a function $f(Y_H)$ as

$$\begin{aligned} f(Y_H) &= \beta_{HV}\beta_{VH}\left(\frac{\Lambda - \alpha Y_H}{d_H} - Y_H - g(Y_H)\right)\frac{K(r - \phi g(Y_H))}{r(\beta_{VH} Y_H + \phi g(Y_H) + d_V)} \\ &\quad -(\nu + \alpha + d_H) = 0. \end{aligned} \tag{13}$$

The investigation of function $f(Y_H)$ leads to following observations,

(i) $f(0) = \beta_{HV}\beta_{VH}\dfrac{(1-p)\Lambda K(r - \phi p(\Lambda/d_H))}{r d_H(\phi p(\Lambda/d_H) + d_V)} - (\nu + \alpha + d_H)$, which is positive provided

$$\frac{\beta_{HV}\beta_{VH}(1-p)\Lambda K(r - \phi p(\Lambda/d_H))}{r d_H(\phi p(\Lambda/d_H) + d_V)(\nu + \alpha + d_H)} > 1. \tag{14}$$

(ii) $f(\frac{\Lambda}{\alpha + d_H}) = -(\nu + \alpha + d_H)$, which is negative.

This suggests that we may obtain a unique positive value of Y_H in the interval $(0, \Lambda/(\alpha + d_H))$ provided $f'(Y_H) < 0$. Let us denote this positive value of Y_H as Y_H^*. Furthermore, the substitution of this value in equations (6), (9), and (10), yields the equilibrium values of N_H, M, A_H as N_H^*, M^*, A_H^*. Finally, using value of Y_H^* in (11) and (12), we get a positive value of N_V and Y_V as N_V^* and Y_V^* provided $r > \phi g(Y_H^*)$ i.e., $r > \phi A_H^*$.

3.2. **Basic reproduction number.** A pivotal concept in the study of infectious diseases, which mathematical studies has brought to epidemic theory, is basic reproduction number, R_0. It represents the average number of secondary infective cases generated by a single infected individual during his or her whole infectious period, in entirely susceptible population, [4]. For model system (2), we have derived a condition for the existence of endemic equilibrium, in terms of parameters as

$$\frac{\beta_{HV}\beta_{VH}(1-p)\Lambda K(r - \phi p(\Lambda/d_H))}{r d_H(\phi p(\Lambda/d_H) + d_V)(\nu + \alpha + d_H)} > 1.$$

From here, we made an assertion that the endemic equilibrium exists, whenever above said condition holds. We denote this quantity by R_0 which represents the '*basic reproduction number*' for model system (2). It follows that disease will eradicate if $R_0 < 1$, whereas if $R_0 > 1$ disease will invade the population giving rise to an endemic, which is a standard result in epidemiology.

As infective human produces infective vectors and vice-versa, the basic reproduction number,

$$R_0 = \frac{\beta_{HV}\beta_{VH}(1-p)\Lambda K(r - \phi p(\Lambda/d_H))}{r d_H(\phi p(\Lambda/d_H) + d_V)(\nu + \alpha + d_H)}, \text{is cumulation of}$$

$(K(r - \phi p(\Lambda/d_H))\beta_{HV})/(r(\nu + \alpha + d_H))$ and $((1-p)\Lambda\beta_{VH})/(d_H(\phi p(\Lambda/d_H) + d_V))$. The first quantity represents the expected number of infective vector cases produced by a single infective human during its entire infectious period, in completely susceptible mosquito population which is $K(r - \phi p(\Lambda/d_H))/r = K_R$. Similarly, the later quantity denotes the average number of infective human cases generated by a typical infective vector in its whole infectious period, when entire human population is susceptible i.e., $(1-p)\Lambda/d_H$. Here, R_0 follows the traditional definition of the reproductive number, which represents the number of secondary infectives in

human population caused by one infected human, [13]. However, the basic reproduction number is also be obtained using next generation matrix (NGM) approach following [3] *(see Appendix A)*.

It is interesting to note here that the aware population have a potential impact on the R_0. Essentially, the aware people will reduce the carrying capacity of the mosquito population by lessening the breeding sites for mosquito population. Also they will alter their vulnerability to infection and hence reduce the number of susceptible people in the population. Moreover from the expression of R_0, it is found that $R_0{}'(p) < 0$, which indicates that R_0 decreases as the value of p increases. Hence, by increasing the value of p above a critical level *(say p_c)*, R_0 can be reduced to less than unity and thus disease can be eradicated. The value of p_c can be obtained by using $R_0(p_c) = 1$, which yields

$$p_c = \frac{R_{00}\left(1+\frac{\phi\Lambda}{rd_H}\right)+\frac{\phi\Lambda}{d_V d_H}+\sqrt{\left[R_{00}\left(1+\frac{\phi\Lambda}{rd_H}\right)+\frac{\phi\Lambda}{d_H d_V}\right]^2-\frac{4\Lambda\phi R_{00}(R_{00}-1)}{rd_h}}}{\frac{2\Lambda\phi R_{00}}{rd_H}},$$

where $R_{00} = \dfrac{\beta_{HV}\beta_{VH}\Lambda K}{d_H d_V(\nu+\alpha+d_H)}$. It is noted that when $p = 0$, R_0 becomes R_{00}. From the definition of p we know that $p = 0$, if either $\lambda = 0$ or $M_0 = 0$. Also $\frac{\partial p}{\partial \lambda} > 0$ and $\frac{\partial p}{\partial M_0} > 0$, which shows that p increases on increasing the value of λ and M_0. So, we assert that in order to eradicate the disease in the population, continuous efforts should be made to maintain a baseline number of awareness programs in the system. Moreover, faster dissemination of awareness also aids in eradication of disease in the system by reducing the value of R_0.

3.3. **Stability of equilibria.** The general variational matrix 'P' for model system (2) is given by:

$$\mathrm{P}=\begin{bmatrix} -a_{11} & -\beta_{HV}Y_V & \beta_{HV}Y_V & a_{14} & 0 & 0 \\ -\lambda M & -a_{22} & \lambda M & 0 & 0 & a_{26} \\ -\alpha & 0 & -d_H & 0 & 0 & 0 \\ a_{41} & -\phi Y_V & 0 & -a_{44} & \beta_{VH}Y_H & 0 \\ 0 & -\phi N_V & 0 & 0 & r-2r\frac{N_V}{K}-\phi A_H & 0 \\ \mu & 0 & 0 & 0 & 0 & -\mu_0 \end{bmatrix}$$

where,

$a_{11} = \beta_{HV}Y_V + \nu + \alpha + d_H$, $a_{14} = \beta_{HV}(N_H - Y_H - A_H)$, $a_{22} = \lambda M + \lambda_0 + d_H$, $a_{26} = \lambda(N_H - Y_H - A_H)$, $a_{41} = \beta_{VH}(N_V - Y_V)$, $a_{44} = \beta_{VH}Y_H + \phi A_H + d_V$.

Let P_i denotes the variational matrix P evaluated at equilibrium E_i (i=0, 1). It is noted that the eigenvalues of P_0 are all negative provided $r < \phi p(\Lambda/d_H)$. Hence, E_0 is stable until $r < \phi p(\Lambda/d_H)$ and it becomes unstable if $r > \phi p(\Lambda/d_H)$ i.e., E_1 exist. Further, for P_1 it is found that four eigenvalues i.e., $-(\lambda M_0 + \lambda_0 + d_H)$, $-d_H$, $-(r - \phi p(\Lambda/d_H))$, $-\mu_0$ are negative. The rest two eigenvalues are roots of following quadratic equation

$$\xi^2 + q_1\xi + q_2 = 0, \tag{15}$$

where,

$q_1 = \nu + \alpha + d_H + \phi p(\Lambda/d_H) + d_V$,

$q_2 = -\beta_{HV}\beta_{VH}\dfrac{(1-p)\Lambda K(r-\phi p(\Lambda/d_H))}{rd_H(\phi(\Lambda/d_H)p + d_V)} + (\nu+\alpha+d_H)$.

It is apparent that if $q_2 > 0$ then the roots of equation (15) will be either negative or with negative real part. On the contrary if $q_2 < 0$, then one root of equation

(15) will be positive. In this case E_1 will become unstable either in Y_H-direction or in Y_V-direction. It is interesting to note here that q_2 becomes negative if $R_0 > 1$, which implies the existence of E^*. So we infer that E_1 becomes unstable whenever E^* exits.

Hence, the results for local stability behavior of both disease-free equilibria (DVFE and DFE) of model system (2) are stated in the form of following theorem:

Theorem 3.1. *The DVFE always exists and is locally asymptotically stable if $r < \phi p(\Lambda/d_H)$. Whenever r is greater than $\phi p(\Lambda/d_H)$, DVFE becomes unstable and DFE exists which is stable until $R_0 < 1$.*

In order to attain full characterization of the endemic equilibrium, we derive the conditions for the local and global stability of E^* using Liapunov's method. The results obtained are presented in following theorems:

Theorem 3.2. *The endemic equilibrium, if exists, is locally asymptotically stable provided the following conditions hold,*

$$\frac{\beta_{HV}^2 {Y_V^*}^2 \lambda^2}{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)(\lambda M^* + \lambda_0 + d_H)^2} < \frac{4}{45}\min\left\{\frac{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)}{5{M^*}^2}, \frac{\beta_{HV}Y_V^* d_H}{\alpha {M^*}^2}, \frac{\mu_0^2(\beta_{HV}Y_V^* + \nu + \alpha + d_H)}{6\mu^2(N_H^* - Y_H^* - A_H^*)^2}\right\}, \tag{16}$$

$$\frac{5\beta_{HV}^2(N_H^* - Y_H^* - A_H^*)^2}{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)(\beta_{VH}Y_H^* + \phi A_H^* + d_V)^2} < \min\left\{\frac{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)}{5\beta_{VH}^2(N_V^* - Y_V^*)^2}, \frac{(\lambda M^* + \lambda_0 + d_H)k_1}{6\phi^2 {Y_V^*}^2}\right\}, \tag{17}$$

$$\frac{\beta_{VH}^2 {Y_H^*}^2}{\beta_{VH}Y_H^* + \phi A_H^* + d_V} k_3 < \frac{(\lambda M^* + \lambda_0 + d_H)r^2}{6K^2\phi^2} k_1, \tag{18}$$

where, k_1 and k_3 are chosen from inequalities (33) and (34), respectively.

Theorem 3.3. *The endemic equilibrium is globally asymptotically stable in Ω, provided the following inequalities hold,*

$$\frac{\beta_{HV}^2 {Y_V^*}^2 \lambda^2}{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)(\lambda_0 + d_H)^2} < \frac{4}{45}\min\left\{\frac{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)}{5M_R^2}, \frac{\beta_{HV}Y_V^* d_H}{\alpha M_R^2}, \frac{\mu_0^2(\beta_{HV}Y_V^* + \nu + \alpha + d_H)}{6\mu^2(N_H^* - Y_H^* - A_H^*)^2}\right\}, \tag{19}$$

$$\frac{5\beta_{HV}^2\Lambda^2}{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)d_V^2 d_H^2} < \min\left\{\frac{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)}{5\beta_{VH}^2(N_V^* - Y_V^*)^2}, \frac{(\lambda_0 + d_H)p_1}{6\phi^2 {Y_V^*}^2}\right\}, \tag{20}$$

$$\frac{\beta_{VH}^2\Lambda^2}{d_H^2 d_V} p_3 < \frac{(\lambda_0 + d_H)r^2}{6K^2\phi^2} p_1, \tag{21}$$

where, p_1 and p_3 are chosen from inequalities (48) and (49), respectively.

For proof of theorems 3.2 and 3.3 see Appendix B and C respectively.

4. **Numerical simulation.** The model has been analyzed numerically using MATLAB 7.5.0., to confirm the results obtained analytically as well as to gain more profound insight about the model. For this purpose we have used the following set of parameter values:
$\Lambda = 3$, $r = 0.5$, $K = 15000$, $\beta_{HV} = 0.0008$, $\beta_{VH} = 0.000002$, $\lambda = 0.001$, $\lambda_0 = 0.05$, $\nu = 0.15$, $\alpha = 0.005$, $d_H = 0.00005$,$\phi = 0.00000001$, $d_V = 0.05$, $\mu = 0.0015$, $\mu_0 = 0.22$, $M_0 = 5$.

These parameter values satisfy the conditions for existence of endemic equilibrium and the equilibrium values for E^* are obtained as:
$Y_H = 589.77$, $A_H = 66.22$, $N_H = 1023.38$, $Y_V = 311.13$, $N_V = 13499.96$, $M = 9.02$.

The value of basic reproduction number, R_0 is obtained as 168.47. The stability conditions are also satisfied for these parameter values. The eigenvalues of variational matrix corresponding to E^* are obtained as -0.4499, -0.4293, -0.2164, -0.0582, -0.0251 and -0.0052. It is apparent that all the eigenvalues are negative, which shows that system is locally asymptotically stable. To show the non-linear stability behavior of (Y_H^*, M^*) and (A_H^*, Y_V^*) in their respective planes, we have plotted the solution trajectories using above set of parameter values with different initial starts. These trajectories are shown in figures 1 and 2, respectively. It is easy to note that all the trajectories initiating inside the region of attraction are approaching towards the equilibrium values.

The variation of Y_H w.r.t. time t for different values of λ, ϕ and μ is given in figures 3, 4 and 5, respectively. It is apparent from these figures that on increasing the value of λ, ϕ and μ, the infective human population Y_H decreases. Therefore, to lessen the magnitude of infective human population in the system, the value of rate of dissemination of awareness, rate of deterioration of vector population due to aware population and rate of executing awareness programs must be sufficiently large. Also, from figure 4, it is interesting to note that for higher value of ϕ, infective human population becomes zero. This indicates that if aware people make effectual efforts to control the abundance of vector population, disease can be eradicated. Further, the variation of total vector population N_V w.r.t. time t for different values of λ and μ is given in figures 6 and 7. It is evident from these figures that as the value of λ and μ increases, total vector population N_V and consequently infective vector population Y_V decreases. So, we infer that by faster dissemination of awareness and execution of awareness programs, abundance of vectors in the system can be controlled.

5. **Conclusion.** Outbreaks of vector borne diseases like malaria, dengue, chikungunya, yellow fever, etc., have devastated several countries around the globe. Due to this, modelling their transmission and control has gained enormous attention. In this paper, we have studied the impact of awareness as a novel intervention for the control of vector borne diseases. In the modelling process, it is assumed that media campaigns create awareness regarding personal protection as well as control of vectors. As a result of this, behavioral changes occur within the human population, which results in the formation of a new class i.e., aware class. The individuals

of this class not only protect themselves from vector bites and hence contracting infection, but also aid in reducing the abundance of vectors in the environment. We have considered that media campaigns are executed in accordance with the size of epidemic. Therefore, as per the classification provided by Funk *et al.*[8], the present model considers prevalence based global information where behavior changes the disease state of individuals. The model exhibits three non-negative equilibria: DVFE, DFE and endemic equilibrium. Until the intrinsic growth rate of vector is less than their deterioration rate due to aware population, only DVFE exists. But as the intrinsic growth rate of vector becomes higher than their deterioration rate, DVFE becomes unstable which leads to the existence of DFE. DFE remains stable until the value of the basic reproduction number, $R_0 < 1$, but as the value of R_0 exceeds 1, DFE becomes unstable, and endemic equilibrium exists, which is a standard result in epidemiology. The endemic equilibrium, if exists, is locally as well as non-linearly stable under certain conditions. The model analysis suggests that if media campaigns have enough potential to mobilize higher fraction of the population, the abundance of vectors 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, R_0. This implies that, the presence of awareness in the population makes the disease invasion difficult. Also, by continuous efforts and effectual media coverage disease can be eradicated. Further, the stability conditions infer that media interventions destabilize the system. Moreover, execution of more awareness campaigns and faster dissemination of awareness, will mobilizes a large fraction of the population. This in turn will widen the aware population and hence slow down the prevalence of diseases. The present work provides a basic framework to assess the impact of media and awareness programs on the prevalence of vector borne infectious diseases. However, the proposed model is much suitable for malaria and the diseases with akin dynamics, it can readily be extended for other diseases like dengue, chikungunya, etc., by incorporating recovered and exposed class in the modelling process.

Appendix A. **Next generation matrix approach.** The basic reproduction number R_0 for model system (2) can also be estimated using next generation matrix (NGM) approach following [3]. We have the transmission matrix T and transition matrix Σ as,

$$T = \begin{bmatrix} 0 & 0 & 0 & \beta_{HV}(1-p)\frac{\Lambda}{d_H} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \beta_{VH}K_R & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \text{ and}$$

$$\Sigma = \begin{bmatrix} -s_{11} & 0 & 0 & 0 & 0 & 0 \\ -\lambda M_0 & -s_{22} & \lambda M_0 & 0 & 0 & \lambda(1-p)\frac{\Lambda}{d_H} \\ -\alpha & 0 & -d_H & 0 & 0 & 0 \\ 0 & 0 & 0 & -\phi p\frac{\Lambda}{d_H} - d_V & 0 & 0 \\ 0 & -\phi K_R & 0 & 0 & -r\frac{K_R}{K} & 0 \\ \mu & 0 & 0 & 0 & 0 & -\mu_0 \end{bmatrix},$$

where $s_{11} = (\nu + \alpha + d_H)$, $s_{22} = (\lambda M_0 + \lambda_0 + d_H)$.

The NGM for large domain $\mathcal{K}_L = -T\Sigma^{-1}$ is six-dimensional,

$$\mathcal{K}_L = T\Sigma^{-1} = \begin{bmatrix} 0 & 0 & 0 & \frac{\beta_{HV}(1-p)\Lambda}{d_H(p\phi\Lambda/d_H+d_V)} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_{HV}K_R}{(\nu+\alpha+d_H)} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

It is apparent that there are only two states-at-infection out of six. The NGM $\mathcal{K}$ is therefore two dimensional. So, we consider an auxiliary matrix E given by,

$$E = \begin{bmatrix} 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 1 & 0 & 0 \end{bmatrix}^T.$$

Now, we compute NGM $\mathcal{K}$ using formula $\mathcal{K} = E^T\mathcal{K}_L E$ which yields,

$$\mathcal{K} = \begin{bmatrix} 0 & \frac{\beta_{HV}(1-p)\Lambda}{d_H(p\phi\Lambda/d_H+d_V)} \\ \frac{\beta_{HV}K_R}{(\nu+\alpha+d_H)} & 0 \end{bmatrix}.$$

The basic reproduction number $\mathcal{R}_0$ is spectral radius of matrix $\mathcal{K}$ and can be obtained as follows,

$$\mathcal{R}_0 = \rho(\mathcal{K}) = \sqrt{\frac{\beta_{HV}\beta_{VH}(1-p)\Lambda K_R}{d_H(\phi p(\Lambda/d_H)+d_V)(\nu+\alpha+d_H)}}.$$

It is noted here that the value of basic reproduction number obtained by using NGM approach ($\mathcal{R}_0$) is square-root of basic reproduction number obtained by inspection from condition of existence of equilibrium E^* (R_0) i.e., $\mathcal{R}_0 = \sqrt{R_0}$.

Appendix B. Proof of Theorem 3.2.

Proof. Consider a Liapunov's function as,

$$V = \frac{1}{2}y_1^2 + \frac{k_1}{2}a_1^2 + \frac{k_2}{2}n_1^2 + \frac{k_3}{2}y_2^2 + \frac{k_4}{2N_V^*}n_2^2 + \frac{k_5}{2}m^2, \tag{22}$$

where, k_1, k_2, k_3, k_4 and k_5 are positive constants to be chosen appropriately. Here y_1, a_1, n_1, y_2, n_2, and m are small perturbations in Y_H, A_H, N_H, Y_V, N_V and M around the equilibrium E^*, respectively. Now differentiating 'V' with respect to 't' and using the linearized system of model system (2) corresponding to E^*, we get

$$\begin{aligned} \dot{V} \;=\; & y_1[-a_{11}^*y_1 - \beta_{HV}Y^*a_1 + \beta_{HV}Y^*n_1 + a_{14}^*y_2] \\ & -k_1a_1[\lambda M^*y_1 + a_{22}^*a_1 + \lambda M^*n_1 + a_{26}^*m] \\ & -k_2n_1[\alpha y_1 + d_Hn_1] \\ & +k_3y_2[a_{41}^*y_1 - \phi Y_V^*a_1 - a_{44}^*y_2 + \beta_{VH}Y_H^*n_2] \\ & -k_4n_2[\phi a_1 + (r/K)n_2] \\ & +k_5m[\mu y_1 - \mu_0 m]. \end{aligned}$$

Here, a_{ij}^* denotes the values of a_{ij} in variational matrix P evaluated at E^*. After choosing $k_2 = \beta_{HV} Y_V^*/\alpha$, a little algebraic manipulation yields,

$$\begin{aligned}\dot{V} &= -a_{11}^* y_1^2 - k_1 a_{22}^* a_1^2 - k_2 d_H n_1^2 - k_3 a_{44}^* y_2^2 - k_4 (r/K) n_2^2 - k_5 \mu_0 m^2 \\ &\quad -[\beta_{HV} Y_V^* + k_1 \lambda M^*] y_1 a_1 + [a_{14}^* + k_3 a_{41}^*] y_1 y_2 + k_5 \mu y_1 m \\ &\quad + k_1 \lambda M^* a_1 n_1 - k_3 \phi Y_V^* a_1 y_2 - k_4 \phi a_1 n_2 + k_1 a_{26}^* a_1 m \\ &\quad + k_3 \beta_{VH} Y_H^* y_2 n_2.\end{aligned}$$

Now, $\dot{V}$ will be negative definite provided the following inequalities are satisfied,

$$\beta_{HV}^2 Y_V^{*2} < \frac{2}{15} k_1 a_{11}^* a_{22}^*, \tag{23}$$

$$k_1 \lambda^2 M^{*2} < \frac{2}{15} a_{11}^* a_{22}^*, \tag{24}$$

$$a_{14}^{*\,2} < \frac{1}{5} k_3 a_{11}^* a_{44}^*, \tag{25}$$

$$k_3 a_{41}^{*\,2} < \frac{1}{5} a_{11}^* a_{44}^*, \tag{26}$$

$$k_5 \mu^2 < \frac{2}{5} a_{11}^* \mu_0, \tag{27}$$

$$k_1 \lambda^2 M^{*2} < \frac{2}{3} \frac{\beta_{HV} Y_V^* d_H a_{22}^*}{\alpha}, \tag{28}$$

$$k_3 \phi^2 Y_V^{*2} < \frac{1}{6} k_1 a_{22}^* a_{44}^*, \tag{29}$$

$$k_4 \phi^2 < \frac{1}{3} k_1 \frac{r}{K} a_{22}^*, \tag{30}$$

$$k_1 a_{26}^{*\,2} < \frac{1}{3} k_5 a_{22}^* \mu_0, \tag{31}$$

$$k_3 \beta_{VH}^2 Y_H^{*2} < \frac{1}{2} k_4 \frac{r}{K} a_{44}^*. \tag{32}$$

From inequality (27), we can choose $k_5 = \dfrac{2 a_{11}^* \mu_0}{6\mu^2}$. Now using this value of k_5, we can choose a positive value of k_1 from inequalities (23), (24), (28) and (31), as

$$\frac{15}{2} \frac{\beta_{HV}^2 Y_V^{*2}}{a_{11}^* a_{22}^*} < k_1 < \min\left\{ \frac{2}{15} \frac{a_{11}^* a_{22}^*}{\lambda^2 M^{*2}}, \frac{2}{3} \frac{\beta_{HV} Y_V^* d_H a_{22}^*}{\lambda^2 M^{*2} \alpha}, \frac{1}{3} \frac{k_5 \mu_0 a_{22}^*}{a_{26}^{*\,2}} \right\}. \tag{33}$$

Using inequalities (25), (26) and (29), a positive value of k_3 can be chosen as

$$\frac{5 a_{14}^{*\,2}}{a_{11}^* a_{44}^*} < k_3 < \min\left\{ \frac{a_{11}^* a_{44}^*}{5 a_{41}^{*\,2}}, \frac{a_{22}^* a_{44}^* k_1}{6 \phi^2 Y_V^{*2}} \right\}. \tag{34}$$

Finally using values of k_1 and k_3 as chosen above, in inequalities (30) and (32) we may choose a positive value of k_4 provided following inequality holds,

$$\frac{\beta_{VH}^2 Y_H^{*\,2}}{a_{44}^*} k_3 < \frac{r^2 a_{22}^*}{6 K^2 \phi^2} k_1. \tag{35}$$

Hence the proof. □

Appendix C. Proof of Theorem 3.3.

Proof. To study the global stability behaviour of endemic equilibrium, consider the following positive definite function,

$$W = \frac{1}{2}(Y_H - Y_H^*)^2 + \frac{p_1}{2}(A_H - A_H^*)^2 + \frac{p_2}{2}(N_H - N_H^*)^2 + \frac{p_3}{2}(Y_V - Y_V^*)^2 + p_4(N_V - N_V^* - N_V^* \ln \frac{N_V}{N_V^*}) + \frac{p_5}{2}(M - M^*)^2 \tag{36}$$

where the coefficients p_1, p_2, p_3, p_4 and p_5 are positive constants to be chosen suitably later on. Differentiating (36) with respect to 't' along the solutions of model system (2), we get

$$\begin{aligned}
\dot{W} = {} & (Y_H - Y_H^*)[\beta_{HV}\{(N_H - Y_H - A_H)Y_V - (N_H^* - Y_H^* - A_H^*)Y_V^*\} \\
& -(\nu + \alpha + d_H)(Y_H - Y_H^*)] \\
& +p_1(A_H - A_H^*)[\lambda\{(N_H - Y_H - A_H)M - (N_H^* - Y_H^* - A_H^*)M^*\} \\
& -(\lambda_0 + d_H)(A_H - A_H^*)] \\
& +p_2(N_H - N_H^*)[-d_H(N_H - N_H^*) - \alpha(Y_H - Y_H^*)] \\
& +p_3(Y_V - Y_V^*)[\beta_{VH}\{Y_H(N_V - Y_V) - Y_H^*(N_V^* - Y_V^*)\} \\
& -\phi(A_H Y_V - A_H^* Y_V^*) - d_V(Y_V - Y_V^*)] \\
& +p_4(N_V - N_V^*)[-\frac{r}{K}(N_V - N_V^*) - \phi(A_H - A_H^*)] \\
& +p_5(M - M^*)[\mu(Y_H - Y_H^*) - \mu_0(M - M^*)].
\end{aligned} \tag{37}$$

On rearranging the terms and setting $p_2 = \beta_{HV} Y_V^*/\alpha$, $\dot{W}$ reduces to,

$$\begin{aligned}
\dot{W} = {} & -(\beta_{HV} Y_V^* + \nu + \alpha + d_H)(Y_H - Y_H^*)^2 - p_1(\lambda M + \lambda_0 + d_H)(A_H - A_H^*)^2 \\
& -\frac{\beta_{HV} d_H Y_V^*}{\alpha}(N_H - N_H^*)^2 - p_3(\beta_{VH} Y_H + \phi A_H + d_V)(Y_V - Y_V^*)^2 \\
& -p_4 \frac{r}{K}(N_V - N_V^*)^2 - p_5 \mu_0 (M - M^*)^2 \\
& +[\beta_{HV}(N_H - Y_H - A_H) + p_3 \beta_{VH}(N_V^* - Y_V^*)](Y_H - Y_H^*)(Y_V - Y_V^*) \\
& -[\beta_{HV} Y_V^* + p_1 \lambda M](Y_H - Y_H^*)(A_H - A_H^*) + p_5 \mu (Y_H - Y_H^*)(M - M^*) \\
& +p_1 \lambda M (A_H - A_H^*)(N_H - N_H^*) - p_3 \phi Y_V^* (A_H - A_H^*)(Y_V - Y_V^*) \\
& -p_4 \phi (A_H - A_H^*)(N_V - N_V^*) \\
& +p_1 \lambda (N_H^* - Y_H^* - A_H^*)(A_H - A_H^*)(M - M^*) \\
& +p_3 \beta_{VH} Y_H (Y_V - Y_V^*)(N_V - N_V^*).
\end{aligned}$$

Using region of attraction Ω, we infer that $\dot{W}$ will be a negative definite provided the following inequalities hold:

$$\beta_{HV}^2 {Y_V^*}^2 < \frac{2}{15}p_1(\beta_{HV}Y_V^* + \nu + \alpha + d_H)(\lambda_0 + d_H), \tag{38}$$

$$p_1\lambda^2 M_R^2 < \frac{2}{15}(\beta_{HV}Y_V^* + \nu + \alpha + d_H)(\lambda_0 + d_H), \tag{39}$$

$$\frac{\beta_{HV}^2\Lambda^2}{d_H^2} < \frac{1}{5}p_3(\beta_{HV}Y_V^* + \nu + \alpha + d_H)d_V, \tag{40}$$

$$p_3\beta_{VH}^2(N_V^* - Y_V^*)^2 < \frac{1}{5}(\beta_{HV}Y_V^* + \nu + \alpha + d_H)d_V, \tag{41}$$

$$p_5\mu^2 < \frac{2}{5}(\beta_{HV}Y_V^* + \nu + \alpha + d_H)\mu_0, \tag{42}$$

$$p_1\lambda^2 M_R^2 < \frac{2}{3}\frac{\beta_{HV}Y_V^* d_H(\lambda_0 + d_H)}{\alpha}, \tag{43}$$

$$p_3\phi^2 {Y_V^*}^2 < \frac{1}{6}p_1(\lambda_0 + d_H)d_V, \tag{44}$$

$$p_4\phi^2 < \frac{1}{3}p_1\frac{r(\lambda_0 + d_H)}{K}, \tag{45}$$

$$p_1\lambda^2(N_H^* - Y_H^* - A_H^*)^2 < \frac{1}{3}p_5\mu_0(\lambda_0 + d_H), \tag{46}$$

$$p_3\frac{\Lambda^2\beta_{VH}^2}{d_H^2} < \frac{1}{2}p_4\frac{rd_V}{K}, \tag{47}$$

From (42), a positive value of p_5 can be chosen as $p_5 = \dfrac{2(\beta_{HV}Y_V^* + \nu + \alpha + d_H)\mu_0}{6\mu^2}$.

Further, using inequalities (38), (39), (43) and (46), we may choose a positive value of p_1 as follows

$$\frac{15\beta_{HV}^2 {Y_V^*}^2}{2(\beta_{HV}Y_V^* + \nu + \alpha + d_H)(\lambda_0 + d_H)} < p_1 < \min\left\{\frac{2(\beta_{HV}Y_V^* + \nu + \alpha + d_H)(\lambda_0 + d_H)}{15\lambda^2 M_R^2}, \frac{2\beta_{HV}Y_V^*(\lambda_0 + d_H)d_H}{3\alpha\lambda^2 M_R^2}, \frac{\mu_0(\lambda_0 + d_H)p_5}{3\lambda^2(N_H^* - Y_H^* - A_H^*)^2}\right\}. \tag{48}$$

Further, from inequalities (40), (41) and (44), we can choose a positive value of p_3 as,

$$\frac{5\beta_{HV}^2\Lambda^2}{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)d_V d_H^2} < p_3 < \min\left\{\frac{(\beta_{HV}Y_V^* + \nu + \alpha + d_H)d_V}{\beta_{VH}^2(N_V^* - Y_V^*)^2}, \frac{p_1(\lambda_0 + d_H)d_V}{6\phi^2 {Y_V^*}^2}\right\}. \tag{49}$$

Using positive value of p_1 and p_3 as obtained from (48) and (49) in inequalities (45) and (47) respectively, we may choose a positive value of p_4 if the following inequality is satisfied,

$$\frac{\beta_{VH}^2\Lambda^2}{d_H^2 d_V}p_3 < \frac{(\lambda_0 + d_H)r^2}{6K^2\phi^2}p_1. \tag{50}$$

Hence, we made the assertion that W is a Liapunov's function for model system (2), provided conditions (19), (20) and (21) hold. □

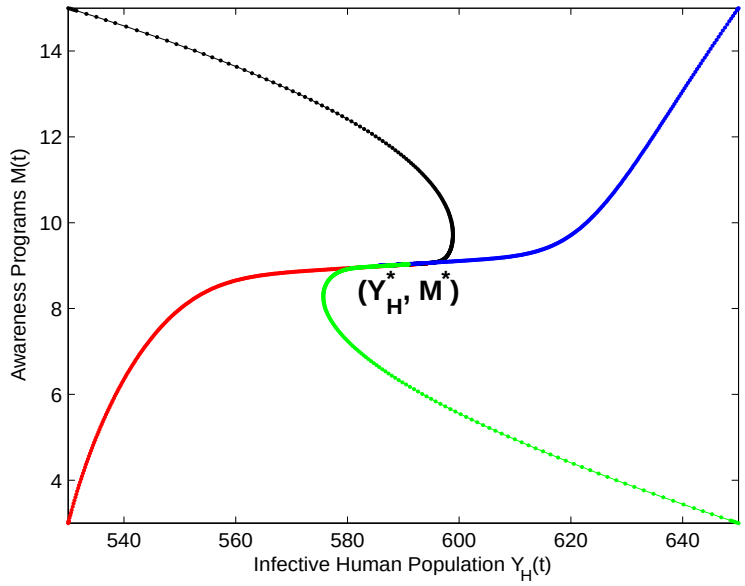

FIGURE 1. Nonlinear stability of (Y_H^*, M^*) in $Y_H - M$ plane

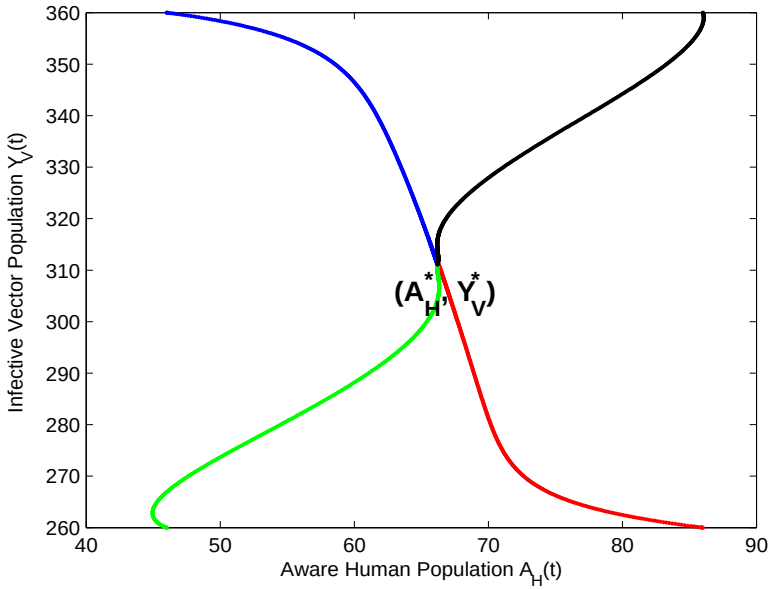

FIGURE 2. Nonlinear stability of (A_H^*, Y_V^*) in $A_H - Y_V$ plane

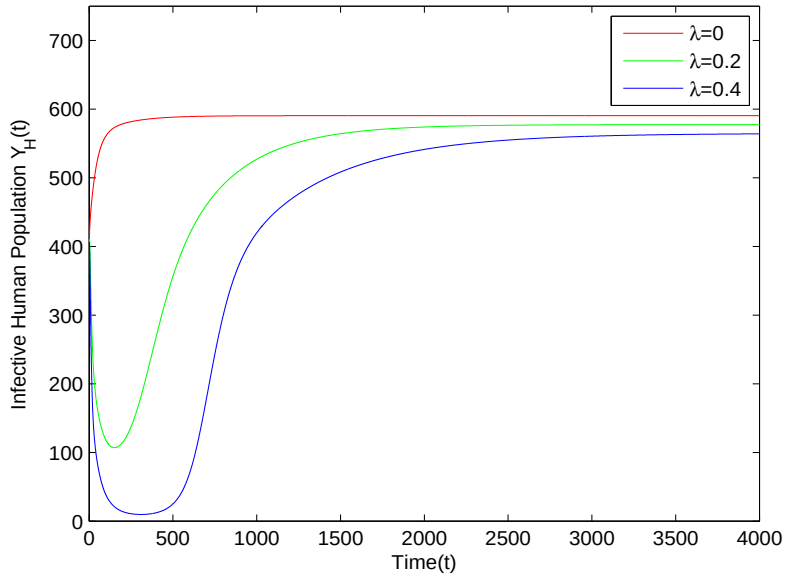

FIGURE 3. Variation of infective human population $Y_H(t)$ w.r.t. time (t) for different values of λ

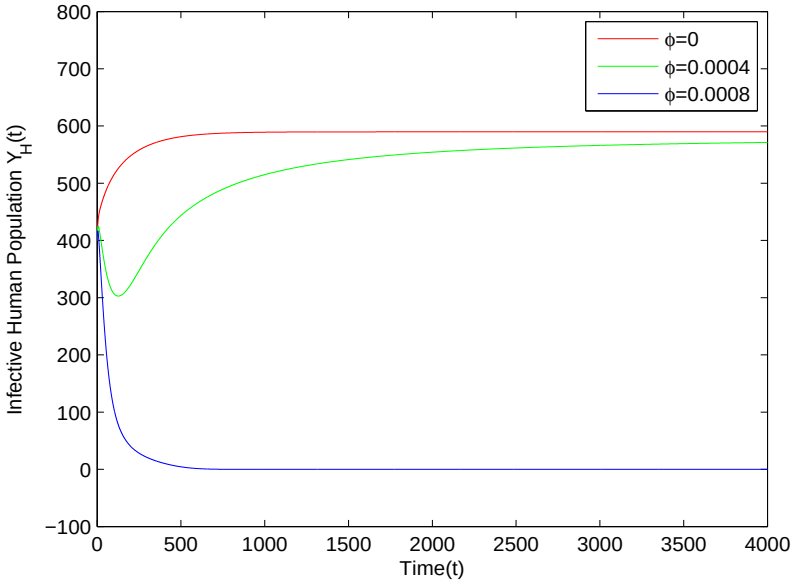

FIGURE 4. Variation of infective human population $Y_H(t)$ w.r.t. time (t) for different values of ϕ

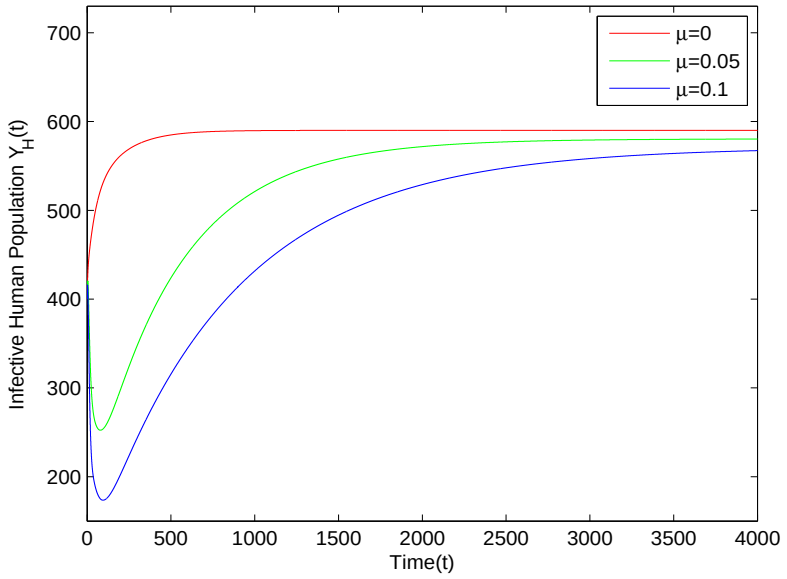

FIGURE 5. Variation of infective human population $Y_H(t)$ w.r.t. time (t) for different values of μ

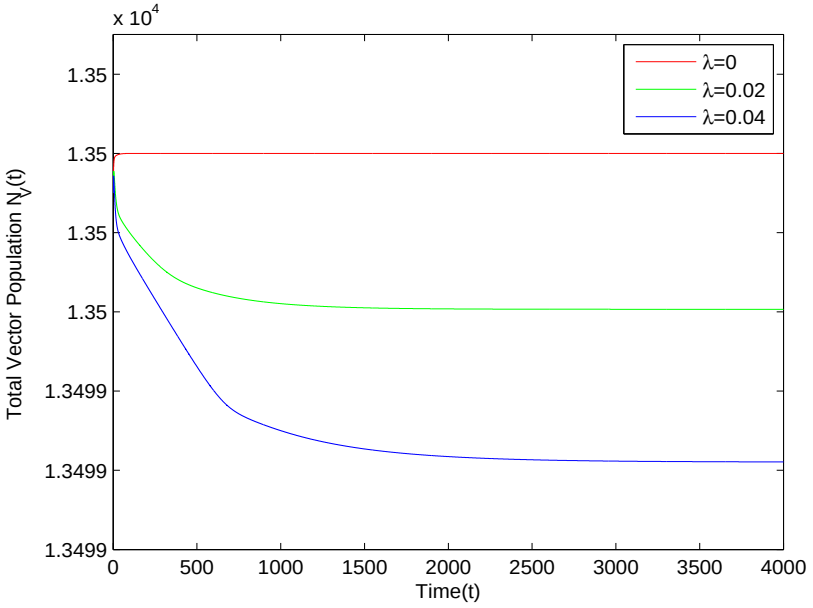

FIGURE 6. Variation of total vector population $N_V(t)$ w.r.t. time (t) for different values of λ

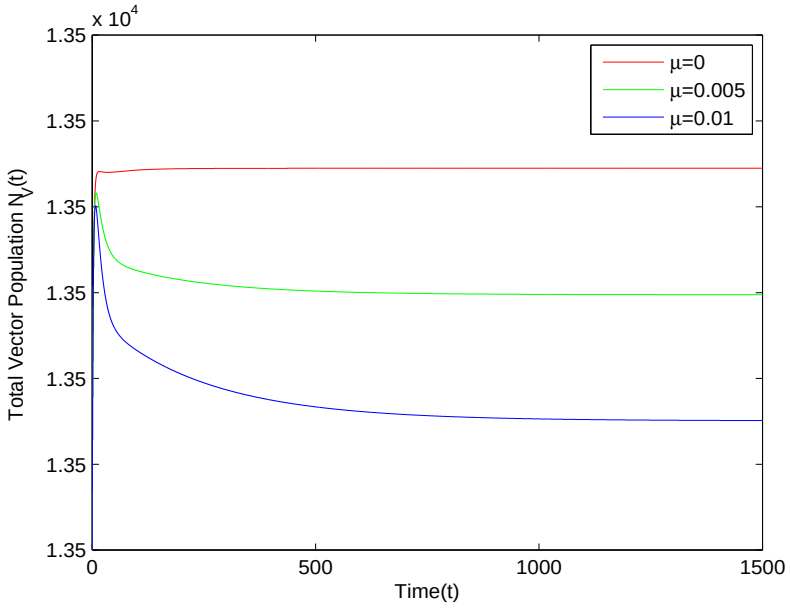

FIGURE 7. Variation of total vector population $N_V(t)$ w.r.t. time (t) for different values of μ

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