The Impact of Media on the Spreading and Control of Japanese Encephalitis

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Abstract— **In this paper, a nonlinear mathematical model for the effect of awareness programs on the spread of infectious disease such as Japanese Encephalitis (JE) has been proposed and analyzed. In the modeling process it is assumed that disease spreads due to contact between susceptible and infected mosquitoes only. The growth rate of awareness programs impacting the population is assumed to be proportional to the number of infective individuals. It is further assumed that due to the effect of media, susceptible individuals form a separate class and avoid contact with the infectives. The model is analyzes by using stability theory of differential equations and computer simulations. The model analysis shows that the spread of an infectious disease (JE) can be controlled by using awareness programs but the growth rates of reservoir population and vector mosquito population increase, the spread of JE increase and the disease become more endemic due to human immigration. Numerical simulations are also carried out to investigate the influence of certain parameters on the spread of disease, to support the analytical results.**

Index Terms— **Nonlinear model, Japanese encephalitis, reservoir population, vector population, awareness programs, stability.**

MSC 2010 Codes — **34D20, 34D23.**

I. Introduction

JAPENSE Encephalitis Virus (JEV) is an arbovirus causing encephalitis and shares a close genetic relationship with other encephalitis viruses, including St. Louis encephalitis virus (SLEV), west Nile virus (WNV), Murray Valley encephalitis (MVEV), Alfuy virus (ALFV) and kunjun virus (KUNV). The disease has been recognized in Japan since the nineteenth century and the virus was first isolated and characterized in 1935, Gould [1]. Japanese Encephalitis (JE) has since been identified throughout Asia, apparently appearing in India during the middle of the twentieth century and finally appearing on the islands of the northeast coast of Australia in the mid- 1990s.

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Japanese Encephalitis (JE) is a viral disease that infects animal and humans. It is transmitted by mosquitoes and in humans causes inflammation of the membranes around the brain. Intensification and expansion of irrigated rice production systems in South and South-East Asia over the past 20 years have had an important impact on the disease burden caused by Japanese Encephalitis (JE). Where irrigation expands into semi-arid areas, the flooding of the fields at the start of each cropping cycle leads to an explosive build-up of the mosquito population. This may cause the circulation of the virus to spill over from their usual hosts (birds and pigs) into the human population.

Japanese Encephalitis (JE) is a disease caused by a flavivirus that affects the membranes around the brain. Most Japanese Encephalitis (JE) virus infections are mild (fever and headache) or without apparent symptoms, but approximately 1 in 200 infections results in severe disease characterized by rapid onset of high fever, headache, neck stiffness, coma, seizures and spastic paralysis and death. The case fatality rate can be as high as 60% among those with disease symptoms, 30% of those who survive suffer from lasting damage to the central nervous system. In areas where the Japanese Encephalitis (JE) virus is common, encephalitis occur mainly in young children because older children and adults have already been infected and are immune.

Japanese Encephalitis Virus (JEV) is passed on by the bite of an infected mosquito that has previously sucked blood from an infected animal or person, Gould, Easmon [2]. The risk for acquiring JEV among most travelers to Asia extremely low; however the risk of transmission is higher in rural areas, especially where pigs are raised and where rice paddies, marshes and standing pools of water provide breeding grounds for mosquitoes and feed for birds, Gould.

Very few modeling studies so far have been done to understand the transmission dynamics of JEV to the best of our knowledge, Mukhopadhyay et al. [3], Tapaswi et al. [4]. Mukhopadhyay et al. formulated a regression equation model using a third order Harmonic Fourier series having a linear trend to simulate the pattern of monthly occurrence of Tapaswi et al. models the spread of JE in human population of varying size from reservoir population through a vector population by considering reservoir population constant size. Their study shows that if a certain threshold is exceeded, then there is a unique equilibrium with disease present which is locally stable to small perturbations and the global stability depends on

death rates and the ratio of the equilibrium population sizes of the infected vector and total human populations. Also, Naresh and Pandey [5] studied the spread of Japanese Encephalitis with environmental effects.

The classical models governing the spread of infectious diseases depend mainly on the interaction between susceptibles and infectives. However, there are other factors, such as media coverage, vaccination, migration of population etc., which also affects the spread of infectious diseases [6-10]. In particular, media has a great influence not only the individual's behavior towards the diseases but it also affects the governmental health care interventions to control the spread of such diseases. It is the awareness program by media which make people knowledgeable about the disease to take precautions such as social distancing, wearing protective masks, vaccination etc., to reduce their chances of being infected. Therefore, to predict the spread of infectious disease the effect of the media must be considered in the modeling process. Some compartmental models have been introduced with the assumption that the media will reduce the contact rate of susceptible with infectives [11-15].

The objective of our study is to investigate the transmission dynamics of JEV in three-population system consisting of humans, reservoir and vector populations by considering the effects of awareness programs conducted by media. It is assumed that the growth rate of cumulative density of awareness programs driven by media depends upon the number of infectives present in the population. Further, the awareness about the disease will alert the susceptible to isolate themselves from infectives and avoid being infected by forming a separate class. The effects of depletion of awareness programs with time have also been taken into account while modeling the system.

II. Mathematical Model

We propose a nonlinear mathematical model to study the spread of Japanese Encephalitis in a three population system consisting of human, reservoir and vector populations. The total human population $N(t)$ at time t, with constant inflow of susceptible at rate A, is divided into three subclasses; the susceptible population $S(t)$, infective population $I(t)$ and the aware population $S_m(t)$. Also, let $M_e(t)$ be the cumulative density of awareness programs driven by the media in that region at time t.

It is assumed that disease spreads due to the direct interaction between susceptible humans and infected vectors (mosquitoes) only. Since the mosquitoes population is subject to the rapid change, it is assumed that the mosquito population is growing logistically with given intrinsic growth rate and carrying capacity. The total vector population $M(t)$ is divided into susceptible mosquito population $M_s(t)$ and infected mosquitoes population $M_i(t)$.

There is no immune class in the mosquito population since it acts as a transmitter of virus only. The susceptible mosquitoes through the direct interaction with the reservoir population $P(t)$ and infected human population $I(t)$. We consider variable reservoir population which forms a 'pool of infection' with constant inflow of infected individuals only, through it was assumed to be constant by taking birth and death rates are equal, Tapaswi et al. The growth rate of density of awareness programs is assumed to be proportional to the number of infective individuals. It is considered that due to the awareness programs susceptible individuals form a different class and avoid contact with the infectives.

Keeping in view of the above discussion and considering the criss-cross interaction of reservoir and mosquito population, mosquito and human population, the dynamics of the transmission of JE is assumed to be governed by the following system of nonlinear ordinary differential equation:

$$\begin{aligned}
\frac{dS}{dt} &= A - \sigma M_i S - dS - \lambda_1 S M_e + \lambda_0 S_m + \nu I,\\
\frac{dI}{dt} &= \sigma M_i S - (\nu + \alpha + d) I,\\
\frac{dS_m}{dt} &= \lambda_1 S M_e - dS_m - \lambda_0 S_m,\\
\frac{dN}{dt} &= A - dN - \alpha I,\\
\frac{dP}{dt} &= A_0 - d_1 P - \alpha_1 P,\\
\frac{dM_s}{dt} &= \gamma M\left(1 - \frac{M}{L}\right) - \lambda_2 M_s P - \lambda_3 M_s I - \gamma_0 M_s,\\
\frac{dM_i}{dt} &= \lambda_2 M_s P + \lambda_3 M_s I - \gamma_0 M_i,\\
\frac{dM}{dt} &= \gamma M\left(1 - \frac{M}{L}\right) - \gamma_0 M,\\
\frac{dM_e}{dt} &= \mu I - \mu_0 M_e,
\end{aligned} \tag{2.1}$$

where

$S(0) > 0,\ I(0) \geq 0,\ S_m \geq 0,\ N(0) > 0,\ P(0) > 0,\ M_s \geq 0,$

In the above model (2.1), A is the constant immigration rate of human population, σ is the transmission coefficient due to mosquito population; d and α are the natural and disease induced death rates of human population, respectively and ν is rate by which infected individuals are recovered and become susceptible again. A_0 is the constant immigration rate of infected reservoir population; and α_1 is the death rate of reservoir population due to disease and control measures; λ_1 represent the dissemination rate of awareness among susceptibles due to which they form a different class. The

constant λ_0 denotes the rate of transfer of aware individuals to susceptible class.

The constant L is the carrying capacity of mosquito population in the natural environment; γ is its growth rate, γ_0 is death rate of mosquito population due to natural causes as well as control measures; λ_2 and λ_3 are the transmission coefficients due to interaction of susceptible mosquito population with reservoir population and with infected human population respectively. The constant μ represent the rate with which awareness programs are being implemented and μ_0 represent the depletion rate of these programs due to infectiveness, social problems in the population, etc. In the model, all the dependent variables and parameters are assumed to be non-negative.

Using the fact that $N = S + I + S_m$ and $M_s + M_i = M$ the above system (2.1) reduced to the following system:
$M \geq 0,\ M_e \geq 0.$

$$\begin{aligned}
\frac{dI}{dt} &= \sigma M_i \left(N - I - S_m\right) - \left(\nu + \alpha + d\right) I, \\
\frac{dS_m}{dt} &= \lambda_1 M_e \left(N - I - S_m\right) - dS_m - \lambda_0 S_m, \\
\frac{dN}{dt} &= A - dN - \alpha I, \\
\frac{dP}{dt} &= A_0 - d_1 P - \alpha_1 P, \\
\frac{dM_i}{dt} &= \lambda_2 (M - M_i) P + \lambda_3 (M - M_i) I - \gamma_0 M_i, \\
\frac{dM}{dt} &= \gamma M \left(1 - \frac{M}{L}\right) - \gamma_0 M, \\
\frac{dM_e}{dt} &= \mu I - \mu_0 M_e.
\end{aligned} \tag{2.2}$$

Now, it is sufficient to study the model system (2.2) in detail rather than the model system (2.1).

III. Boundedness of Solutions

Continuity of right hand side of system (2.2) and its derivative imply that the model is well posed for $N > 0.$

The invariant region where solution exists is obtained as follows

$$\frac{A}{(d+\varepsilon)} \leq \liminf N(t) \leq \limsup N(t) \leq \frac{A}{d} \ (\text{as } t \to \infty),$$

since $N(t) > 0$ for all $t \geq 0.$ Therefore, $N(t)$ cannot blow up to infinity in finite time and consequently, the model system is dissipative (solutions are bounded). Hence, the solution exists for all $t > 0$ in the invariant and compact set,

$$\Omega = \left\{ \begin{array}{l} (I, S_m, N, P, M_i, M, M_e) \in R_+^7 : 0 \leq I \leq S_m \leq \frac{A}{d} + \varepsilon,\ 0 \leq P \leq P_m, 0 \leq M_i \leq M \leq M_m, \\ 0 \leq M_e \leq M_R \end{array} \right\},$$

which is a region of attraction for any arbitrary small constant $\varepsilon > 0$ and $\gamma > \gamma_0.$

Here, $P_m = \frac{A_0}{d_1 + \alpha_1}$, $M_m = \frac{L}{\gamma}\left(\gamma - \gamma_0\right)$, and $M_R = \frac{\mu A}{\mu_0 d}.$

As $N(t)$ tends to zero, $S(t)$, $I(t)$ and $S_m(t)$ also tends to zero. Hence, each of these subpopulations tends to zero as $N(t)$ does. It is therefore natural to interpret these terms as zero at $N(t) = 0.$

IV. Equilibrium Analysis

In this section we investigate the existence of equilibria of system (2.2). Solving the right hand side of the model system (2.2) by equating it to zero, we obtain the following biologically relevant equilibria.

(1)Disease-free equilibrium (DFE) $E_1\left(0, 0, \frac{A}{d}, \tilde{P}, 0, 0, 0\right)$,

exists without any condition, where: $\tilde{P} = \frac{A_0}{d_1 + \alpha_1}.$

The existence of E_1 is obvious. This equilibrium implies that if the carrier population, which serves as a medium of transport of JEV, does not participate in the system then the equilibrium level of human population will reach the value $\frac{A}{d}$ and the reservoir population will remain at its equilibrium $\tilde{P}_m$. It may also be noted that in the absence of mosquito population, the infected human population will become zero.

(2) Endemic equilibrium $E_2\left(I^*, S_m^*, N^*, P^*, M_i^*, M^*, M_e^*\right)$

This equilibrium implies that if the mosquito population is present in the system, then the infection will be transmitted to the human population. The equilibrium values of different variables will be given by $I^*, S_m^*, N^*, P^*, M_i^*, M^*$ and M_e^*.

These equilibrium values are explicitly given by equations (4.2-4.7). We prove the existence of endemic equilibrium E_2 by setting right hand side of equations (2.2) to zero and solving the resulting algebraic equations, we get,

$$\sigma M_i (N - I - S_m) - (\nu + \alpha + d) I = 0, \tag{4.1}$$

$$I^* = \frac{\left(A - dN^*\right)}{\alpha}, \tag{4.2}$$

$$P^* = \frac{A_0}{(d_1 + \alpha_1)}, \tag{4.3}$$

$$S_m^* = \frac{\lambda_1 (N^* - I^*) M_e^*}{(d + \lambda_0 + \lambda_1 M_e^*)}, \tag{4.4}$$

$$M^* = \frac{L}{\gamma}\left(\gamma - \gamma_0\right) \tag{4.5}$$

$$M_i^* = \frac{(\lambda_2 P^* + \lambda_3 I^*)M^*}{(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)}, \tag{4.6}$$

$$M_e^* = \frac{\mu I^*}{\mu_0}. \tag{4.7}$$

In the equilibrium E_2, N^* is the positive root of the following equation, which can be obtained from equation (4.1) after using I^*, S_m^* and M_i^* from equations (4.2), (4.4) and (4.6) respectively.

Using this value of $N = N^* > 0$ in equations (4.2-4.7) we obtain other equilibrium values,

$$\begin{aligned} F(N) = \sigma\left(\lambda_2 P + \lambda_3 \frac{(A - dN)}{\alpha}\right) M\{N(d + \lambda_0)\} \\ - \gamma_0(\nu + \alpha + d)\left(\frac{A - dN}{\alpha}\right)(d + \lambda_0 + \lambda_1 M_e) \\ - \{(\nu + \alpha + d)(d + \lambda_0 + \lambda_1 M_e) + (d + \lambda_0) M\sigma\} \\ \left(\lambda_2 P + \lambda_3 \frac{(A - dN)}{\alpha}\right)\left(\frac{A - dN}{\alpha}\right). \end{aligned} \tag{4.8}$$

It would be sufficient if we show that $F(N) = 0$ has one and one only root. From equation (4.1), if we note that $F\left(\frac{A}{(d+\alpha)}\right) < 0$ and $F\left(\frac{A}{d}\right) > 0$. This implies that there exists a root N^* of $F(N) = 0$ in $\frac{A}{d+\alpha} < N < \frac{A}{d}$. Also, $F'(N) > 0$, provided $\left[\left(\lambda_2 P + \lambda_3 \frac{(A - dN)}{\alpha}\right)\right] > \frac{Nd}{\alpha}$ and $(d + \lambda_0) < \left(\frac{\lambda_1 \mu d}{\mu_0 \alpha}\right)$ in $\frac{A}{d+\alpha} < N < \frac{A}{d}$.

Thus there exists a unique positive root of $F(N) = 0$, (say N^*) in $\frac{A}{d+\alpha} < N < \frac{A}{d}$. Knowing the value of N^*, the value of I^*, S_m^*, M_i^* and M_e^* can be computed from equations (4.2, 4.4, 4.6 and 4.7). Also, the value of P^* and M^* can be found from equation (4.3) and (4.5) respectively.

V. Stability Analysis

Now, we analyze the stability of equilibria E_1 and E_2 and the stability results of these equilibria are stated in the following theorems.

Theorem 5.1

(i) The disease-free equilibrium (DFE), E_1 is locally asymptotically stable if $\gamma > \gamma_0$, otherwise, E_1 is unstable.

(ii) The endemic equilibrium E_2 is locally asymptotically stable provided the following inequality is satisfied:

$$\frac{15\sigma^2 M_i^{*2} \lambda_1^2}{4(\sigma M_i^* + \nu + \alpha + d)(\lambda_1 M_e^* + d + \lambda_0)^2} < \min\left\{\frac{4(\sigma M_i^* + \nu + \alpha + d)}{15 M_e^{*2}}, \frac{2\sigma M_i^* d}{3 M_e^{*2} \alpha}, \frac{8(\sigma M_i^* + \nu + \alpha + d)\mu_0^2}{75(N^* - I^* - S_m^*)^2 \mu^2}\right\}$$

$$\frac{15\sigma^2 (N^* - I^* - S_m^*)^2}{16(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)^2 (\sigma M_i^* + \nu + \alpha + d)} < \frac{1}{(M^* - M_i^*)^2} \min\left\{\frac{1}{3\lambda_2^2}, \frac{(\sigma M_i^* + \nu + \alpha + d)}{15\lambda_3^2}\right\}$$

$$k_3(\lambda_2 P^* + \lambda_3 I^*)^2 < \frac{4}{3}\left\{\frac{2\gamma}{L} M^* - (\gamma - \gamma_0)\right\}(\lambda_2 P^* + \lambda_3 I^* + \gamma_0).$$

Proof: (i) The variational matrix J_1 for the system (2.2) corresponding to equilibrium $E_1\left(0,0,\frac{A}{d},\tilde{P},0,0,0\right)$ is given by,

$$J_1 = \begin{bmatrix} -(\nu+\alpha+d) & 0 & 0 & 0 & \sigma A/d & 0 & 0 \\ 0 & -(d+\lambda_0) & 0 & 0 & 0 & 0 & \lambda_1 A/d \\ -\alpha & 0 & -d & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(d_1+\alpha_1) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -(\lambda_2\tilde{P}+\gamma_0) & \lambda_2\tilde{P} & 0 \\ 0 & 0 & 0 & 0 & 0 & -(\gamma-\gamma_0) & 0 \\ \mu & 0 & 0 & 0 & 0 & 0 & -\mu_0 \end{bmatrix}$$

The eigen values of J_0 are $\psi_1 = -(\nu+\alpha+d)$, $\psi_2 = -(d+\lambda_0)$, $\psi_3 = -d$, $\psi_4 = -(d_1+\alpha_1)$, $\psi_5 = -(\lambda_2\tilde{P}+\gamma_0)$, $\psi_6 = -(\gamma-\gamma_0)$, and $\psi_7 = -\mu_0$. Since all the model parameters are assumed to be non-negative, it follows that $\psi_i = (i = 1,2,3,4,5,6,7) < 0$. The stability of E_1 will depends on the sign of ψ_6. Thus the disease free equilibrium (DFE) is locally asymptotically stable if $\gamma > \gamma_0$, i.e., the growth rate of mosquito population is higher than their decay coefficient.

(ii) To establish the local stability of the endemic equilibrium E_2, we consider the following positive definite function,

$$V_1 = \frac{1}{2}\left(k_0 i^2 + k_1 s_m^2 + k_2 p^2 + k_3 m_i^2 + k_4 m^2 + k_5 n^2 + k_6 m_e^2\right)$$

where $k_i's\ (i = 0,1,2,3,4,5,6)$ are positive constant to be chosen appropriately and i, s_m, p, m_i, m, n and m_e are small perturbations about E_2, defined as follows, $I = I^* + i$, $S_m = S_m^* + s_m$, $P = P^* + p$, $M_i = M_i^* + m_i$, $M = M^* + m$, $N = N^* + n$ and $M_e = M_e^* + m_e$.

Differentiating above equation, with respect to $'t'$, and using the linearized system corresponding to E_2, we get

$$\frac{dV_1}{dt} = -k_0(\sigma M_i^* + \nu + \alpha + d)i^2 + k_0\sigma M_i^* in + k_0\sigma(N^* - I^* - S_m^*)im_i$$
$$- k_0\sigma M_i^* is_m - k_1(\lambda_1 M_e^* + d + \lambda_0)s_m^2 + k_1 M_e^*\lambda_1 s_m n - k_1 M_e^*\lambda_1 is_m$$
$$+ k_1\lambda_1(N^* - I^* - S_m^*)s_m m_e - k_2(d_1 + \alpha_1)p^2 - k_3(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)m_i^2$$
$$+ k_3(\lambda_2 P^* + \lambda_3 I^*)mm_i + k_3\lambda_3(M^* - M_i^*)im_i + k_3\lambda_2(M^* - M_i^*)pm_i$$
$$- k_4\left(\frac{2\gamma}{L}M^* - (\gamma - \gamma_0)\right)m^2 - k_5 dn^2 - k_5\alpha ni + k_6\mu im_e - k_6\mu_0 m_e^2.$$

Now, $\frac{dV_1}{dt}$ will be negative definite under the following conditions,

(i) $\left(k_0\sigma M_i^* - k_5\alpha\right)^2 < \frac{2}{5}k_0k_5d(\sigma M_i^* + \nu + \alpha + d)$,

(ii) $k_0\sigma^2\left(N^* - I^* - S_m^*\right)^2 < \frac{4}{15}k_3(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)(\sigma M_i^* + \nu + \alpha + d)$,

(iii) $k_0\sigma^2 M_i^{*2} < \frac{4}{15}(\sigma M_i^* + \nu + \alpha + d)k_1(\lambda_1 M_e^* + d + \lambda_0)$,

(iv) $k_1\lambda_1^2 M_e^{*2} < \frac{2}{3}(\lambda_1 M_e^* + d + \lambda_0)k_5 d$,

(v) $k_1\lambda_1^2 M_e^{*2} < \frac{4}{15}(\sigma M_i^* + \nu + \alpha + d)k_0(\lambda_1 M_e^* + d + \lambda_0)$,

(vi) $k_1\lambda_1^2(N^* - I^* - S_m^*)^2 < \frac{2}{3}(\lambda_1 M_e^* + d + \lambda_0)k_6\mu_0$,

(vii) $k_3\lambda_2^2(M^* - M_i^*)^2 < \frac{4}{3}(d_1 + \alpha_1)k_2(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)$,

(viii) $k_3\lambda_3^2(M^* - M_i^*)^2 < \frac{4}{15}(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)k_0(\sigma M_i^* + \nu + \alpha + d)$,

(ix) $k_3(\lambda_2 P^* + \lambda_3 I^*)^2 < \frac{4}{3}\left(\frac{2\gamma}{L}M^* - (\gamma - \gamma_0)\right)k_4(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)$,

(x) $k_6\mu^2 < \frac{2}{5}(\sigma M_i^* + \nu + \alpha + d)k_0\mu_0$.

After choosing $k_0 = 1$, $k_5 = \frac{\sigma M_i^*}{\alpha}$, $k_2 = \frac{1}{d_1 + \alpha_1}$, $k_4 = 1$ and $k_6 = \frac{4(\sigma M_i^* + \nu + \alpha + d)\mu_0}{25\mu^2}$ we can choose k_1 and k_3 such that,

$$\frac{15\sigma^2 M_i^{*2}\lambda_1^2}{4(\sigma M_i^* + \nu + \alpha + d)(\lambda_1 M_e^* + d + \lambda_0)^2} <$$
$$\min\left\{\frac{4(\sigma M_i^* + \nu + \alpha + d)}{15M_e^{*2}}, \frac{2\sigma M_i^* d}{3M_e^{*2}\alpha}, \frac{8(\sigma M_i^* + \nu + \alpha + d)\mu_0^2}{75(N^* - I^* - S_m^*)^2\mu^2}\right\}$$
$$\frac{15\sigma^2(N^* - I^* - S_m^*)^2}{16(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)^2(\sigma M_i^* + \nu + \alpha + d)}$$
$$< \frac{1}{(M^* - M_i^*)^2}\min\left\{\frac{1}{3\lambda_2^2}, \frac{(\sigma M_i^* + \nu + \alpha + d)}{15\lambda_3^2}\right\}$$
$$k_3(\lambda_2 P^* + \lambda_3 I^*)^2 < \frac{4}{3}\left\{\frac{2\gamma}{L}M^* - (\gamma - \gamma_0)\right\}(\lambda_2 P^* + \lambda_3 I^* + \gamma_0).$$

Theorem 5.2

The endemic equilibrium E_2 is non-linearly stable in the region Ω if the following conditions are satisfied:

$$\frac{15\sigma^2 M_i^{*2}\lambda_1^2\mu^2}{4(\sigma M_i^* + \nu + \alpha + d)(\lambda_1 M_e^* + d + \lambda_0)^2\mu_0^2} <$$
$$\min\left\{\frac{4(\sigma M_i^* + \nu + \alpha + d)d^2}{15A^2}, \frac{2\sigma M_i^* d^3}{3A^2\alpha}, \frac{8(\sigma M_i^* + \nu + \alpha + d)}{75(N^* - I^* - S_m^*)^2}\right\}$$
$$\frac{15\sigma^2(A^2/d^2)}{16(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)^2(\sigma M_i^* + \nu + \alpha + d)} <$$
$$\frac{1}{(M^* - M_i^*)^2}\min\left\{\frac{1}{3\lambda_2^2}, \frac{(\sigma M_i^* + \nu + \alpha + d)}{15\lambda_3^2}\right\}$$
$$k_3(\lambda_2 P_m + \lambda_3 A/d)^2 < \frac{4\gamma}{3L}(\lambda_2 P^* + \lambda_3 I^* + \gamma_0).$$

where, $P_m = \frac{A_0}{(d_1 + \alpha_1)}$.

Proof: Consider the following positive function,

$$V_2 = \frac{k_0}{2}(I - I^*)^2 + \frac{k_1}{2}(S_m - S_m^*)^2 + \frac{k_2}{2}(P - P^*)^2 + \frac{k_3}{2}(M_i - M_i^*)^2$$
$$+ k_4\left(M - M^* - M^*\log\frac{M}{M^*}\right) + \frac{k_5}{2}(N - N^*)^2 + \frac{k_6}{2}(M - M_e^*)^2.$$

where the coefficients $k_0, k_1, k_2, k_3, k_4, k_5$ and k_6 can be chosen appropriately. Differentiating the above equation with respect to 't', and using (2.2), we get

$$\frac{dV_2}{dt} = k_0\sigma(N - I - S_m)(M_i - M_i^*)(I - I^*) + k_0\sigma M_i^*(N - N^*)(I - I^*) - k_0(\sigma M_i^* + \nu + \alpha + d)(I - I^*)^2$$
$$- k_0\sigma M_i^*(S_m - S_m^*)(I - I^*) + k_1\lambda_1(N^* - I^* - S_m^*)(M_e - M_e^*)(S_m - S_m^*) - (\lambda M_e + d + \lambda_0)(S_m - S_m^*)^2$$
$$+ k_1\lambda_1 M_e(N - N^*)(S_m - S_m^*) - k_1\lambda_1 M_e(S_m - S_m^*)(I - I^*) - k_2(d_1 + \alpha_1)(P - P^*)^2$$
$$+ k_3(\lambda_2 P + \lambda_3 I)(M - M^*)(M_i - M_i^*) + k_3\lambda_2(M^* - M_i^*)(P - P^*)(M_i - M_i^*)$$
$$+ k_3\lambda_3(M^* - M_i^*)(I - I^*)(M_i - M_i^*) - k_3(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)(M_i - M_i^*)^2 - k_4\frac{\gamma}{L}(M - M^*)^2$$
$$- k_5 d(N - N^*)^2 - k_5\alpha(I - I^*)(N - N^*) + k_6\mu(I - I^*)(M_e - M_e^*) - k_6\mu_0(M_e - M_e^*)^2.$$

Now, $\frac{dV_2}{dt}$ will be negative definite under the following conditions,

(i) $\left(k_0\sigma M_i^* - k_5\alpha\right)^2 < \frac{2}{5}k_0k_5d(\sigma M_i^* + \nu + \alpha + d)$,

(ii) $k_0\sigma^2\left(N - I - S_m\right)^2 < \frac{4}{15}k_3(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)(\sigma M_i^* + \nu + \alpha + d)$,

(iii) $k_0\sigma^2 M_i^{*2} < \frac{4}{15}(\sigma M_i^* + \nu + \alpha + d)k_1(\lambda_1 M_e^* + d + \lambda_0)$,

(iv) $k_1\lambda_1^2 M_e^2 < \frac{2}{3}(\lambda_1 M_e^* + d + \lambda_0)k_5 d$,

(v) $k_1\lambda_1^2 M_e^2 < \frac{4}{15}(\sigma M_i^* + \nu + \alpha + d)k_0(\lambda_1 M_e^* + d + \lambda_0)$,

(vi) $k_1\lambda_1^2(N^* - I^* - S_m^*)^2 < \frac{2}{3}(\lambda_1 M_e^* + d + \lambda_0)k_6\mu_0$

(vii) $k_3\lambda_2^2(M^* - M_i^*)^2 < \frac{4}{3}(d_1 + \alpha_1)k_2(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)$,

(viii) $k_3\lambda_3^2(M^* - M_i^*)^2 < \frac{4}{15}(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)k_0(\sigma M_i^* + \nu + \alpha + d)$,

(ix) $k_3(\lambda_2 P + \lambda_3 I)^2 < \frac{4\gamma}{3L} k_4(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)$,

(x) $k_6 \mu^2 < \frac{2}{5}(\sigma M_i^* + \nu + \alpha + d) k_0 \mu_0$.

Now, choosing $k_0 = 1$, $k_5 = \frac{\sigma M_i^*}{\alpha}$, $k_2 = \frac{1}{d_1 + \alpha_1}$,

$k_4 = 1$ and $k_6 = \frac{4(\sigma M_i^* + \nu + \alpha + d)\mu_0}{25\mu^2}$ such that:

$$\frac{15\sigma^2 M_i^{*2} \lambda_1^2 \mu^2}{4(\sigma M_i^* + \nu + \alpha + d)(\lambda_1 M_e^* + d + \lambda_0)^2 \mu_0^2} <$$

$$\min\left\{\frac{4(\sigma M_i^* + \nu + \alpha + d)d^2}{15A^2}, \frac{2\sigma M_i^* d^3}{3A^2\alpha}, \frac{8(\sigma M_i^* + \nu + \alpha + d)}{75(N^* - I^* - S_m^*)^2}\right\}$$

$$\frac{15\sigma^2 (A^2/d^2)}{16(\lambda_2 P^* + \lambda_3 I^* + \gamma_0)^2 (\sigma M_i^* + \nu + \alpha + d)} <$$

$$\frac{1}{(M^* - M_i^*)^2} \min\left\{\frac{1}{3\lambda_2^2}, \frac{(\sigma M_i^* + \nu + \alpha + d)}{15\lambda_3^2}\right\}$$

$$k_3(\lambda_2 P_m + \lambda_3 A/d)^2 < \frac{4\gamma}{3L}(\lambda_2 P^* + \lambda_3 I^* + \gamma_0).$$

where $P_m = \frac{A_0}{(d_1 + \alpha_1)}$. The stability condition obtained, as given in the statement of the theorem.

Thus $\frac{dV_2}{dt}$ will be negative definite under the given conditions in the statement of the theorem.

Hence, the proof. ■

VI. Numerical Simulation

It is noted here that our aim to study, through a nonlinear model and its qualitative analysis, the role of the awareness programs by media on the spread of Japanese Encephalitis. It is therefore desirable that we show the existence of equilibria of the model as well as the feasibility of stability conditions numerically for a set of parameter values.

To study the dynamical behavior of the model, numerical simulation of the system (2.2) is carried out by MATLAB 6.1, using the following parameter values;

$\sigma = 0.0003$, $\nu = 0.005$, $\alpha = 1/45$, $d = 1/15$, $A = 150$,
$A_0 = 90$, $\alpha_1 = 1/15$, $d_1 = 1/10$, $\gamma = 0.6$, $\lambda_3 = 0.00021$,
$\gamma_0 = 0.3$, $\lambda_1 = 0.0001$, $\lambda_2 = 0.0002$, $\mu = 0.005$, $\mu_0 = 0.06$,
$\lambda_0 = 0.2$, $L = 100$.

The equilibrium values for the model (2.2) are computed as follows:

$I^* = 1243.4375138$, $S_m^* = 308.01757080$, $P^* = 540.00$,
$N^* = 7953.9235910$, $M_i^* = 27.5825593$, $M^* = 50.00$
and $M_e^* = 103.619728$.

The eigenvalues of the variational matrix corresponding to the equilibrium E_2 for the model system (2.2) are
-0.683391, -0.3, -0.226455, -0.167, -0.055305,
$-0.027303 + 0.020532\,i$, $-0.027303 - 0.020532\,i$.

We note that the five eigenvalues found to be negative and the other two eigenvalues have a negative real part. Hence, the endemic equilibrium E_2 is locally asymptotically stable.

For the above data, the computer generated graphs of infective population $'I'$ versus cumulative density of awareness programs $'M_e'$ and infective mosquito population $'M_i'$ versus reservoir population $'P'$ have been drawn in Figs.1 and 2, we may see that all the trajectories initiating inside the region of attraction approach towards the equilibrium values (I^*, M_e^*) and (M_i^*, P^*) respectively. This shows the non-linear stability of (I^*, M_e^*) in $I - M_e$ plane and (M_i^*, P^*) in $M_i - P$ plane.

The variation of infective population $I(t)$ and awareness programs $M_e(t)$ with respect to time $'t'$ for different values of rate of dissemination $'\lambda_1'$ is shown in Figs.3 and 4 respectively. From these figures, it can be noted that as the rate of dissemination $'\lambda_1'$ increases infective population $I(t)$ and awareness programs $M_e(t)$ both decreases.

Further, the variations of the infective population $I(t)$ and the aware population $S_m(t)$ with respect to time $'t'$ for different values of rate of implementation of awareness programs $'\mu'$ are shown in Figs.5 and 6 respectively. From these figures, it is apparent that as the rate of implementation of awareness programs $'\mu'$ increases the infective population $I(t)$ decreases whereas the aware population $S_m(t)$ increases. Also, from Fig.6 it is interesting to observe that for $\mu = 0$ the aware population S_m approaches 0.

It is found that as the growth rate and carrying capacity of mosquito population i.e. $'\gamma'$ and $'L'$ decreases then the infective population $I(t)$ decreases with respect to time $'t'$ are shown in Figs.7 and 8 respectively. Also, the value of transmission coefficients $'\lambda_2'$ and $'\lambda_3'$ due to interaction of susceptible mosquito population with reservoir population and with infected human population increases, the infective population $I(t)$ increases with respect to time $'t'$ are shown in Figs.9 and 10 respectively.

VII. Conclusion

In this paper, a nonlinear mathematical model has been proposed and analyzed to study the effect of awareness programs driven by media on the spread of infectious disease Japanese Encephalitis (JE) considering human, reservoir and

mosquito population, all with variable size structures. It has been considered that the growth rate of awareness programs is proportional to the number of infectives. It has been assumed further that awareness cases some susceptible to isolate themselves from infectives forming a separate subclass in the population. The mosquito population assumed to be governed by a general logistic model. The model is analyzed using stability theory of differential equations and numerical simulation. The model exhibits two equilibria namely, the disease free (DF) and the endemic equilibrium. Results show that the disease free equilibrium (DFE) is stable and unstable under certain condition. The analysis demonstrates that an endemic equilibrium is locally as well as nonlinearly stable under certain conditions. It is shown that with the increase in the carrying capacity and growth rate of mosquito population due to environmental and human related factors, the infected human population increases. Also, when the transmission coefficients due to interaction of susceptible mosquito population with reservoir population and with infected human population increases, the infected population also increases. The model analysis further shows that awareness programs through the media campaigning are helpful in decreasing the spread of infectious disease Japanese Encephalitis (JE) by isolating a fraction of susceptible from infectives.

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Figures with Captions

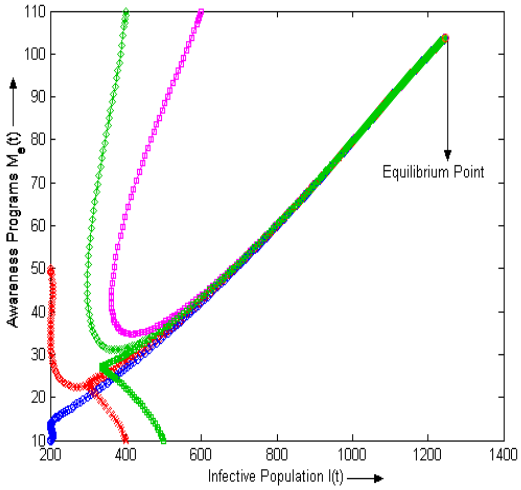

Fig.1. Nonlinear stability of (I^*, M_e^*) in $I - M_e$ plane.

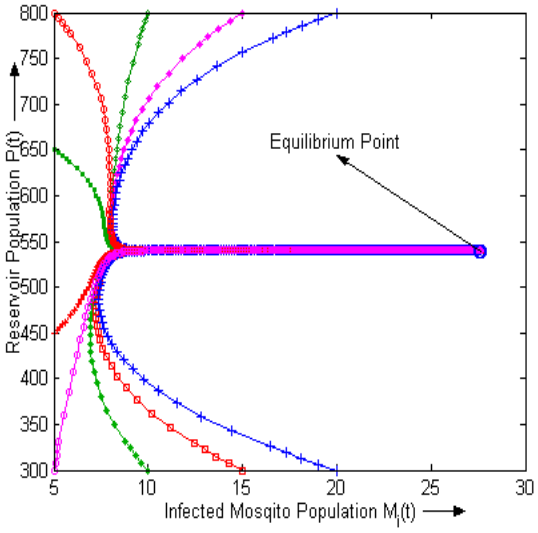

Fig.2. Nonlinear stability of (M_i^*, P^*) in $M_i - P$ plane.

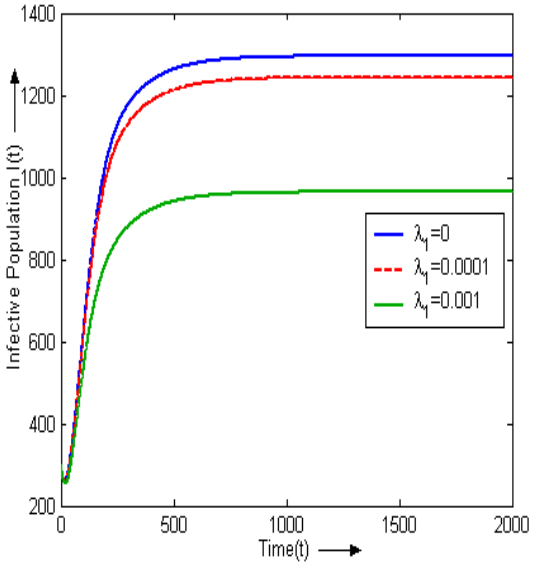

Fig.3. Variation of infective population with time for different values of λ.

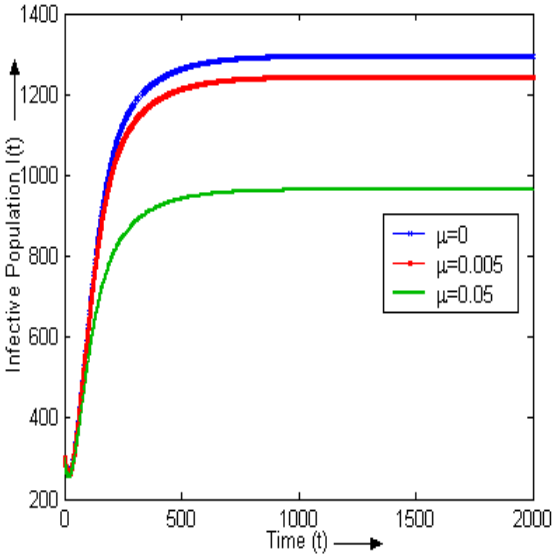

Fig.5. Variation of infective population with time for different values of μ.

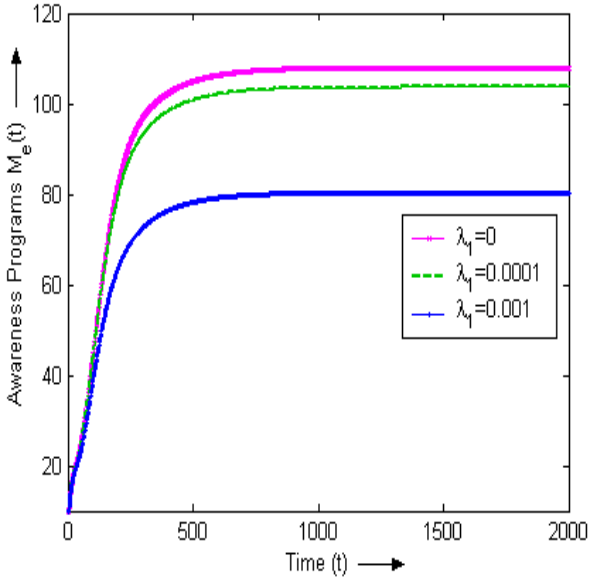

Fig.4. Variation of awareness programs with time for different values of λ.

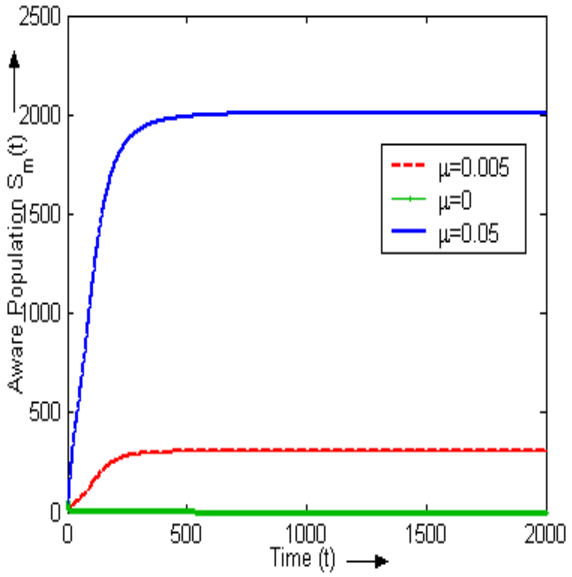

Fig.6. Variation of aware population with time for different values of μ.

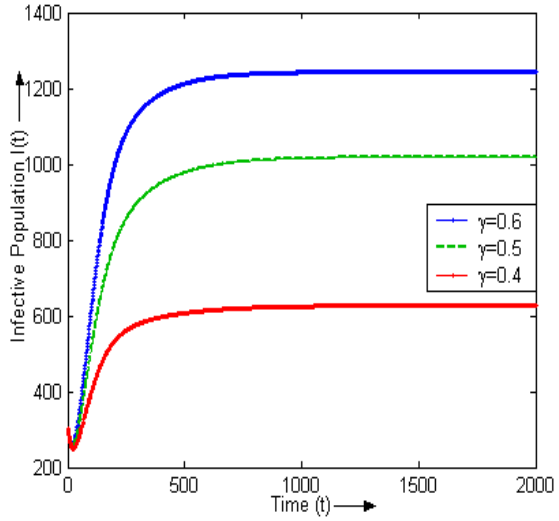

Fig.7. Variation of infective population with time for different values of γ.

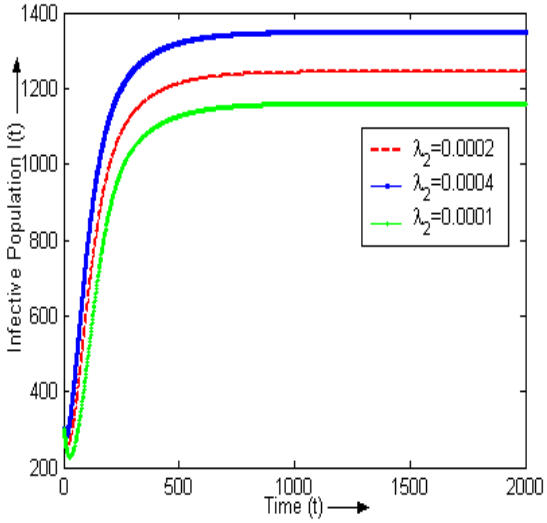

Fig.9. Variation of infective population with time for different values of λ_2.

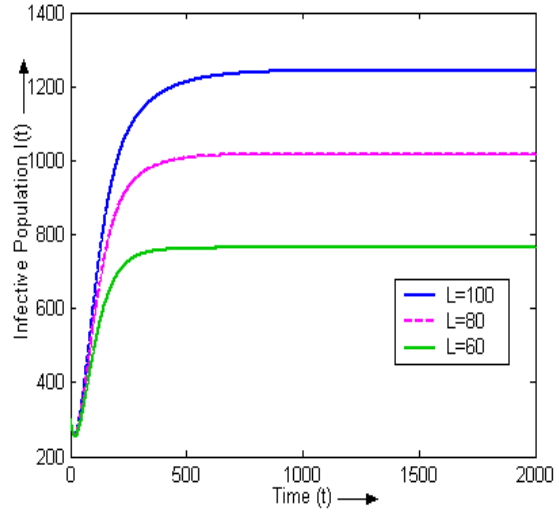

Fig.8. Variation of infective population with time for different values of L.

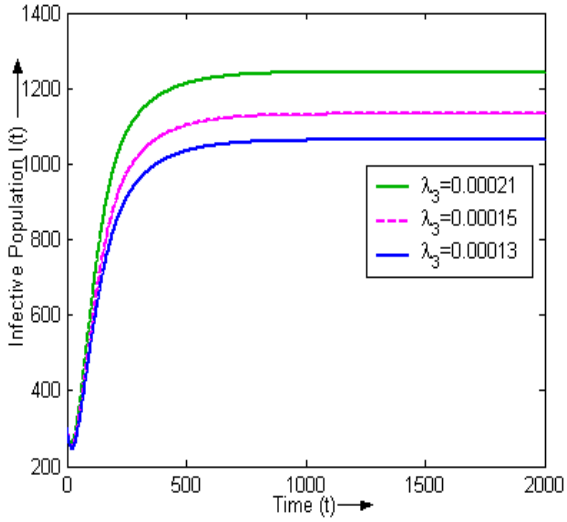

Fig.10. Variation of infective population with time for different values of λ_3.