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Modeling and Analysis of the Spread of Japanese Encephalitis with Environmental Effects

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Abstract

A nonlinear mathematical model for the spread of Japanese Encephalitis, caused by infected mosquito feeding on susceptible human population incorporating demographic and environmental factors is proposed and analyzed. In the modeling process, it is assumed that the growth rates of reservoir animal population and vector mosquito population are enhanced due to environmental discharges caused by human population related factors. The model is analyzed by stability theory of differential equations and computer simulation. Both the disease-free and the endemic equilibria are found and their stability is investigated. It is found that whenever the disease-free equilibrium is locally asymptotically stable, the endemic equilibrium does not exist. The analysis of the model shows that if the growth rates of reservoir animal population and vector mosquito population caused by environmental factors increase, the spread of Japanese Encephalitis increases and the disease becomes 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 and illustrate possible behavioral scenario of the model.

Keywords: Nonlinear Model; Japanese Encephalitis; Reservoir population; Vector Population; Environmental Discharge

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1. Introduction

Japanese Encephalitis Virus (JEV) is an arbovirus causing encephalitis and shares a close genetic relationship with other encephalitic viruses, including St. Louis encephalitis virus (SLEV), West Nile virus (WNV), Murray Valley encephalitis virus (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 (2002). 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.

JEV is the cause of the most important veterinary flavivirus diseases and human epidemic encephalitis in the world, Thongcharoen (1989). The virus is transmitted in an enzootic cycle among mosquitoes and vertebrate amplifying hosts, chiefly Ardeid (wading) birds and domestic pigs, providing a link to humans through their proximity to housing, especially in Asia. Culex mosquitoes, primarily Culex tritaeniorhynchus are the principal vectors, Rosen (1986), Sucharit et al. (1989). The virus is also able to replicate itself in pigs and birds. This means that pig and bird populations, constitute a reservoir of the disease, which may be difficult to eradicate, see Burke and Leake (1988).

JEV is passed on by the bite of an infected mosquito that has previously sucked blood from an infected animal or person, Gould (2002), Easmon (2005). The risk for acquiring JEV among most travelers to Asia is 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 (2002).

In areas where JE is endemic, annual incidence ranges from 1 to 10 per 10,000. Children less than 15 years of age are principally affected, CDC (1993). Infection leads to overt (open) encephalitis in only 1 of 20 to 1000 cases. Encephalitis usually is severe, resulting in neuropsychiatric sequelae in 30% of cases, Okuno (1978), Umenai et al. (1985), Burke and Leake (1988), Halstead (1992), CDC (1993). In tropical zones, the virus has an endemic pattern, with sporadic cases occurring year round, and because most young adults will have acquired immunity the ratio of inapparent to apparent infections may be as high as 300:1. Different strains of JEV show wide variations in virulence for humans, which to some extent is reflected in the very wide case fatality ratios reported, varying from 5 to 40%. In some rural areas, where medical care is not easily available, case fatality rates as high as 70% have been reported. In contrast, high quality medical care may result in rates less than 10%, CDC (1993), Gould (2002), Easmon (2005).

Most people who are infected show only mild symptoms or no symptoms at all. However, at advanced stages, the disease may be fatal. It has an incubation period of 1-2 weeks. The disease begins like flu with headache, fever, chills, anorexia, vomiting, dizziness and drowsiness which in children is often accompanied by abdominal pain and diarrhea, Gould (2002). Gastrointestinal problems including vomiting, as well as confusion and delirium may also be present. In about 1

of every 200 cases, the illness progresses to inflammation of the brain, with more than half of those cases ending in permanent disability or death.

Although, vaccine against JEV is present but it does not give 100% protection. It is, therefore, no alternative to ordinary protection against mosquito bites and in addition to the long term policy of immunizing animals that reduces the potential for amplification of the virus in environment. Unlike, Japan, China and Taiwan, the disease is still spreading in India. This may be because of the high exposure rates of non-immune individuals working outdoors, Gould (2002).

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. (1993), Tapaswi et al. (1995). Mukhopadhyay et al. (1993) 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. (1995) models the spread of JE in human population of varying size from reservoir population through a vector population by considering reservoir population of 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.

We have, however, considered a variable reservoir population having fluctuations due to immigration with a constant rate. This population further increases due to unhygienic environmental conditions, like discharge of household wastes, open drainage of sewage water, manmade water ponds and tanks, ill-ventilated houses, unused tyres, water coolers in the reservoir area. The human and vector (mosquito) populations are considered to be varying in size. This is because the size of human population is subject to frequent deaths due to fatality of the disease and the mosquito population which acts as a transmitter of virus, rapidly changes its size due to environmental considerations. We have used similar techniques from previous studies, Ghosh et al. (2000, 2004), Hethcote (2000), Hsu and Zee (2004), Singh et al. (2003, 2005), Bowman et al. (2005), for constructing and analyzing our proposed model.

The objective of our study is to investigate the transmission dynamics of JEV in three-population system consisting of human, reservoir and vector populations by considering the effects of environmental factors, which are conducive to the growth of reservoir and vector populations.

2. 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 by taking into account the demographic and environmental factors. The total human population $N(t)$ at time t, with constant inflow of susceptibles at the rate A, is divided into two subclasses that is susceptibles $S(t)$ and infecteds $I(t)$ with the number of deaths taken to be proportional to the size in each class. The human population is considered to be varying in size because of the considerable degree of fatality due to the disease. The disease is spread through the direct interaction between susceptible humans and infected vectors (mosquitoes) which is modeled using bilinear interaction (law of mass action), Ghosh et al. (2004, 2005), Naresh et al. (2008),

Singh et al. (2003, 2005). It is known that various kinds of household and other wastes, discharged into the environment in residential areas of population, provide a very conducive environment for the growth of mosquito population. Thus, unhygienic environmental conditions caused by human population become responsible for the spread of the disease. Since the mosquito population is subject to rapid change, it is assumed that the mosquito population is growing logistically with given intrinsic growth rate and carrying capacity. The growth rate of it is further assumed to increase with increase in the cumulative density of environmental discharges by the human population, Ghosh et al. (2000, 2004), Singh et al. (2003, 2005). The total vector (Culex species mosquitoes) population $M(t)$ is divided into susceptible mosquito population $M_S(t)$ and infected mosquito population $M_I(t)$. There is no immune class in the mosquito population since it acts as a transmitter of virus only. The susceptible mosquito is infected 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, though it was assumed to be constant by taking birth and death rates equal, Tapaswi et al. (1995). It may be noted that our purpose is to study the effect of environmental factors on the spread of JE in the human population and therefore we do not consider compartments in the reservoir population. The growth rate of reservoir population is further assumed to increase with increase in the cumulative density of environmental discharges by the human population. The block diagram of the model is given in Figure 1 (dash line denotes interaction).

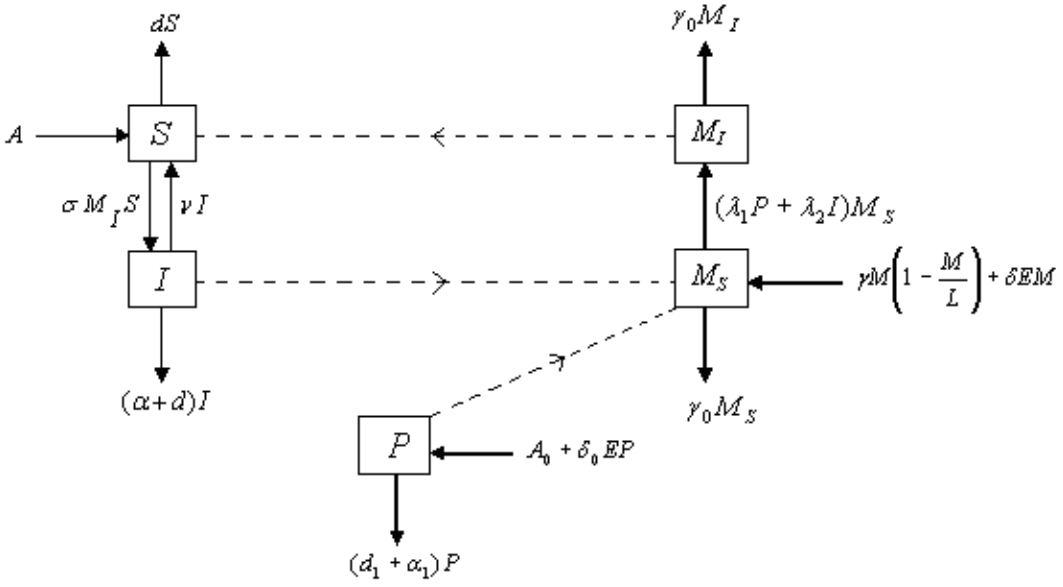

Figure 1. Block diagram of the model.

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 equations:

$$\frac{dS}{dt} = A - \sigma M_I S - dS + \nu I$$
$$\frac{dI}{dt} = \sigma M_I S - (\nu + \alpha + d)I$$
$$\frac{dN}{dt} = A - dN - \alpha I$$
$$\frac{dP}{dt} = A_0 - (d_1 + \alpha_1)P + \delta_0 EP \tag{2.1}$$
$$\frac{dM_S}{dt} = \gamma M\left(1 - \frac{M}{L}\right) - \lambda_1 M_S P - \lambda_2 M_S I - \gamma_0 M_S + \delta EM$$
$$\frac{dM_I}{dt} = \lambda_1 M_S P + \lambda_2 M_S I - \gamma_0 M_I$$
$$\frac{dM}{dt} = \gamma M\left(1 - \frac{M}{L}\right) - \gamma_0 M + \delta EM$$
$$\frac{dE}{dt} = Q_0 + \theta N - \theta_0 E$$
$$S + I = N,\ M_S + M_I = M,$$

and,

$$S(0) > 0,\ I(0) \geq 0,\ N(0) > 0,\ P(0) \geq 0,\ M_S(0) \geq 0,\ M_I(0) \geq 0,\ M(0) \geq 0,\ E(0) \geq 0.$$

Here, $E(t)$ is the cumulative density of environmental discharges conducive to the growth of reservoir and mosquito population; 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 the rate by which infected individuals are recovered and become susceptible again. A_0 is the constant immigration rate of infected reservoir population; d_1 is the natural death rate of reservoir population and α_1 is the death rate of reservoir population due to disease and control measures.

The constant L is the carrying capacity of mosquito population in the natural environment; γ is its growth rate, γ_0 is the death rate of mosquito population due to natural cause as well as control measures; λ_1 and λ_2 are the transmission coefficients due to interaction of susceptible mosquito population with reservoir population and with infected human population respectively; δ_0 and δ are the per capita growth rate coefficients of the reservoir and mosquito population, respectively due to conducive environmental discharges, the cumulative density of environmental discharge grows due to constant influx Q_0 as well as due to human activities at the rate θ and θ_0 is the depletion rate coefficient of the environmental discharges. In the model, all the dependent variables and parameters are assumed to be non-negative.

3. Equilibrium Analysis

To analyze the model (2.1), we consider the following reduced system (since $S + I = N$ and $M_S + M_I = M$),

$$\begin{aligned}
\frac{dI}{dt} &= \sigma M_I(N-I) - (\nu+\alpha+d)I\,,\\
\frac{dN}{dt} &= A - dN - \alpha I\,,\\
\frac{dP}{dt} &= A_0 - (d_1+\alpha_1)P + \delta_0 EP\,,\\
\frac{dM_I}{dt} &= \lambda_1(M-M_I)P + \lambda_2(M-M_I)I - \gamma_0 M_I\,,\\
\frac{dM}{dt} &= \gamma M\left(1-\frac{M}{L}\right) - \gamma_0 M + \delta EM\,,\\
\frac{dE}{dt} &= Q_0 + \theta N - \theta_0 E\,.
\end{aligned} \tag{3.1}$$

Solving the right hand side of the model system (3.1) by equating it to zero, we obtain the following biologically relevant equilibria.

(1) Disease-free equilibrium, $W_0 = \left(0,\ \frac{A}{d},\ \widetilde{P},\ 0,\ 0,\ \widetilde{E}\right)$, exists without any condition,

where

$$\widetilde{P} = \frac{A_0\theta_0}{\theta_0(d_1+\alpha_1) - \delta_0(Q_0+\theta A/d)} \quad \text{and} \quad \widetilde{E} = \frac{Q_0+\theta A/d}{\theta_0}\,.$$

The existence of W_0 is obvious. This equilibrium implies that if the mosquito 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 $\widetilde{P}$. It may also be noted that in the absence of mosquito population, the infected human population will become zero.

(2) Endemic equilibrium, $W_1 = (I^*,\ N^*,\ P^*, M_I^*,\ M^*,\ E^*)$

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^*, N^*, P^*, M_I^*, M^* and E^*. These equilibrium values are explicitly given by equations (3.3- 3.7).

We prove the existence of the second equilibrium W_1 by setting right hand side of equations (3.1) to zero and solving the resulting algebraic equations, we get,

$$\sigma M_I(N-I)-(\nu+\alpha+d)I=0, \tag{3.2}$$

$$I^*=\frac{A-dN^*}{\alpha}, \tag{3.3}$$

$$E^*=\frac{Q_0+\theta N^*}{\theta_0}, \tag{3.4}$$

$$P^*=\frac{A_0}{(d_1+\alpha_1)-\delta_0 E^*}, \tag{3.5}$$

$$M^*=\frac{L}{\gamma}(\gamma-\gamma_0+\delta E^*), \tag{3.6}$$

$$M_I^*=\frac{(\lambda_1 P^*+\lambda_2 I^*)M^*}{\lambda_1 P^*+\lambda_2 I^*+\gamma_0}\ . \tag{3.7}$$

In the equilibrium W_1 , N^* is the positive root of the following equation, which can be obtained from equation (3.2) after using I^* and M_I^* from equations (3.3) and (3.7), respectively. Using this value of $N=N^*>0$ in equations (3.3 - 3.7) we obtain other equilibrium values,

$$F(N)=\sigma\left[\lambda_1 P+\lambda_2\left(\frac{A-dN}{\alpha}\right)\right]MN-\gamma_0(\nu+\alpha+d)\left(\frac{A-dN}{\alpha}\right)$$
$$-(\sigma M+\nu+\alpha+d)\left[\lambda_1 P+\lambda_2\left(\frac{A-dN}{\alpha}\right)\right]\left(\frac{A-dN}{\alpha}\right). \tag{3.8}$$

It would be sufficient if we show that $F(N) = 0$ has one and only one root. From equation (3.8), we note that $F\left(\frac{A}{\alpha+d}\right)<0$ and $F\left(\frac{A}{d}\right)>0$. This implies that there exists a root N^* of $F(N) = 0$ in $\frac{A}{\alpha+d}<N<\frac{A}{d}$. Also, $F'(N)>0$, provided $\alpha\theta\lambda_1\delta_0 A_0>d\theta_0\lambda_2(d_1+\alpha_1)^2$ in $\frac{A}{\alpha+d}<N<\frac{A}{d}$.

Thus, there exists a unique positive root of $F(N) = 0$, (say N^*) in $\frac{A}{\alpha+d}<N<\frac{A}{d}$. Knowing the value of N^*, the values of I^*, P^*, M_I^*, M^* and E^* can be computed from equations (3.3 – 3.7).

3.1. Boundedness of Solutions

Continuity of right hand side of system (3.1) and its derivative imply that the model is well posed for $N > 0$. The invariant region where solution exists is obtained as follows:

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

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 globally for all $t > 0$ in the invariant and compact set,

$$\Omega = \{(I, N, P, M_I, M, E) \in R_+^6 : 0 \leq I \leq N \leq \frac{A}{d} + \varepsilon,\ 0 \leq P \leq P_m,\ 0 \leq M_I \leq M \leq M_m,\ 0 \leq E \leq E_m\},$$

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

Here,

$$P_m = \frac{A_0 \theta_0}{\theta_0 (d_1 + \alpha_1) - \delta_0 (Q_0 + \theta A / d)},\quad M_m = \frac{L}{\gamma}\left[\gamma - \gamma_0 + \frac{\delta (Q_0 + \theta A / d)}{\theta_0}\right] \text{ and}$$

$$E_m = \frac{Q_0 + \theta A / d}{\theta_0}.$$

As $N(t)$ tends to zero, $S(t)$ and $I(t)$ also tend 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 $N(t) = 0$.

3.2. Positivity of Solutions

Let the initial data be $I(0) = I_0 \geq 0$, $N(0) = N_0 > 0$, $P(0) = P_0 > 0$, $M_I(0) = M_{I0} \geq 0$, $M(0) = M_0 \geq 0$ and $E(0) = E_0 > 0$ for all $t \geq 0$. Then, the solution $[I(t), N(t), P(t), M_I(t), M(t), E(t)]$ of the model remain positive for all time $t \geq 0$. From the first equation of model (3.1) we get $I'(t) \geq -(\nu + \alpha + d) I(t)$, which gives $I(t) \geq c_1 e^{-(\nu + \alpha + d)\, t}$.

Here c_1 is a constant of integration. A similar reasoning for the remaining equations shows that they are always positive in Ω for $t > 0$. We assume that at $t = 0$, $N(t)$, $I(t)$, $P(t)$, $M_I(t)$, $M(t)$ and $E(t)$ are all non-negative and that $N(0) > 0$.

We notice that

$\frac{A}{(\alpha + d)} \leq \liminf N(t) \leq \limsup N(t) \leq \frac{A}{d}$, this implies that $S(t) > 0$ for all t.

4. Stability Analysis

Now, we analyze the stability of equilibria W_0 and W_1 and the stability results of these equilibria are stated in the following theorems.

Theorem 4.1.

(i) The disease-free equilibrium W_0 is locally asymptotically stable if

$$\gamma_0 > \gamma + \frac{\delta(Q_0 + \theta A/d)}{\theta_0} \text{ otherwise, } W_0 \text{ is unstable.}$$

(ii) The endemic equilibrium W_1 exists whenever the disease-free equilibrium W_0 is unstable and is locally asymptotically stable, under the following conditions:

$$\alpha\theta^2\delta_0^2 P^{*2} < \frac{4}{9}\sigma d\theta_0^2 (d_1+\alpha_1-\delta_0 E^*)^2 M_I^* \tag{4.1}$$

$$\sigma^2(N^*-I^*)^2(M^*-M_I^*)^2 < \frac{1}{3}(\sigma M_I^*+\nu+\alpha+d)(\lambda_1 P^*+\lambda_2 I^*+\gamma_0)^2 \min\left\{\frac{1}{2\lambda_1^2}, \frac{(\sigma M_I^*+\nu+\alpha+d)}{3\lambda_2^2}\right\} \tag{4.2}$$

$$k_3\delta^2(\lambda_1 P^*+\lambda_2 I^*)^2 < \frac{\gamma^2(\lambda_1 P^*+\lambda_2 I^*+\gamma_0)}{3L^2} \tag{4.3}$$

Proof:

See Appendix I.

Theorem 4.2.

The endemic equilibrium W_1 is nonlinearly asymptotically stable in the region Ω if the following conditions are satisfied:

$$\alpha\lambda_1^2\theta^2\delta_0^2 A_0^2 < \frac{4}{9}\sigma d(d_1+\alpha_1-\delta_0 E^*)^2[\theta_0(d_1+\alpha_1)-\delta_0(Q_0+\theta A/d)]^2 M_I^*, \tag{4.4}$$

$$\sigma^2\frac{A^2}{d^2}(M^*-M_I^*)^2 < \frac{1}{3}\gamma_0^2(\sigma M_I^*+\nu+\alpha+d)\min.\left\{\frac{1}{2\lambda_1^2}, \frac{(\sigma M_I^*+\nu+\alpha+d)}{3\lambda_2^2}\right\}, \tag{4.5}$$

$$k_3\delta^2\left(\frac{\lambda_1 A_0\theta_0}{\theta_0(d_1+\alpha_1)-\delta_0(Q_0+\theta A/d)}+\frac{\lambda_2 A}{d}\right)^2 < \frac{\gamma_0\gamma^2}{3L^2}. \tag{4.6}$$

Proof:

See Appendix II.

Remark

If the per capita growth rate coefficients of the mosquito and reservoir population due to conducive environmental discharge tend to zero i.e., $\delta \to 0$ and $\delta_0 \to 0$, then inequalities (4.1), (4.3), (4.4) and (4.6) are automatically satisfied. This implies that the environmental conditions conducive to the growth of reservoir animal population and mosquito population have destabilizing effect on the system.

The above theorems imply that under certain conditions, if the reservoir and mosquito population increase due to environmental factors, the number of infecteds increases, which lead to fast spread of encephalitis.

5. Numerical Simulations

It is noted here that our aim is to study, through a nonlinear model and its qualitative analysis, the role of environmental factors 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 (3.1) is carried out by MAPLE 7.0, using the parameter values; Ghosh et al. (2000, 2004):
$\sigma = 0.0003, \nu = 4.5, \alpha = 1/45, d = 1/65, A = 150, A_0 = 50, \alpha_1 = 1/15, d_1 = 1/10, \delta = 0.0001,$
$\delta_0 = 0.000001, \lambda_1 = 0.0001, \lambda_2 = 0.00021, \gamma = 0.6, \gamma_0 = 0.3, L = 1000, Q_0 = 2, \theta = 0.0002,$
$\theta_0 = 0.0001.$

The equilibrium values for the model system (3.1) are computed as follows:

$I^* = 1646.987294, N^* = 7608.9165157, P^* = 380.3761404, M_I^* = 4178.390656,$
$M^* = 7443.566607, E^* = 35217.83303.$

The eigenvalues of the variational matrix corresponding to the endemic equilibrium of the model are

-6.016911750, -0.4517175783, -0.02177481172, -0.0001094418333, 3.721783284, -0.1314488664.

Since all the eigenvalues are found to be negative, the endemic equilibrium is locally asymptotically stable for the above set of parameter values.

The results of numerical simulation are displayed graphically in figures (2-10). Figure 2 shows that the system (3.1) is nonlinearly asymptotically stable in M_I -P plane. All the trajectories starting from different initial starts, reach the endemic equilibrium W_1.

1. $I(0) = 1000$, $N(0) = 7000$, $P(0) = 500$, $M_I(0) = 9000$, $M(0) = 10000$, $E(0) = 40000$.
2. $I(0) = 1000$, $N(0) = 7000$, $P(0) = 200$, $M_I(0) = 1000$, $M(0) = 7000$, $E(0) = 40000$.
3. $I(0) = 1000$, $N(0) = 7000$, $P(0) = 500$, $M_I(0) = 1000$, $M(0) = 6800$, $E(0) = 40000$.
4. $I(0) = 1000$, $N(0) = 7000$, $P(0) = 200$, $M_I(0) = 9000$, $M(0) = 10000$, $E(0) = 40000$.

Hence, we infer that the system (3.1) may be nonlinearly asymptotically stable about this equilibrium point W_1 for the above set of parameter values. In Figures (3-5), the variation of reservoir animal population, infected mosquito population and infected human population respectively for different values of cumulative density of environmental discharge with time is shown. From these figures, it is clear that with the increase in the level of environmental discharge due to constant influx (Q_0), the reservoir animal population and infected mosquito population increases. This increase in infected mosquito population and reservoir animal population ultimately results in increasing the infected human population, (see Figure 5). Thus, the unhygienic environmental discharge conducive to the growth of reservoir animal population and the mosquito population should be controlled to stop spreading of JE. In figure 6, we show the variation of infected human population with time to see the effect of cumulative density of environmental discharge due to human activities (θ).

It is found that as the rate of cumulative density of environmental discharge due to human activities increases, the number of infected individuals also increases. Figures 7-8 depict the role of conducive environmental discharge (δ_0) on the reservoir animal population and infected human population, respectively with time. It is found that the reservoir animal population increases with increase in the per capita growth rate coefficient of the reservoir population due to conducive environmental discharge. This increase in the infected reservoir population increases the infected human population i.e. the increase in the spread of JE, (see Figure 8).

It is, therefore, urged that suitable mechanism be devised to keep the environmental hygiene pollution-free so that the spread of JEV is minimal. In Figures 9-10, we have shown the variation of infected mosquito population and infected human population with (δ) the per capita growth rate coefficient of mosquito population due to conducive environmental discharge with time. We observe from these figures that infected mosquito population increases with increase in the value of (δ), which, in turn, increases the infected human population.

From the above analysis, it may be concluded that the unhygienic environmental discharge conducive to the growth of mosquito population and the reservoir animal population is mainly responsible for the spread of Japanese Encephalitis. Thus, in order to keep the spread of JE controlled, the unhygienic environmental discharges should be kept at minimum so that the accumulation of infected mosquito population and reservoir animal population is restricted. For this, a suitable control mechanism may be devised to curb the growth of mosquito population and reservoir animal population which otherwise increases due to human population related factors and unhygienic environmental conditions leading to fast spread of Japanese Encephalitis.

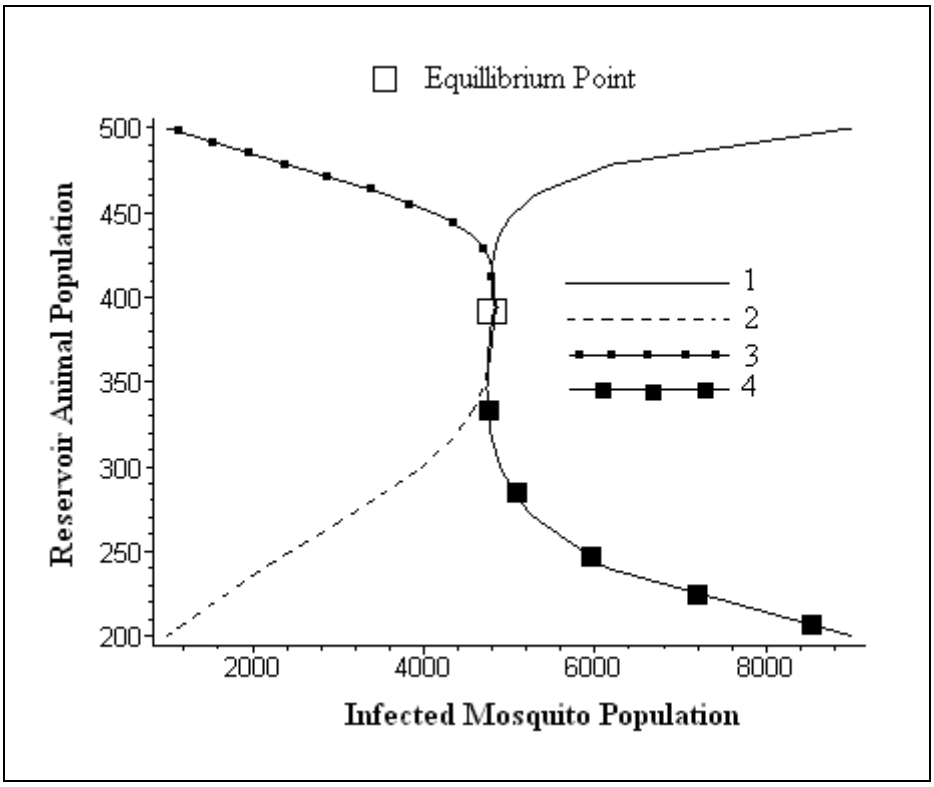

Figure 2. Variation of infected mosquito population with reservoir animal population.

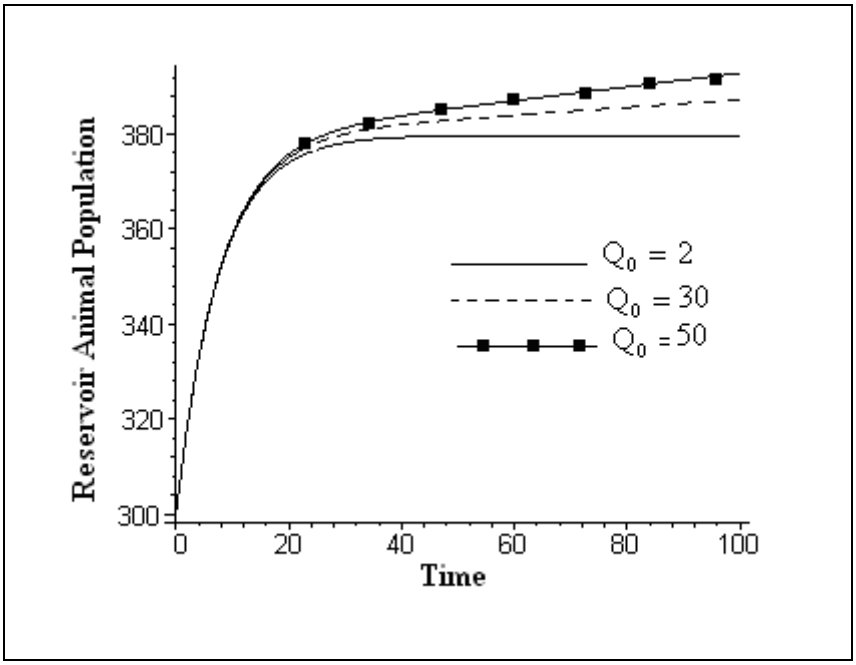

Figure 3. Variation of reservoir animal population for different values of Q_0

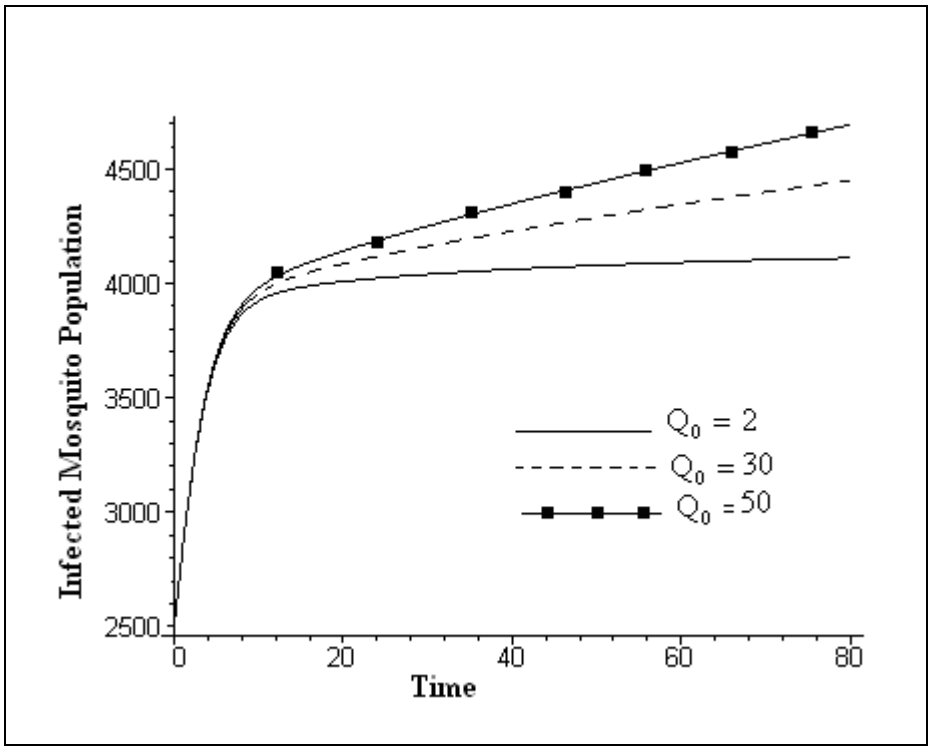

Figure 4. Variation of infected mosquito population for different values of Q_0

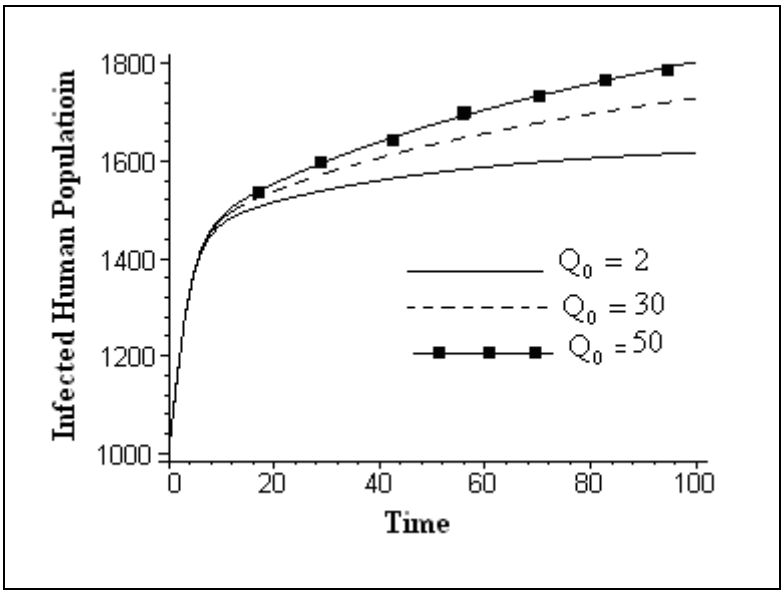

Figure 5. Variation of infected human population for different values of Q_0

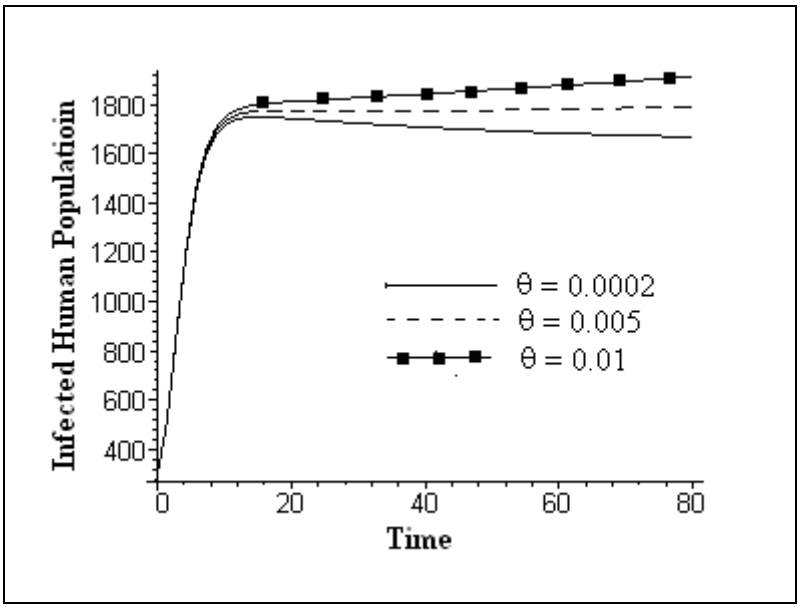

Figure 6. Variation of infected human population for different values of θ

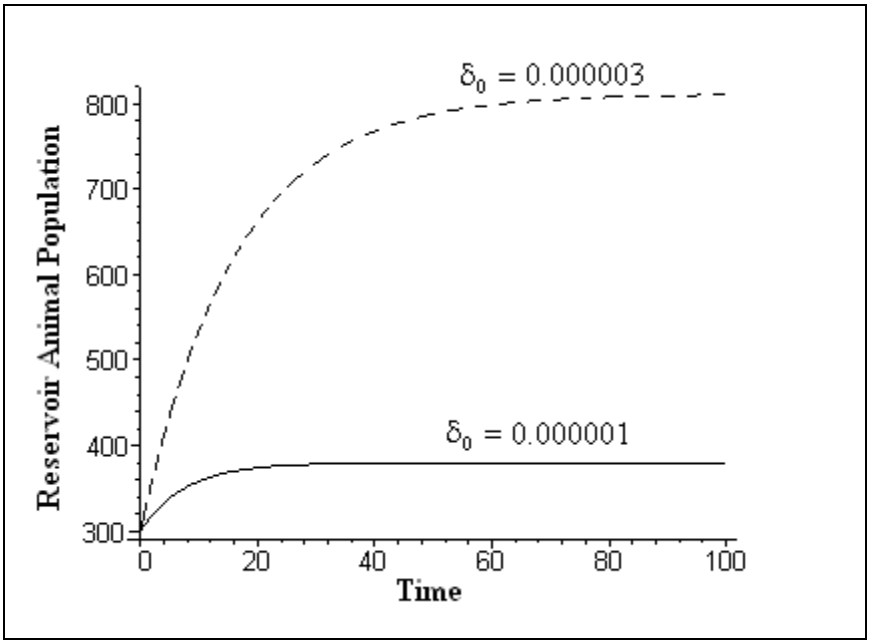

Figure 7. Variation of reservoir animal population for different values of δ_0

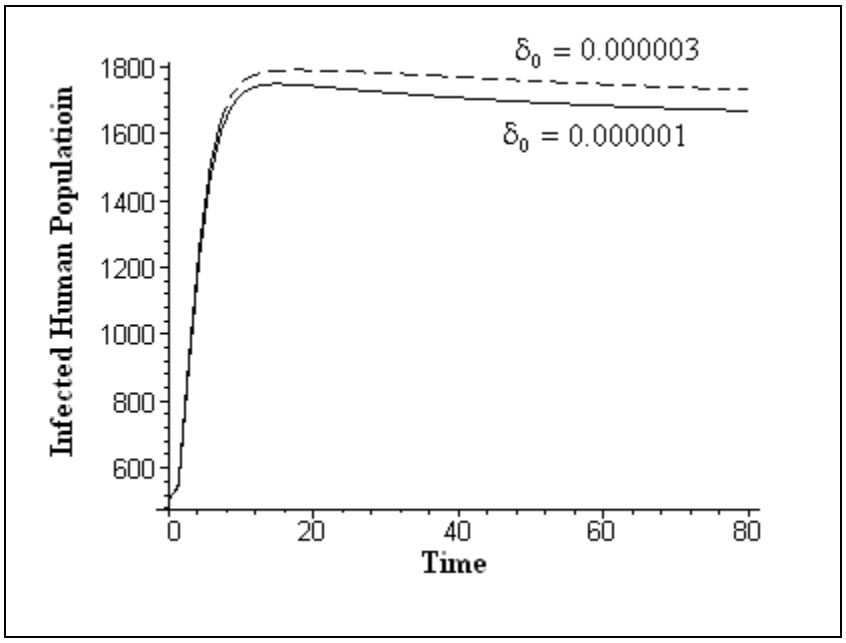

Figure 8. Variation of infected human population for different values of δ_0

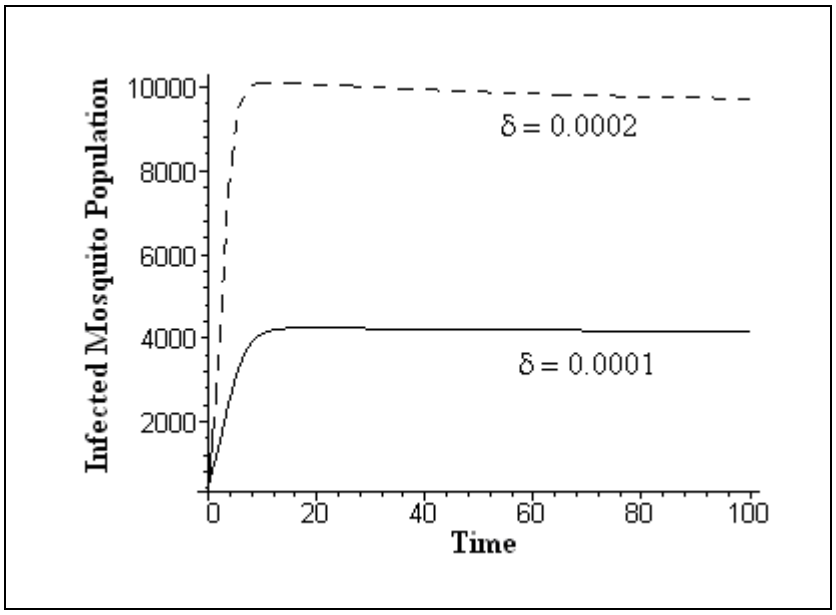

Figure 9. Variation of infected mosquito population for different values of δ

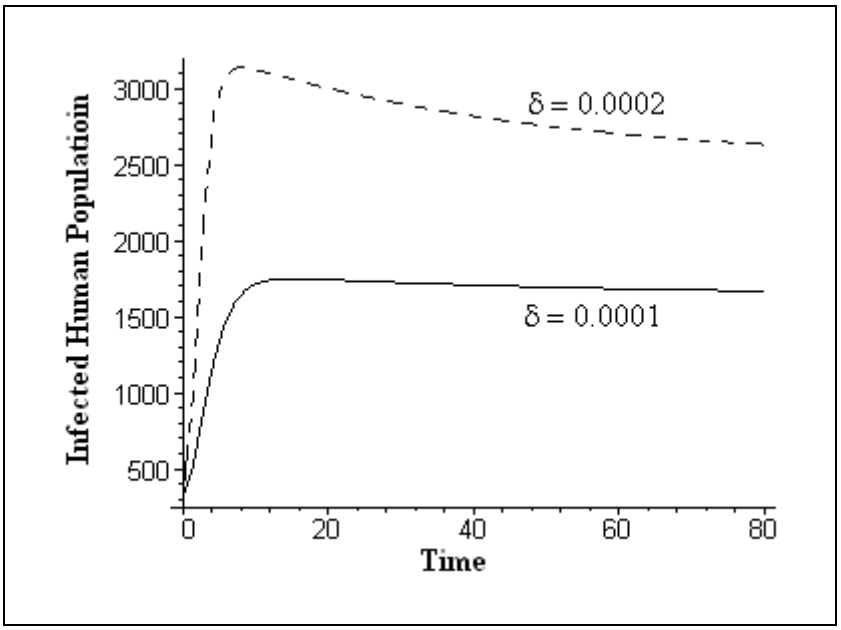

Figure 10. Variation of infected human population for different values of δ

6. Conclusion

In this paper, a nonlinear mathematical model is proposed and analyzed to study the effect of environment on the transmission dynamics of Japanese Encephalitis considering human, reservoir and mosquito population, all with variable size structures. The reservoir and mosquito populations are assumed to increase by environmental and human population related factors. The mosquito population is 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 and the endemic equilibrium. Results show that the disease-free equilibrium is stable if decay coefficient of mosquito population is maintained at a level higher than their growth rate. Also, whenever the disease-free equilibrium is stable, the endemic equilibrium does not exist. The endemic equilibrium, which exists whenever disease-free equilibrium is unstable, is found to be nonlinearly asymptotically stable under certain conditions.

It is shown that with the increase in the reservoir and mosquito population due to environmental and human related factors, the infected human population increases. It has been pointed out that constant migration in human and reservoir population makes the disease more endemic. Our study shows that in the absence of infected reservoir or mosquitoes into the human community, JE can be eradicated from entire mosquito-reservoir-human population. Also, as the level of environmental conditions improves through improved drainage system, preventing stagnant water, fogging, etc. the spread of JE can be controlled. Therefore, in order to control the spread of JE, an effective control mechanism should be adopted to curb the growth of infected reservoir animal population and mosquito population and preventive measures should be taken against mosquito bites.

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APPENDIX – I

Proof of Theorem 4.1.

The variational matrix for the system (3.1) corresponding to equilibrium

$W_0 = \left(0, \frac{A}{d}, \widetilde{P}, 0, \widetilde{E}\right)$ is given by,

$$J_0 = \begin{bmatrix} -(\nu+\alpha+d) & 0 & 0 & \sigma A/d & 0 & 0 \\ -\alpha & -d & 0 & 0 & 0 & 0 \\ 0 & 0 & -(d_1+\alpha_1-\delta_0\widetilde{E}) & 0 & 0 & \delta_0 P \\ 0 & 0 & 0 & -(\lambda_1\widetilde{P}+\gamma_0) & \lambda_1\widetilde{P} & 0 \\ 0 & 0 & 0 & 0 & \gamma-\gamma_0+\dfrac{\delta(Q_0+\theta A/d)}{\theta_0} & 0 \\ 0 & \theta & 0 & 0 & 0 & -\theta_0 \end{bmatrix}$$

The eigenvalues of J_0 are $\psi_1 = -(\nu+\alpha+d)$, $\psi_2 = -d$, $\psi_3 = -(d_1+\alpha_1-\delta_0\widetilde{E})$, $\psi_4 = -(\lambda_1\widetilde{P}+\gamma_0)$, $\psi_5 = \gamma-\gamma_0+\dfrac{\delta(Q_0+\theta A/d)}{\theta_0}$ and $\psi_6 = -\theta_0$. Since all the model parameters are assumed to be nonnegative, it follows that ψ_i (i = 1, 2, 3, 4, 6) < 0. The stability of W_0 will depend on the sign of ψ_5. Thus, the disease free equilibrium W_0 is stable if $\gamma_0 > \gamma + \dfrac{\delta(Q_0+\theta A/d)}{\theta_0}$, i.e., the decay coefficient of mosquito population is higher than their growth rate.

To establish the local stability of the endemic equilibrium W_1, we consider the following positive definite function,

$$U_1 = \frac{1}{2}(k_0 i^2 + k_1 n^2 + k_2 p^2 + k_3 m_i^2 + k_4 m^2 + k_5 e^2),$$

where k_i's (i = 0, 1, 2, 3, 4, 5) are positive constants to be chosen appropriately and i, n, p, m_i, m and e are small perturbations about W_1, defined as follows,

$$I = I^* + i,\ N = N^* + n,\ P = P^* + p,\ M_i = M_i^* + m_i,\ M = M^* + m \text{ and } E = E^* + e.$$

Differentiating above equation, with respect to 't', and using the linearized system corresponding to W_1, we get

$$\begin{aligned}\frac{dU_1}{dt} &= -k_0[\sigma M_I^* + \nu + \alpha + d]i^2 + k_0\sigma M_I^* in + k_0\sigma(N^* - I^*)im_i - k_1\alpha ni - k_1 d n^2 \\ &\quad - k_2(d_1 + \alpha_1 - \delta_0 E^*)p^2 + k_2\delta_0 P^* pe + k_3\lambda_2(M^* - M_I^*)im_i + k_3\lambda_1(M^* - M_I^*)pm_i \\ &\quad - k_3(\lambda_1 P^* + \lambda_2 I^* + \gamma_0)m_i^2 + k_3(\lambda_1 P^* + \lambda_2 I^*)mm_i - k_4\frac{\gamma}{L}M^* m^2 + k_4\delta M^* me + k_5\theta ne - k_5\theta_0 e^2.\end{aligned}$$

Now, $\frac{dU_1}{dt}$ will be negative definite under the following conditions,

(i) $(k_0\sigma M_I^* - k_1\alpha)^2 < \frac{2}{3}k_0 k_1 d(\sigma M_I^* + \nu + \alpha + d)$

(ii) $k_0\sigma^2(N^* - I^*)^2 < \frac{1}{3}k_3(\lambda_1 P^* + \lambda_2 I^* + \gamma_0)(\sigma M_I^* + \nu + \alpha + d)$

(iii) $k_2\delta_0^2 P^{*2} < \frac{2}{3}k_5\theta_0(d_1 + \alpha_1 - \delta_0 E^*)$

(iv) $k_3\lambda_2^2(M^* - M_I^*)^2 < \frac{1}{3}k_0(\sigma M_I^* + \nu + \alpha + d)(\lambda_1 P^* + \lambda_2 I^* + \gamma_0)$

(v) $k_3\lambda_1^2(M^* - M_I^*)^2 < \frac{1}{2}k_2(d_1 + \alpha_1 - \delta_0 E^*)(\lambda_1 P^* + \lambda_2 I^* + \gamma_0)$

(vi) $k_3(\lambda_1 P^* + \lambda_2 I^*)^2 < \frac{1}{2}k_4\frac{\gamma}{L}M^*(\lambda_1 P^* + \lambda_2 I^* + \gamma_0)$

(vii) $k_4\delta^2 M^{*2} < \frac{2}{3}k_5\frac{\gamma}{L}\theta_0 M^*$

(viii) $k_5\theta^2 < \frac{2}{3}k_1 d\theta_0$.

After choosing $k_0 = 1$, $k_1 = \frac{\sigma M_I^*}{\alpha}$, $k_2 = \frac{1}{(d_1 + \alpha_1 - \delta_0 E^*)}$ and $k_5 = \frac{1}{\theta_0}$ we can choose k_4 and k_3 such that

$$\alpha\theta^2\delta_0^2 P^{*2} < \frac{4}{9}\sigma d\theta_0^2 (d_1+\alpha_1-\delta_0 E^*)^2 M_I^*$$

$$\frac{3\sigma^2(N^*-I^*)^2}{(\lambda_1 P^*+\lambda_2 I^*+\gamma_0)(\sigma M_I^*+\nu+\alpha+d)} < k_3 < \frac{(\lambda_1 P^*+\lambda_2 I^*+\gamma_0)}{(M^*-M_I^*)^2}\min.\left\{\frac{1}{2\lambda_1^2}, \frac{(\sigma M_I^*+\nu+\alpha+d)}{3\lambda_2^2}\right\}$$

$$\frac{2k_3 L(\lambda_1 P^*+\lambda_2 I^*)^2}{\gamma M^*(\lambda_1 P^*+\lambda_2 I^*+\gamma_0)} < k_4 < \frac{2}{3}\frac{\gamma}{L\delta^2 M^*}.$$

The stability conditions are then obtained as given in the theorem. Hence, $\frac{dU_1}{dt}$ will be negative definite under the conditions (4.1), (4.2) and (4.3) as stated in the statement of the theorem, showing that W_1 is locally asymptotically stable. Hence, the proof.

APPENDIX – II

Proof of Theorem 4.2.

Consider the following positive definite function,

$$U_2 = \frac{k_0}{2}(I-I^*)^2 + \frac{k_1}{2}(N-N^*)^2 + \frac{k_2}{2}(P-P^*)^2 + \frac{k_3}{2}(M_I-M_I^*)^2 + k_4\left(M-M^*-M^*\ln\frac{M}{M^*}\right) + \frac{k_5}{2}(E-E^*)^2,$$

where the coefficients k_0, k_1, k_2, k_3 k_4 and k_5 can be chosen appropriately. Differentiating the above equation with respect to 't' and using (3.1), we get

$$\begin{aligned}\frac{dU_2}{dt} &= -k_3(\lambda_1 P+\lambda_2 I)(M_I-M_I^*)^2 - k_0(\sigma M_I^*+\nu+\alpha+d)(I-I^*)^2 - k_1 d(N-N^*)^2 \\ &\quad - k_2(d_1+\alpha_1-\delta_0 E^*)(P-P^*)^2 - k_3\gamma_0(M_I-M_I^*)^2 - k_4\frac{\gamma}{L}(M-M^*)^2 - k_5\theta_0(E-E^*)^2 \\ &\quad + (k_0\sigma M_I^*-k_1\alpha)(I-I^*)(N-N^*) + k_3\lambda_1(M^*-M_I^*)(P-P^*)(M_I-M_I^*) \\ &\quad + k_2\delta_0 P(P-P^*)(E-E^*) + k_3(\lambda_1 P+\lambda_2 I)(M_I-M_I^*)(M-M^*) \\ &\quad + k_0\sigma(N-I)(M_I-M_I^*)(I-I^*) + k_3\lambda_2(M^*-M_I^*)(M_I-M_I^*)(I-I^*) \\ &\quad + k_4\delta(M-M^*)(E-E^*) + k_5\theta(E-E^*)(N-N^*).\end{aligned}$$

Now, $\frac{dU_2}{dt}$ will be negative definite under the following conditions,

(i) $(k_0\sigma M_I^* - k_1\alpha)^2 < \frac{2}{3}k_0k_1d(\sigma M_I^* + \nu + \alpha + d)$

(ii) $k_0\sigma^2(N-I)^2 < \frac{1}{3}k_3\gamma_0(\sigma M_I^* + \nu + \alpha + d)$

(iii) $k_2\delta_0^2P^2 < \frac{2}{3}k_5\theta_0(d_1 + \alpha_1 - \delta_0E^*)$

(iv) $k_3(\lambda_1P + \lambda_2I)^2 < \frac{1}{2}k_4\frac{\gamma\gamma_0}{L}$

(v) $k_3\lambda_1^2(M^* - M_I^*)^2 < \frac{1}{2}k_2\gamma_0(d_1 + \alpha_1 - \delta_0E^*)$

(vi) $k_3\lambda_2^2(M^* - M_I^*)^2 < \frac{1}{3}k_0\gamma_0(\sigma M_I^* + \nu + \alpha + d)$

(vii) $k_4\delta^2 < \frac{2}{3}k_5\frac{\gamma\theta_0}{L}$

(viii) $k_5\theta^2 < \frac{2}{3}k_1\theta_0d$.

Now, choosing $k_0 = 1$, $k_1 = \frac{\sigma M_I^*}{\alpha}$, $k_2 = \frac{1}{(d_1 + \alpha_1 - \delta_0E^*)}$ and $k_5 = \frac{1}{\theta_0}$ such that:

$$\alpha\theta^2\delta_0^2P_m^2 < \frac{4}{9}\sigma d\ \theta_0^2(d_1 + \alpha_1 - \delta_0E^*)^2\ M_I^*$$

$$\frac{3\sigma^2A^2/d^2}{\gamma_0(\sigma M_I^* + \nu + \alpha + d)} < k_3 < \frac{\gamma_0}{(M^* - M_I^*)^2}\min.\left\{\frac{1}{2\lambda_1^2}, \frac{(\sigma M_I^* + \nu + \alpha + d)}{3\lambda_2^2}\right\}$$

$$\frac{2k_3L(\lambda_1P_m + \lambda_2A/d)^2}{\gamma\gamma_0} < k_4 < \frac{2\gamma}{3\delta^2L}.$$

The stability conditions can than be obtained, as given in the statement of the theorem. Thus $\frac{dU_2}{dt}$ will be negative definite under the conditions (4.4), (4.5) and (4.6) as stated in the Theorem. Hence, the proof.