Article

The impact of media coverage on the dynamics of vector-borne diseases

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Abstract

In this paper, the impact of media coverage on the transmission of vector borne diseases has been investigated. The model exhibits two equilibria: a disease-free and a unique endemic equilibrium. The model is analyzed using Hurwitz criterion and Geometric approach. The numerical simulations of model reveals that, the effects of media coverage on the transmission of vector-borne disease endemic may decrease the peak value of the infectives or the average number of the infectives in different cases.

Keywords vector-borne diseases; media coverage; global stability; numerical simulation.

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1 Introduction

When an infectious disease appears and starts spreading in a region, there is a need for effective treatment to control the epidemic (Shams and Khansari, 2019; Zhang et al., 2020; Naresh et al., 2021). The board for disease control and prevention will do everything possible to avert the disease spreading (Saranya et al., 2022). The preventive measures include making individual familiar people with the right preventive knowledge of the disease as soon as possible through media and education. The knowledge about the transmission modality of infection can help them to take necessary preventive measures to avoid infection. Media coverage may play a crucial role in mitigating contact rate of human beings as observed during the spreading of severe acute respiratory syndrome (SARS) during 2002 and 2004 SARS (Wang and Ruan, 2004; Webb et al., 2004). The public's devotion to their anticipated opinions is the significant obstacle to the adoption of any disease management strategy. People have several misconceptions regarding vectors and their breeding spots in rural and semi-rural regions of nations where these diseases are more endemic (Adedotun et al., 2010; Tyagi et al., 2005). However, if correct information is provided on time, individuals may change their behaviour to reduce their chance of contacting infection and adjust the future of epidemic.

The findings of surveys (Rahman and Rahman, 2007; Hossain et al., 2014) suggest that people who

regularly read articles or magazine or watch television corresponding to the public health, are more aware about the transmission of infection in comparison to those who do not do so. The change in behaviour of susceptible facing infection is due to the result of awareness programs that are run by media campaigns. The effort of information realised through media emphasize on the awearness of individual's about the transmission of infection and promote steps that can decrease the chance of being infected. (Kiss et al., 2010; Samanta et al., 2013) have proposed mathematical models analysing the the impact of media-sponsored public awareness campaigns on the transmission and control of epidemic. Cui et al. (2008) have studied impact of the media coverage on the spreading and control of the disease by considering a new incidence function. Many authors (Chunara et al., 2012; Misra et al., 2013, 2011a, b) have proposed diseases transmission models in a more evident way by incorporating media coverage into epidemic model. The media coverage or awareness programs are represented by equation or equations and included into disease transmission models. The compartmental disease models can also be incorporated by being associated to the changes in behaviour of individuals. The behavioural changes are expressed in terms of contact rates or other model terms and hence, the infection or incidence rates (Cui et al., 2008; Mummert and Weiss, 2013; Tchuenche et al., 2011; Xiao et al., 2015). Lu et al. (2017) introduced the change in behavior of an individual towards the infected individuals due to the awareness by media and analyzed an SEI model to study the impact of media on the spread and control of infectious diseases. Misra et al. (2018) studied the effect of television and social media advertisement on the transmission of infectious disease. Their analysis reviles that the increase in growth rate of cumulative number of television and social media advertisement after a threshold value destabilizes the system and periodic oscillations arise through Hopf-bifurcation. Srivastav et al. (2019) proposed mathematical model to study the effectiveness of the role of media on the spread of Zika virus. The study of model shows that media has a beneficial impact on the infected human as increase in media parameters, the equilibrium level of infected humans decreases. Kar et al. (2019) analyzed an SEIR epidemic model to assess the effect of media, considering saturated type of incidence rate and treatment control function. The media is assumed to be considered as treatment control as well as another disease control measure. Salman (2021) formulated an HIV/AIDS infectious disease model to study the effect of media coverage on the spread of the disease by incorporating a nonlinear function of the number of infective in the transmission term. In this article, we will focus at how media coverage affects the dynamics of host-vector dynamics by considering a Ross-Macdonald type model with a kind of new contact rate, where the population of the vector is described by a system for the susceptible and infected vector while the dynamics of the host is described by an SIR model.

The main purpose of this paper is to study the effectiveness of media coverage on vector-borne disease by including the new contact rate in transmission rate. Accordingly, we formulate a vector-host model in section 2. We calculate the reproduction number and the stability of disease-free equilibrium in section 3. The existence of the endemic equilibrium and proof of the stability of endemic equilibrium are discussed in Section 4. Numerical simulation and conclusion are given in Section 5 and Section 6, respectively. Appendices A and B explains the basic structure used to prove the global stability of endemic equilibrium.

2 Mathematical Model

In this section we assume that total population size at time t, given by $N_1(t)$, is divided into subclasses of individuals who are susceptible population of size $S(t)$, Infectious population of size $I(t)$ and recovered population of size $R(t)$. Thus $N_1(t) = S(t) + I(t) + R(t)$. Furthermore, the total vector population size at time t is assumed to be $N_2(t)$ and is divided into subclasses of vectors that are susceptible vector of size $M(t)$ and infected vector of size $V(t)$. So we have $N_2(t) = M(t) + V(t)$. The mathematical model can be represented by the following non-linear differential equations.

$$\frac{dS}{dt} = b_1 - \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) SI - \beta_1 SV - \mu_1 S,$$
$$\frac{dI}{dt} = \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) SI + \beta_1 SV - (d + \mu_1 + \gamma) I,$$
$$\frac{dR}{dt} = \gamma I - \mu_1 R \qquad (1)$$
$$\frac{dM}{dt} = b_2 - \beta_2 MI - \mu_2 M,$$
$$\frac{dV}{dt} = \beta_2 MI - \mu_2 V,$$

where b_1 and b_2 are the birth or immigrant rate for host and vector population respectively. We assume that vertical transmission in the host does not occur so that all newly recruited individuals are susceptible. β_1 is the rate of Infection spread by the pathogen-carrier vectors. The per capita recovery rate of the hosts is given by γ. λ_1 is the contact rate before media alert; $\beta(I) = \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right)$ denotes the contact rate after media alert and brings down the transmission rate when the infection appear and cases are reported through media. The reduced rate of the transmission tends to its maximum value λ_1, as $I \to \infty$ and when the infectious individuals are reported at m, the transmission rate equals half of the maximum λ_2 (Liu and Cui, 2008). Since it is not possible that the media reports can completely cease the disease from spreading, therefore, $\lambda_1 \geq \lambda_2$. m is the reactive velocity of people and media coverage to the disease. d is disease induced death rate of host population. μ_1 and μ_2 natural death rates of host and vector population respectively. Since the recovered population R appears only in third equation of system (1). We have

$$N_1' = b_1 - \mu_1 N_1$$

which is derived by adding the host population of the system (1) and also,

$$N_2' = b_2 - \mu_2 N_2,$$

which is derived by adding the vector population of the system (1).

It is clear that $\lim_{t\to\infty} N_1(t) = \frac{b_1}{\mu_1}$ and $\lim_{t\to\infty} N_2(t) = \frac{b_2}{\mu_2}$.

Therefore, the system (1) can be reduced as:

$$\frac{dS}{dt} = b_1 - \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) SI - \beta_1 SV - \mu_1 S,$$
$$\frac{dI}{dt} = \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) SI + \beta_1 SV - (d + \mu_1 + \gamma) I, \qquad (2)$$
$$\frac{dV}{dt} = \beta_2 \left(\frac{b_2}{\mu_2} - V\right) I - \mu_2 V.$$

The values of R and M can be determined correspondingly from $R = \frac{b_1}{\mu_1} - S - I$, and $M = \frac{b_2}{\mu_1} - V$ or from $R' = \gamma I - \mu_1 R$ and $M' = b_2 - \beta_2 MI - \mu_2 M$, respectively. We need the non-negative solutions for biological reasons. The solutions lead us to study the system (2) in the closed set

$$\Omega = \{(S, I, V) \in \mathbf{R}_+^3 | 0 \leq S + I \leq \frac{b_1}{\mu_1}, \quad 0 \leq V \leq \frac{b_2}{\mu_2}, \quad S \geq 0, I \geq 0, V \geq 0\},$$

where $\mathbf{R}^3_+$ denotes the non-negative cone of $\mathbf{R}^3$ including its lower dimensional faces. It can be verified that Ω is positively invariant with respect to the system (2).

3 Disease Free Equilibrium and Reproduction Number

The equilibria for the model (2), can be obtained by setting right hand side of model (2) equal to zero. The model clearly has a unique disease free equilibrium point E_0 in the region Ω, given by $E_0 = \left(\frac{b_1}{\mu_1}, 0, 0\right)$.

We will obtain the expression of reproduction number. The reproduction number denoted by $\mathcal{R}_0$ and denoted by the number of secondary infection produced when a single infectious host is introduced into a totally susceptible population. We use the next generation matrix method described in Driessche and Watmough (2002) to define the reproduction number $\mathcal{R}_0$ as:

Let $x = (S, I, V)^T$. Then the model (2) can be written as:

$$\frac{dx}{dt} = \mathcal{F}(x) - \mathcal{V}(x),$$

where

$$\mathcal{F}(x) = \begin{pmatrix} \alpha_1 S^0 & \beta_1 S^0 \\ 0 & 0 \end{pmatrix} \text{and } \mathcal{V}(x) = \begin{pmatrix} d + \mu_1 + \gamma & 0 \\ -\frac{\beta_2 b_2}{\mu_2} & \mu_2 \end{pmatrix}$$

$$\mathcal{F}\mathcal{V}^{-1} = \begin{pmatrix} \left(\frac{\lambda_1 S^0}{(d+\gamma+\mu_1)} + \frac{\beta_1 \beta_2 S^0}{(d+\gamma+\mu_1)}\right) & \frac{\beta_1 S^0}{\mu_2} \\ 0 & 0 \end{pmatrix}.$$

The reproduction number $\mathcal{R}_0$, denoted as the spatial radius $\mathcal{F}\mathcal{V}^{-1}$ is given as:

$$\mathcal{R}_0 = \left(\frac{\lambda_1 S^0}{(d+\gamma+\mu_1)} + \frac{\beta_1 \beta_2 S^0}{(d+\gamma+\mu_1)}\right),$$

$$= \left(\frac{\lambda_1 b_1}{\mu_1 (d+\gamma+\mu_1)} + \frac{\beta_1 \beta_2 b_1 b_2}{\mu_1 \mu_2^2 (d+\gamma+\mu_1)}\right).$$

Theorem-1 The disease-free equilibrium point E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$ and $(\mu_1(d + \mu_1 + \gamma) + \mu_1\mu_2 > \lambda_1 b_1)$ otherwise unstable.

Proof. The variational matrix at the disease free equilibrium point E_0 of the model (2) is given as:

$$J(x) = \begin{pmatrix} -\mu_1 & -\lambda_1 \frac{b_1}{\mu_1} & -\beta_1 \frac{b_1}{\mu_1} \\ 0 & \lambda_1 \frac{b_1}{\mu_1} - (d + \mu_1 + \gamma) & \beta_1 \frac{b_1}{\mu_1} \\ 0 & \beta_2 \frac{b_2}{\mu_2} & -\mu_2 \end{pmatrix}.$$

From the characteristic equation, we obtain one root as $-\mu_1$ and the remaining eigenvalue can be obtained from the following quadratic equation.

$$a_2\lambda^2 + a_1\lambda + a_0 = 0,$$

where

$a_2 = \mu_1$, $a_1 = (\mu_1(d + \mu_1 + \gamma) + \mu_1\mu_2 - \lambda_1 b_1)$, and $a_0 = \mu_1\mu_2(d + \mu_1 + \gamma)(1 - \mathcal{R}_0)$.

Using Routh-Hurwitz criterion, a second-degree polynomial with all the coefficients positive will obviously

have negative roots.

Now, $a_1 > 0$ if $(\mu_1(d+\mu_1+\gamma)+\mu_1\mu_2 > \lambda_1 b_1)$ and $a_0 > 0 \Rightarrow \mathcal{R}_0 < 1$.

Hence the theorem.

4 Existence of Endemic Equilibrium

Let the left-hand side of each differential equations of the system (2) be zero, the endemic (S, I, M, V) satisfies $(S > 0, I > 0, V > 0)$ and from equation third, we have

$$V^* = \frac{\beta_2 b_2 I^*}{\mu_2(\beta_2 I^* + \mu_2)}.$$

Using the value of V^* in equation first, we get

$$S^* = \frac{\mu_2(m+I^*)(\beta_2 I^* + \mu_2)(d+\mu_1+\gamma)}{\mu_2(\lambda_1 m + (\lambda_1-\lambda_2)I^*)(\beta_2 I^* + \mu_2) + (\beta_1\beta_2 b_2 m + \beta_1\beta_2 b_2 I^*)}.$$

Using the expression of V^* and S^* in equation second and the value of I^* can be obtained from the following equation

$$a_3 I^{*3} + a_2 I^{*2} + a_1 I^* + a_0 = 0. \qquad (3)$$

where

$$a_3 = \mu_2\beta_2(d+\mu_1+\gamma)(\lambda_1-\lambda_2),$$

$$a_2 = (d+\mu_1+\gamma)(m\lambda_1\mu_2\beta_2 + \mu_2^2(\lambda_1-\lambda_2) + \beta_1\beta_2 b_2 + \mu_1\mu_2\beta_2) - b_1\beta_2(\lambda_1-\lambda_2),$$

$$a_1 = (d+\mu_1+\gamma)(m\mu_2^2\lambda_1 + m\beta_1\beta_2 b_2 + m\mu_1\mu_2\beta_2 + \mu_1\mu_2^2(m\beta_2+\mu_2)) - b_1(\mu_2(\lambda_1-\lambda_2) + m\beta_2\lambda_1 + \beta_1\beta_2 b_2),$$

$$a_0 = m\mu_1\mu_2^2(d+\mu_1+\gamma)(1-\mathcal{R}_0).$$

Now, we state the theorem given in (Burnside and Panton, 1892) to determine the existence of endemic equilibrium.

Theorem-2 Every equation of an odd degree has at least one real root of a sign opposite to that of its last term.

Since it is assumed that $\lambda_1 > \lambda_2$. Therefore, $a_3 > 0$. Now, two cases arise:

Case i) when $\mathcal{R}_0 > 1$, then using the theorem 2, equation (3) has atleast one positive root.

Again two cases arise:

Case ii) when $\mathcal{R}_0 > 1$, and if $a_1 < 0; a_2 < 0$ then using Descarts rule of signs, equation (3) has exactly one Positive root.

Case iii)when $\mathcal{R}_0 > 1$, and if $a_1 > 0; a_2 > 0$ then using Descarts rule of signs, equation (3) has atleast one Positive root.

Case iv) when $\mathcal{R}_0 \leq 1$ then using Discart's rule of signs equation (3) has no positive root.

Thus, we summarize the result as:

Lemma 1 The system (2) has a unique endemic equilibrium point whenever $\mathcal{R}_0 > 1$ and no positive endemic equilibrium when $\mathcal{R}_0$ 1.

4.1 Local stability of endemic equilibrium

Theorem-3 If $\mathcal{R}_0 > 1$, then the endemic equilibrium $\mathrm{E}^* = (S^*, I^*, V^*)$ of the system (2) is locally asymptotically stable in Ω.

Proof. We linearize the system (2) at the equilibrium $E^* = (S^*, I^*, V^*)$ as:

$$\mathcal{J}(x)=\begin{pmatrix} -\left(\lambda_1-\frac{\lambda_2 I^*}{m+I^*}\right)I^*-\beta_1 V^*-\mu_1 & -\left(\lambda_1 S^*-\lambda_2 S^*\frac{mI^*}{(m+I^*)^2}\right) & -\beta_1 S^* \\ \left(\lambda_1-\frac{\lambda_2 I^*}{m+I^*}\right)I^*+\beta_1 V^* & \left(\lambda_1 S^*-\lambda_2 S^*\frac{mI^*}{(m+I^*)^2}\right)-(d+\mu_1+\gamma) & \beta_1 S^* \\ 0 & -\beta_2\left(\frac{b_2}{\mu_2}-V^*\right) & -\beta_2 I^*-\mu_2 \end{pmatrix}.$$

let

$$A=\left(\lambda_1-\frac{\lambda_2 I^*}{m+I^*}\right)I^*+\beta_1 V^*>0,$$

$$B=-\left(\lambda_1 S^*-\lambda_2 S^*\frac{mI^*}{(m+I^*)^2}\right)<0,$$

$$C=-(d+\mu_1+\gamma)+\left(\lambda_1 S^*-\lambda_2 S^*\frac{mI^*}{(m+I^*)^2}\right)<0.$$

The roots can be obtained from the following characteristic equation

$$X^3+c_3X^2+c_2X+c_1=0,$$

where

$$c_3=(A+\beta_2 I^*+\mu_1+\mu_2-C)>0,$$

$$c_2=\left((A+\mu_1)(\beta_2 I^*+\mu_2-C)-\beta_1\beta_2 S^*\left(\frac{b_2}{\mu_2}-V^*\right)-C(\beta_2 I^*+\mu_2)-AB\right)>0,$$

$$c_1=\left(-C(A+\mu_1)(\beta_2 I^*+\mu_2)+\mu_1\beta_1\beta_2 S^*\left(\frac{b_2}{\mu_2}-V^*\right)-AB(\beta_2 I^*+\mu_2)\right)>0.$$

Since,

$c_1c_2-c_3=(\beta_2 I^*+\mu_2-C)\left((A+\mu_1)(\beta_2 I^*+\mu_2-C)-C(\beta_2 I^*+\mu_2)+\beta_1\beta_2 S^*\left(\frac{b_2}{\mu_2}-V^*\right)\right)+$

$A\left(BC+\beta_1\beta_2 S^*\left(\frac{b_2}{\mu_2}-V^*\right)\right)+\left((A+\mu_1)(\beta_2 I^*+\mu_1-C)-AB\right)>0$. Threfore, by using Routh Hurwitz criteria, it is clear that all the eigenvalues of the system (2) have negative real parts if and only $\mathcal{R}_0>1$. Thus, endemic equilibrium E^* is locally asymptotically stable in Ω. This proves the theorem.

4.2 Global stability of endemic equilibrium

We now use the theory outlined in the Appendix A , in particular Theorem $(A.1)$, to prove that endemic equilibrium E^* is globally asymptotically stable in Ω as stated in Theorem $(A.1)$.

Theorem-4 The endemic state E^* is locally and globally asymptotically stable for $\mathcal{R}_0>1$.

Proof. Let us take $x=(S,I,V)$ and $f(x)$ denote the vector field of the system (2). The Jacobian matrix $\mathcal{J}(x)=\frac{\partial f}{\partial x}$ about x of the system (2) is given as:

$$\mathcal{J}(x)=\begin{pmatrix} -\left(\lambda_1-\frac{\lambda_2 I}{m+I}\right)I-\beta_1 V-\mu_1 & -\lambda_1 S+\frac{\lambda_2 S(mI)}{(m+I)^2} & -\beta_1 S \\ \left(\lambda_1-\frac{\lambda_2 I}{m+I}\right)I-\beta_1 V & \lambda_1 S-\frac{\lambda_2 S(mI)}{(m+I)^2}-(\mu_1+d+\gamma) & \beta_1 S \\ 0 & \lambda_2\left(\frac{\beta_2}{\mu_2}-V\right) & -\lambda I-\mu_2 \end{pmatrix},$$

and its second additive compound matrix $\mathcal{J}^{[2]}$ (see Appendix-B) is obtained as

$$\mathcal{J}^{[2]} = \begin{pmatrix} A_{11} & \beta_1 S & \beta_1 S \\ \lambda_2\left(\frac{b_2}{\mu_2} - V\right) & A_{22} & -\lambda_1 S + \lambda_2 S\frac{mI}{(m+I)^2} \\ 0 & \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) I + \beta_1 V & A_{33} \end{pmatrix},$$

where

$$A_{11} = -\left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) I - \beta_1 V - \mu_1 + \lambda_1 S - \frac{\lambda_2 S(mI)}{(m+I)^2} - (\mu_1 + d + \gamma),$$

$$A_{22} = -\left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) I - \beta_1 V - \mu_1 - \lambda_2 I - \mu_2,$$

$$A_{33} = \lambda_1 S - \frac{\lambda_2 S(mI)}{(m+I)^2} - \lambda_2 I - \mu_2 - (\mu_1 + d + \gamma).$$

Let us take the function $P(X) = P(S, I, V) = diag\{1, \frac{I}{V}, \frac{I}{V}\}$,

$$PfP^{-1} = diag\{0, \frac{\dot{I}}{I} - \frac{\dot{V}}{V}, \frac{\dot{I}}{I} - \frac{\dot{V}}{V}\}.$$

The matrix $B = PfP^{-1} + PJ^{[2]}P^{-1}$ can be written in block form as

$$B = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix},$$

where

$$B_{11} = -\left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) I - \beta_1 V + \mu_1 - \lambda_1 S - \frac{\lambda_2 S(mI)}{(m+I)^2} - (\mu_1 + d + \gamma),$$

$$B_{12} = \begin{pmatrix} \frac{\beta_1 SV}{I} & \frac{\beta_1 SV}{I} \end{pmatrix}, B_{21} = \begin{pmatrix} \frac{\lambda_2 I}{V}\left(\frac{b_2}{\mu_2} - V\right) & 0 \end{pmatrix}^T,$$

$$B_{22} = \begin{pmatrix} -\left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) I - \beta_1 V - \mu_1 - \lambda_2 I - \mu_2 + \frac{\dot{I}}{I} - \frac{\dot{V}}{V} & \lambda_1 S + \frac{\lambda_2 S(mI)}{(m+I)^2} \\ \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) I - \beta_1 V & \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) I - \beta_1 V - (\mu_1 + d + \gamma) + \frac{\dot{I}}{I} - \frac{\dot{V}}{V} \end{pmatrix}.$$

Taking the vector norm $|(\zeta, \vartheta, \omega)| = \max\{|\zeta|, |\vartheta| + |\omega|\}$ for all $(\zeta, \vartheta, \omega) \in \mathbb{R}^3$(Li and Muldowney, 1996), we have

$$\zeta(B) \leq \sup\{p_1, p_2\}, \quad (4)$$

where

$$p_1 = \zeta_1(B_{11}) + |B_{12}|,$$
$$p_2 = \zeta_1(B_{22}) + |B_{21}|.$$

ζ_1 denotes the Lozinskii measure with respect to the l_1 norm. Therefore,

$$p_2 = -\left(\lambda_1 - \frac{\lambda_2 I}{(m+I)}\right) I - \beta_1 V - \mu_1 + \lambda_1 S - \lambda_2 S\left(\frac{mI}{(m+I)^2}\right) - (\mu_1 + d + \gamma) + \frac{\beta_1 SV}{I} + \frac{\dot{I}}{I} - \frac{\dot{V}}{V}, \quad (5)$$

$$p_2 = \max\left\{-\mu_1 - \lambda_2 I - \mu_2 + \tfrac{\dot{I}}{I} - \tfrac{\dot{V}}{V}, -\lambda_2 I - \mu_2 + \tfrac{\dot{I}}{I} - \tfrac{\dot{V}}{V}\right\} + \tfrac{\lambda_2 I}{V}\left(\tfrac{b_2}{\mu_2} - V\right). \quad (6)$$

Rewriting the second and the third equations of the model (2), we obtain, respectively,

$$\frac{\dot{I}}{I} + (\mu_1 + d + \gamma) = \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right) S + \frac{\beta_1 SV}{I}, \quad (7)$$

$$\frac{\dot{V}}{V} = \frac{\lambda_2 I}{V}\left(\frac{b_2}{\mu_2}\right) I - \mu_2. \quad (8)$$

Substituting (7) into (5) and (8) into (6), we have

$$p_1 = \frac{\dot{I}}{I} - (\lambda_1 I + \lambda_1 S + \mu_1 + \beta_1 V), \quad (9)$$

$$p_2 = \frac{\dot{I}}{I} - (\mu_1 + \lambda_2 I). \quad (10)$$

which implies

$$\zeta(B) = \sup\{p_1, p_2\} \leq \frac{\dot{I}}{I} - \mu_1 + \max\ \{\lambda_1 I + \lambda_1 S + \beta_1 V, \lambda_2 I\},$$

i.e. $\zeta(B) = \sup\{p_1, p_2\} \leq \frac{\dot{I}}{I} - \mu_1 + c,$

where $c = \max\{\lambda_1 I + \lambda_1 S + \beta_1 V, \lambda_2 I\}$.

This holds along each solution $\big(S(t), I(t), V(t)\big)$ of the system with $\big(S(0), I(0), V(0)\big) \in K$, where K is the compact absorbing set. We have

$$\frac{1}{t}\int_0^t \zeta(B)\, dI \leq \frac{1}{t}\log\frac{I(t)}{I(0)} - (\mu + c),$$

$d = (\mu_1 + c)$, which implies that

$$\bar{q} = \lim_{t\to\infty} \sup\sup \frac{1}{t}\int_0^t \zeta(B)\, dI \leq -\frac{d}{2} < 0.$$

Thus, by result of Li and Muldowney (1996), it implies that E^* is globally as asymptotically stable.

5 Numerical Simulation

The primary aim of the present study is to investigate the impact of media coverage the spread of vector borne disease. Therefore, we perform a comprehensive numerical study to gauge the impact of media coverage related parameters on the disease dynamics. we integrate the systems by fourth order Runge-Kutta method using MATLAB 2018a. To study the dynamical behaviour of the system (2), numerical simulation of the system is done by using the following parameters.

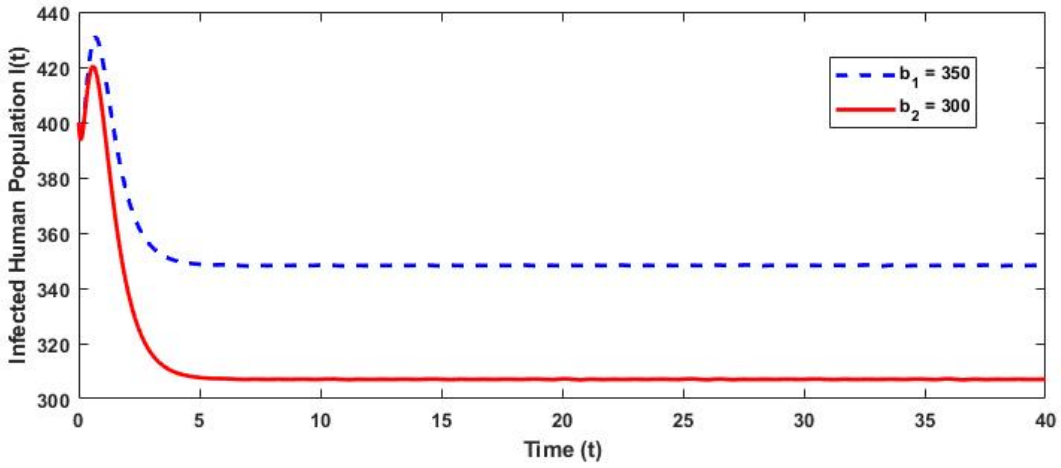

Fig. 1 Variation of infective human (host) population for parameter b_1 with respect to time *t*.

Fig. 1 show the variation of infective human population with time for the parameter λ_2 , the media coverage parameter. It is seen that when $\lambda_2 = 0$, the infective human population significantly increases as compared to when $\lambda_2 \neq 0$. Therefore, it suggests that in presence of effective media coverage there is delay in the appearance of the peak of infection, and there by decrease in the number of infected individuals. It shows that effectual media coverage is beneficial for the control of vector born disease. As we are aware of the fact that as soon as more and more infected people are reported by the media, the susceptible will take preventive methods by themselves from being infected. Fig. 2 show the variation of infective human population with time for the parameter b_1. It is clear from the Fig. 2 that as the immigration rate of host population increases, the susceptible population increases thereby increasing the infective population. Fig. 3 show the variation of infective human population with time for the parameter λ_1. It is clear from the figure that as the contact rate (without media coverage) λ_1 increases the infective population is also significantly increased.

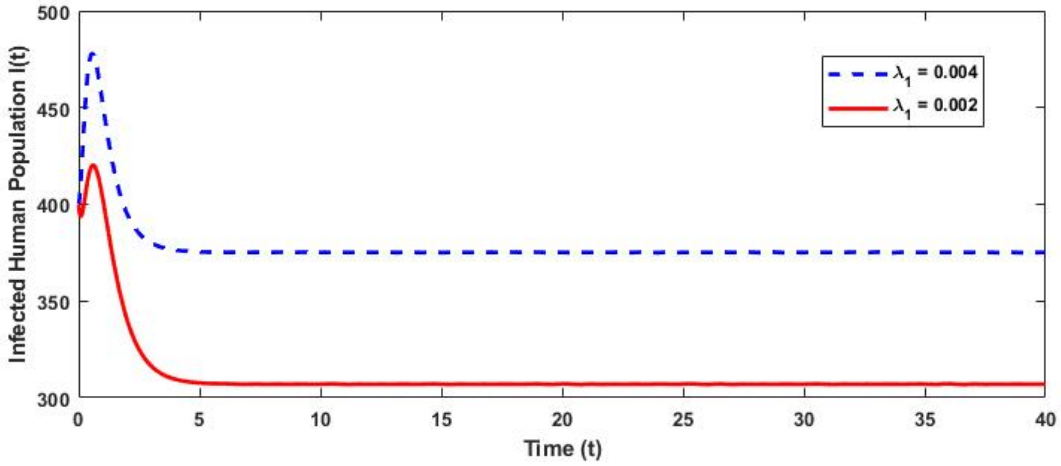

Fig. 2 Variation of infective human (host) population for parameter λ_1 with respect to time *t*.

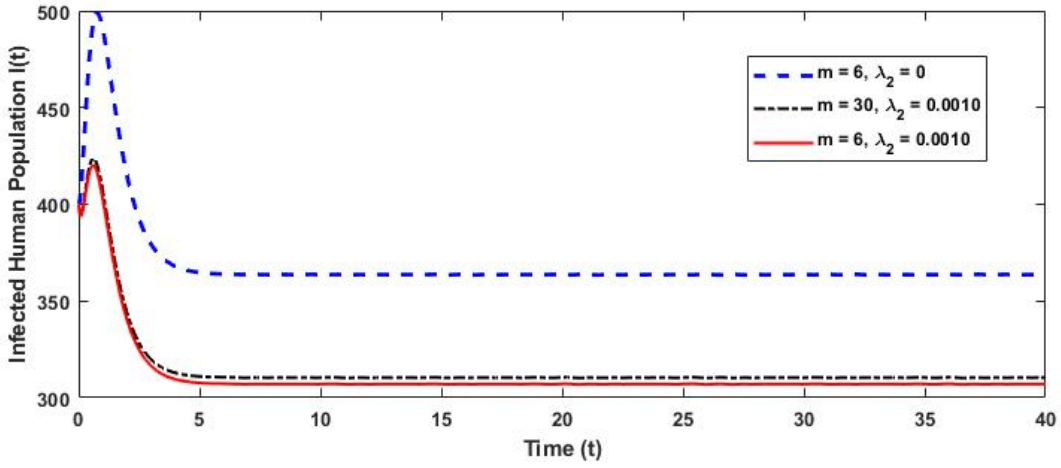

Fig. 3 Variation of infective human (host) population for media coverage parameter λ_2 with respect to time *t*.

6 Conclusion

In this paper, we developed and analyzed a mathematical model to study the impact of vector borne diseases. We have studied the stability of the disease free equilibrium and the endemic equilibrium for the system (2). The expression for the Reproduction number $\mathcal{R}_0$, suggest that the media coverage doesn't have any effect on the reproduction number. It can be easily analyzed that m and λ_2 doesn't change the stability condition for endemic equilibrium, instead, only coordinates of endemic equilibrium are changed. The expression of the media coverage function $\lambda(I) = \left(\lambda_1 - \frac{\lambda_2 I}{m+I}\right)$ clearly indicates that as the value of m decreases, the spread of of disease will decrease. It suggests that as soon as the infection starts to spread, the media should report the current situation and suggest various methods for the protection from infection.

Appendix A

In this Appendix we briefly outline geometric approach method of Li and Muldowney (1996) to study the global stability of endemic equilibrium point x^*. The method is briefly discussed as:

Consider

$\dot{x} = f(x)$,(A.1)

where $f: D \to \mathbb{R}^n; D \subset \mathbb{R}^n$ is an open set and simply connected and $f \in C^1(D)$.

Let $x(t, x_0)$ be the solution of equation (A. 1).

We assume that

(H_1) : a compact absorbing set exists i.e. $K \subset D$,

(H_2) : there exists a unique equilibrium for (A. 1) denoted by x^* in D,

are satisfied. If all the trajectories in D converge to x^* and are locally stable, then the point x^* is known to be globally stable in D. The global stability principal of Li and Muldowney (1996) for an autonomous system is presented as follows.

Lemma A.1.1 Let the two conditions (H_1) and (H_2) hold, assuming that $(A. 1)$ satisfies the Bendixson criteria (robustness under C^1) for local perturbations of $f(x)$ at all non-equilibrium, non-wandring points for (A. 1.1) . Then x^* is globally stable in D, provided it is stable.

The robustness which is required by lemma A.1.1 is proved by the following Bendixson criterion given in Li and Muldowney (1993). Let $P(x)$ represents $\binom{m}{2} \times \binom{m}{2}$ a matrix valued function i.e. C^1 on D. Also P^{-1} exists and is continuous for $x \in K$, the compact absorbing set.

We define the quantity $\bar{q}$ as ,

$$\bar{q} = \lim_{t\to\infty} \sup\sup \frac{1}{t}\int_0^t \Im\left(B\left(x(s, x_0)\right)\right) ds,$$

where

$B = P_f P^{-1} + P J^{[2]} P^{-1}$.

Here the matrix P_f is

$$\left(P_{ij}(x)\right)_f = \left(\frac{\partial P_{ij}(x)}{\partial x}\right)^T f(x) = \nabla P_{ij}(x).$$

The matrix $J^{[2]}$ represents the second additive compound matrix of the Jacobean matrix

$J(x) = Df(x)$.

Let $\Im(B)$ represents the Lozinskii measure of B with respect to the vector norm . in $\mathbb{R}^M$,

where $M = \binom{m}{2}$ is defined by

$$\Im(B) = \lim_{h\to 0^+} \frac{1+hB-1}{h}.$$

It has been proved in Lahrouz et al. (2011) that D is simply connected, then the condition $\bar{q} < 0$ deletes the presence of any orbit that gives rise to a simply closed rectifiable curve which is invariant for $(A.1)$. The global stability result which is proved in Li and Muldowney (1996) is stated as follow:

Lemma A.1.2 Let the simple connectivity of D together with (H_1) and (H_2) hold. Then E^*, the endemic equilibrium point of $(A.1)$ is stable globally in D if $\bar{q} < 0$. We find the global stability of endemic equilibrium using the approach of Li and Muldowney (1996). The uniform persistence exists in case E_0 is unstable (Freedman et al., 1994), i.e., there exists a constant $a > 0$ such that any solution $\big(S(t), I(t), V(t)\big)$ with $\big(S(0), I(0), V(0)\big)$ in the orbit of the system (2) satisfies

$$\liminf_{t\to\infty} S(t) > a, \quad \liminf_{t\to\infty} I(t) > a, \quad \liminf_{t\to\infty} V(t) > a,$$

where $a > 0$.

Theorem-A.1 Assume that D is simply connected and that assumptions $(H_1), (H_2)$ hold. Then the unique equilibrium x^* of (A.1) is globally stable in D if $\bar{q}_2 < 0$.

Appendix B

The second additive compound matrix of A is a $\binom{m}{2} \times \binom{m}{2}$ matrix and satisfies the property $(A+B)^{[2]} = A^{[2]} + B^{[2]}$. In particular, when $n = 2$, we have $A^{[2]}_{2\times 2} = trA^{[2]}$. When n = 3, the second additive compound matrix of $A = (a_{ij})$ is

$$A^{[2]} = \begin{pmatrix} a_{11}+a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11}+a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22}+a_{33} \end{pmatrix}.$$

A comprehensive survey on compound matrices and their relations to differential equations is given in Muldowney (1990).

References

Adedotun AA, Morenikeji OA, Odaibo AB, et al. 2010. Knowledge, attitudes and practices about malaria in an urban community in south-western Nigeria. Journal of Vector Borne Diseases, 47(3): 155-159

Burnside WS, Panton AW. 1892. The Theory Of Equations: With An Introduction To The Theory of Binary Algebraic Forms. Hodges, Figgis, Ireland

Chunara R, Jason RA, Brownstein JS. 2012. Social and news media enable estimation of epidemiological patterns early in the 2010 Haitian cholera outbreak. American Journal of Tropical Medicine and Hygiene, 86(1): 39

Cui J, Sun Y, Zhu H. 2008.The impact of media on the control of infectious diseases. Journal of Dynamics and Differential Equations, 20(1): 31-53

Driessche PV, Watmough J. 2002. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Mathematical Biosciences, 180(1-2): 29-48

Freedman HI, Ruan S, Tang M. 1994. Uniform persistence and flows near a closed positively invariant set. Journal of Dynamics and Differential Equations, 6(4): 583-600

Hossain M, Mani KC, Sidik SM, Shahar HK, Islam R. 2014. Knowledge and awareness about STDs among

women in bangladesh. BMC Public Health, 14(1): 1-7

Kar TK, Nandi SK, Jana S, Mandal M. 2019. Stability and bifurcation analysis of an epidemic model with the effect of media. Chaos, Solitons and Fractals, 120: 188-199

Kiss IZ, Cassell J, Recker M, Simon P L. 2010. The impact of information transmission on epidemic outbreaks. Mathematical Biosciences, 225(1): 1-10

Lahrouz A, Omari L, Kiouach D. 2011. Global analysis of a deterministic and stochastic nonlinear SIRS epidemic model. Nonlinear Analysis: Modelling and Control, 16(1): 59-76

Li MY, Muldowney J S. 1993. On Bendixsonâzs criterion. Journal of Differential Equations, 106: 27-39

Li MY, Muldowney JS. 1996. A geometric approach to global-stability problems. SIAM Journal on Mathematical Analysis, 27(4): 1070-1083

Liu Y, Cui JA. 2008. The impact of media coverage on the dynamics of infectious disease. International Journal of Biomathematics, 1(1): 65-74

Lu X, Wang S, Liu S, Li J. 2017. An SEI infection model incorporating media impact. Mathematical Biosciences and Engineering, 14(5): 1317

Misra AK, Rai RK, Takeuchi Y. 2018. Modeling the control of infectious diseases: Effects of TV and social media advertisements. Mathematical Biosciences and Engineering, 15(6): 1315

Misra AK, Sharma A, Li J. 2013. A mathematical model for control of vector borne diseases through media campaigns. Discrete and Continuous Dynamical Systems-B, 18(7): 1909

Misra AK, Sharma A, Shukla JB. 2011a. Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases. Mathematical and Computer Modelling, 53(5-6): 1221-1228

Misra AK, Sharma, Singh V. 2011b. Effect of awareness programs in controlling the prevalence of an epidemic with time delay. Journal of Biological Systems, 19(2): 389-402

Muldowney JS. 1990. Compound matrices and ordinary differential equations. Rocky Mountain Journal of Mathematics, 20(4)

Mummert A, Weiss H. 2013. Get the news out loudly and quickly: the influence of the media on limiting emerging infectious disease outbreaks. PloS one, 8(8): e71692

Naresh R, Verma SR, Shukla JB, Agarwal M. 2021. Analysis of a model for carrier dependent infectious diseases with sanitation as a control strategy. Computational Ecology and Software, 11(1): 1-20

Rahman MS, Rahman ML. 2007. Media and education play a tremendous role in mounting AIDS awareness among married couples in Bangladesh. AIDS Research and Therapy, 4(1): 1-7

Salman SM. 2021. Memory and media coverage effect on an HIV/AIDS epidemic model with treatment. Journal of Computational and Applied Mathematics, 385: 113203

Samanta S, Rana S, Sharma A, Misra AK, Chattopadhyay J. 2013. Effect of awareness programs by media on the epidemic outbreaks: A mathematical model. Applied Mathematics and Computation, 219(12): 6965-6977

Saranya S, Vijaya S, Rekha E. 2022. Susceptible-Infected-Treatment model with disease infection in prey population recovered by treatment. Computational Ecology and Software, 12(1): 12-22

Shams B, Khansari M. 2019. Average reachability: A new metric to estimate epidemic growth considering the network structure and epidemic severity. Network Biology, 9(3): 42-57

Srivastav AK, Goswami NK, Ghosh M, Li XZ. 2018. Modeling and optimal control analysis of Zika virus with media impact. International Journal of Dynamics and Control, 6(4): 673-1689

Tchuenche JM, Dube N, Bhunu CP, Smith RJ, Bauch CT. 2011. The impact of media coverage on the transmission dynamics of human influenza. BMC Public Health, 11(1): 1-14

Tyagi P, Roy A, Malhotra MS. 2005. Knowledge, awareness and practices towards malaria in communities of

rural, semi-rural and bordering areas of east Delhi (India). Journal of Vector Borne Diseases, 42(1): 30

Webb G, Blaser MJ, Zhu H, Ardal S, Wu J. 2004. Critical role of nosocomial transmission in the Toronto SARS outbreak. Mathematical Biosciences and Engineering, 1(1): 1

Wendi W, Ruan S. 2004. Simulating the SARS outbreak in Beijing with limited data. Journal of Theoretical Biology, 227(3): 369-379

Xiao Y, Tang S, Wu J. 2015. Media impact switching surface during an infectious disease outbreak. Scientific Reports, 5(1): 1-9

Zhang WJ, Chen ZL, Lu Y, et al. 2020. A generalized discrete dynamic model for human epidemics. Computational Ecology and Software, 10(3): 94-104