International Journal on Emerging Technologies **11**(3): 792-796(2020)

ISSN No. (Print): 0975-8364
ISSN No. (Online): 2249-3255

Mathematical Modelling of Impact of Vaccination in Controlling Japanese Encephalitis

Bapan Kalita[1] and Anuradha Devi[2]
[1]Assistant Professor, Department of Mathematics,
Royal School of Applied and Pure Sciences, Assam Royal Global University, 781035 (Assam), India.
[1]Research Scholar, Assam Science and Technology University (Assam), India.
[2]Professor, Department of of Mathematics,
Royal School of Applied and Pure Sciences, Assam Royal Global University, 781035 (Assam), India.

(Corresponding author: Bapan Kalita)
(Received 14 March 2020, Revised 08 May 2020, Accepted 11 May 2020)
(Published by Research Trend, Website: www.researchtrend.net)

ABSTRACT: Japanese Encephalitis is a deadly infectious epidemic disease. Its treatment is mainly based on prevention. Vaccination can protect human against Japanese Encephalitis in a very effective way. Vaccination against this epidemic had been approved by W.H.O. only from 2009. Here, a mathematical model is developed to trace the transmission pattern of the outbreak after the vaccination. Susceptible human are vaccinated before the warm cum rainy season as the disease turns around during this days. It is vector borne epidemic. So a vector borne infectious model along with vaccination as a control variable is planned in both deterministic and stochastic ambiences. Eigen values, Lyapunov functions are utilized to verify the stability of the model and found asymptotically stable. Reproduction number R_0 has been determined and found greater than one which supports the stability of the endemic equilibrium of the models. Impacts of different parameters associated to the reproduction number are studied in detail incorporating sensitivity analysis. Increase of the death rate of carrier mosquitoes, natural recovery rate of the infective, the natural death rate and the disease-induced death rate affect the reproduction number inversely, whereas the contact rate of carrier mosquitoes and human and the rate of vaccinated-infected rate are directly proportional to the reproduction number.

Keywords: Brownian motions, Control, Eigen values, Nonlinear, Sensitivity, Simulation, Stability, Stochastic.

Abbreviations: JPEN, Japanese Encephalitis.

I. INTRODUCTION

Every vector borne disease follows certain transmission behaviour. Japanese Encephalitis (JPEN) is also a vector borne disease. Flavivirus is the causal virus. This virus is a family member of the JPEN serogroup of the Flaviviridae family [1]. The virus exists in a zoonotic transmission cycle among aquatic birds, bats, mosquitoes, swine etc. belonging to the Ardeidae family [2]. Humans become infected when bitten by an infected mosquito and are a dead-end host because of low viremia, preventing the virus from being transmitted further. A Culex type mosquito employs itself in conveying the virus from this virus-reservoir to the susceptible human population [3]. The ambiences which assist viral survival are associated to both viral and host factors that let virus entry from the blood into the brain. Host factors are vehemently significant in JPEN susceptibility [4]. The deadly virus takes many lives within a short period of time. It is declared as epidemic disease. It may or may not show any symptoms at the early period of infection. But suddenly it starts showing headache, vomiting, fever, confusion and seizers along with inflammation of the brain. Disease incubation period is from five to fifteen days. The disease first appeared in the 1871 in Japan. In the year 1924, this became the epidemic and thrashed the Japanese society by taking 6000 lives [5-7]. As per WHO record, about 69,000 clinical cases were registered out of which 17,000 vigorous people have lost their lives [8].

Various researchers are researching on this epidemic from various fields. Researchers are also working on developing mathematical model of JPEN. Most of the researchers have done their works on deterministic model which study the robust behaviour of the disease. But in reality the outbreak doesn't understand the robust nature. Tapaswi *et al.,* developed a logistic differential equation model [9] considering density-dependent birth rate for mosquito population accepting the reservoir population fixed. Tapaswi and Ghosh (1999) formulated a populations model with a vector population [10]. Naresh and Pandey (2009) worked it with using differential equations and computational result [11]. Ghosh *et al.,* examined a SIS model [12] with colonization. Kalita and Devi studied control model of JPEN with the inclusion of media awareness [13].

At this juncture, the influence of vaccination is taken as the studying variable with a non-linear deterministic model. The same model is shifted to a stochastic model [13-16] using Geometric Brownian Motion [16-18]. A suitable Lyapunov function is constructed to determine the global stability. The stability of the model is also verified with the help of its phase portrait. For the stochastic stability again another Lyapunov function has been constructed with the help of two theorems as discussed in Mathematical Theory of Control Systems Design [19] and Stochastic Stability of differential equations [20].

II. MATERIALS AND METHODS

A. State variables

Mathematical model of infectious diseases can talk and shorten the work of studying the transmission behavior of a disease [21]. The infectious epidemic JPEN is disseminated by the carrier as described in the introduction. Being the carrier Culex mosquitoes carry the flavivirus from the pigs and wild birds and bite the susceptible human. Vaccination acts as the resistant of the JPEN. The mosquitoes keep on carrying the virus to the human until its death. Vaccinated human generally does not get infected. But sometimes vaccinated human may be infected because of expiry of the span of the power of vaccine. It does not transmit from human to human. Hence a mathematical model can be formulated for the infection cycle. To build the model, some state variables are taken as N(t) be the total population at time t, H(t) be the total susceptible population at time t, J(t) be the total JPEN infected population at time t, M(t) be the total mosquito population at time t who act as the carrier of the disease, $H_v(t)$ be the total vaccinated population at time t.

B. Mathematical model

Assumptions for the model: For a random region at a special time the following assumptions are presumed:

β be the rate of contact between susceptible human and carrier mosquitoes at time t,

γ be the rate of natural recovery at time t,

λ be the rate of infection of vaccinated human due to the bite of mosquitoes at time t,

d be the natural death rate at time t,

α be the death rate of carrier mosquitoes at time t,

a be the recovery rate of vaccinated-infected human at time t,

b be the vaccinated population from before time t,

g be the rate of vaccinated-infected human at time t,

f be the disease-induced death rate at time t.

Model: Based on the above assumptions and Karmack-McKendrick model [22-28] a nonlinear differential equation model is set up so as to understand the transmission trajectory as under:

$$\left.\begin{aligned} \frac{dH}{dt} &= -\beta HM + aH_vM + \gamma J - dH \\ \frac{dH_v}{dt} &= b - \lambda H_vM - dH_v \\ \frac{dJ}{dt} &= \beta HM + gH_vM - \gamma J - dJ - fJ \\ \frac{dM}{dt} &= -\alpha M \end{aligned}\right\} \quad (1)$$

From $\frac{dM}{dt} = -\alpha M$, we can write $M = ke^{-\alpha t}, k = e^C, c\ is$ integrating constant (2)

Using Eqns. (2), (1) can now be rewritten as

$$\left.\begin{aligned} \frac{dH}{dt} &= -\beta Hke^{-\alpha t} + aH_vke^{-\alpha t} + \gamma J - dH \\ \frac{dH_v}{dt} &= b - \lambda H_vke^{-\alpha t} - dH_v \\ \frac{dJ}{dt} &= \beta Hke^{-\alpha t} + gH_vke^{-\alpha t} - \gamma J - dJ - fJ \end{aligned}\right\} \quad (3)$$

Such that $\Theta = \{(H_0, H_{v_0}, J_0) \in R_+^3 : H + J + H_v = N, H > 0, J \geq 0, H_v \geq 0, N > 0, M \geq 0, \alpha > 0, \beta > 0, t \geq 0, a > 0, b > 0, d > 0, f > 0, g > 0, k \geq 0, \gamma > 0,\ H_v \leq N \leq \frac{b}{d}\}$.

C. Boundedness of the state variables

Lemma: Suppose $(H(t), H_v(t), J(t))$ be the solution of the system (3). If the initial condition (H_0, H_{v_0}, J_0) is in the probability space, then ∃ a unique positive solution $(H(t), H_v(t), J(t))\forall\ t \geq 0$ such that the solution will remain in the probability space with probability one. The solution (H, H_v, J) is defined in the interval $[0, \infty)$ and $\lim_{t \to \infty} Sup\ N(t) \leq \frac{b}{d}$, where $N(t) = H(t) + H_v(t), +J(t)$.

Proof: It is proposed that $(H_0, H_{v_0}, J_0) \in \Theta$, the probability space. Therefore the coefficients of the equations of (3) are Lipchitz continuous. Hence, for any given initial condition $(H_0, H_{v_0}, J_0) \in \Theta, \exists$ a unique local solution $(H(t), H_v, J(t))\forall\ t \in [0, T)$, where T is final time. It can be deduced that $H(t) + H_v(t) + J(t) \leq \frac{b}{d} \in [0, T)$.

Adding all the equations of (3), we get $\frac{dN(t)}{dt} \leq b + aH_vke^{-\alpha t} + gH_vke^{-\alpha t} - \lambda H_vke^{-\alpha t} - dH - dH_v - dJ - fJ = b + (a + g - \lambda)H_vke^{-\alpha t} - dN - fJ$

At no infection stage, $J = 0$ and $H_v = 0 \Rightarrow \frac{dN}{dt} \leq b - dN$

Integrating both sides, we get $N \leq \frac{b}{d}$

Therefore the solution $(H(t), H_v(t), J(t))$ is bounded within the interval $[0, T]$. This gives $N(t) \leq \frac{b}{d}\ \forall\ t \in [0, T)$. At $t \to \infty, e^{-\alpha t} \to 0 \Rightarrow \frac{dN(t)}{dt} \leq b - dH - dH_v - dJ - fJ = b - dN\ as\ (d + f) \to d$

Hence, $\lim_{t \to \infty} \text{Sup}\ N(t) \leq \frac{b}{d}$.

So for non-negative initial conditions $H_0 > 0, H_{v_0} \geq 0, J_0 \geq 0,\ \exists$ a non-negative solution defined in R and the set $\Theta = \{(\text{H}, H_v, \text{J})/\text{H} > 0, H_v \geq 0, J \geq 0 \text{ and } H + H_v + J = \frac{b}{d}\}$ is invariant.

D. Equilibrium point

Equating the equations of system (3) to zero as

$$-\beta Hke^{-\alpha t} + aH_vke^{-\alpha t} + \gamma J - dH = 0 \quad (4)$$

$$b - \lambda H_vke^{-\alpha t} - dH_v = 0 \quad (5)$$

$$\beta Hke^{-\alpha t} + gH_vke^{-\alpha t} - \gamma J - dJ - fJ = 0 \quad (6)$$

(i) Disease-free equilibrium is $B_0(H, H_v, J) = B_0(N, 0, 0)$

(ii) For endemic equilibrium:

From Eqn. (4), we have $H = \frac{aH_vke^{-\alpha t} + \gamma J}{\beta ke^{-\alpha t} + d}$

From Eqn. (5), we have $H_v = \frac{b}{\lambda ke^{-\alpha t} + d}$

And from Eqn. (6), we have $J = \frac{\beta kHe^{-\alpha t} + gkH_ve^{-\alpha t}}{f + d + \gamma}$

So the endemic equilibrium point is

$B_1(H^*, H_v^*, J^*) =$

$B_1\left(\frac{aH_vke^{-\alpha t} + \gamma J}{\beta ke^{-\alpha t} + d}, \frac{b}{\lambda ke^{-\alpha t} + d}, \frac{\beta khe^{-\alpha t} + gkH_ve^{-\alpha t}}{f + d + \gamma}\right)$

Endemic equilibrium exists if

$$\beta Hke^{-\alpha t} + gH_vke^{-\alpha t} - \gamma J - dJ - fJ > 0$$

$$\Rightarrow \frac{\beta kHe^{-\alpha t} + gkH_ve^{-\alpha t}}{(f + d + \gamma)J} > 1$$

Hence the Reproduction number is

$$R_0 = \frac{\beta kHe^{-\alpha t} + gkH_ve^{-\alpha t}}{(f + d + \gamma)J}$$

E. Stability of the deterministic model

Global Stability of the disease-free equilibrium:

Let us consider a Lyapunov function as $L(\theta) = \theta_1 H + \theta_2 H_v + \theta_3 J$ where $\theta_1, \theta_2\ and\ \theta_3$ are constants to be chosen in course of time.

And $\frac{dL}{dt} = \frac{\partial L}{\partial H}.\frac{dH}{dt} + \frac{\partial L}{\partial H_v}.\frac{dH_v}{dt} + \frac{\partial L}{\partial J}.\frac{dJ}{dt} = \theta_1(-\beta Hke^{-\alpha t} + aH_vke^{-\alpha t} + \gamma J - dH) + \theta_2(b - \lambda H_vke^{-\alpha t} - dH_v) + \theta_3(\beta Hke^{-\alpha t} + gH_vke^{-\alpha t} - \gamma J - dJ - fJ) = (\theta_1 - \theta_3)(-\beta Hke^{-\alpha t}) - \theta_1 dH - H_vke^{-\alpha t}(\theta_2\lambda - a\theta_1 - g\theta_2) - J\{\theta_3(f + d + \gamma) - \theta_1\gamma\} - \theta_2(dH_v - b)$

Disease-free equilibrium will be stable if

(i) $\theta_1 - \theta_3 > 0$

(ii) $\theta_2\lambda - a\theta_1 - g\theta_2 > 0$

(iii) $\theta_3(f + d + \gamma) - \theta_1\gamma > 0$

(iv) $dH_v - b > 0$

Global stability of the endemic equilibrium:

Let us consider a Lyapunov function as $L(\varphi) = \frac{1}{2}\varphi_1 H^2 + \frac{1}{2}\varphi_2 {H_v}^2 + \frac{1}{2}\varphi_3 J^2, where\ \varphi_1, \varphi_2\ and\ \varphi_3$ are constants to be chosen in course of time.

Now $\frac{dL}{dt} = \frac{\partial L}{\partial H}.\frac{dH}{dt} + \frac{\partial L}{\partial H_v}.\frac{dH_v}{dt} + \frac{\partial L}{\partial J}.\frac{dJ}{dt} = \varphi_1 H(-\beta Hke^{-\alpha t} + aH_vke^{-\alpha t} + \gamma J - dH) + \varphi_2 H_v(b - \lambda H_vke^{-\alpha t} - dH_v) + \varphi_3 J(\beta Hke^{-\alpha t} + gH_vke^{-\alpha t} - \gamma J - dJ - fJ) = -\beta Hke^{-\alpha t}(\varphi_1 H - \varphi_3 J) - H_vke^{-\alpha t}(\lambda\varphi_2 H_v - a\varphi_1 H - g\varphi_3 J) - J\{\varphi_3(\gamma + d + f) - \gamma\varphi_1 H\} - d\varphi_1 H^2 - \varphi_2 H_v(dH_v - b)$

Endemic equilibrium will be stable if

(i) $\varphi_1 H - \varphi_3 J > 0$

(ii) $\lambda\varphi_2 H_v - a\varphi_1 H - g\varphi_3 J > 0$

(iii) $\varphi_3(\gamma + d + f) - \gamma\varphi_1 H > 0$

(iv) $dH_v - b > 0$

F. Stochastic model

Deterministic model (3) can be converted to stochastic model by using Geometric Brownian Motion [15-17, 29-32] as follows:

$$\left.\begin{aligned} dH &= (-\beta Hke^{-\alpha t} + aH_vke^{-\alpha t} + \gamma J - dH)dt + \pi_1(H - H^*)d\theta_1 \\ dH_v &= (b - \lambda H_vke^{-\alpha t} - dH_v)dt + \pi_2(H_v - {H_v}^*)d\theta_2 \\ dJ &= (\beta Hke^{-\alpha t} + gkH_ve^{-\alpha t} - \gamma J - dJ - fJ)dt + \pi_3(J - J^*)d\theta_3 \end{aligned}\right\} \quad (7)$$

Eqn. (7) can be expressed as

$$dp(t) = f(p(t))dt + g(p(t))d\theta_r \quad (8)$$

Where $r = 1, 2, 3;\ p(t) = (p_1(t)\quad p_2(t)\quad p_3(t))^T;$

$$f(p(t)) = \begin{pmatrix} -\beta ke^{-\alpha t} - d & ake^{-\alpha t} & \gamma \\ 0 & -\lambda ke^{-\alpha t} - d & 0 \\ \beta ke^{-\alpha t} & gke^{-\alpha t} & -(\gamma + d + f) \end{pmatrix} p(t) \quad ;$$

$$g(p(t)) = \begin{pmatrix} \pi_1 p_1 & 0 & 0 \\ 0 & \pi_2 p_2 & 0 \\ 0 & 0 & \pi_3 p_3 \end{pmatrix}$$

G. Stochastic Stability

To understand the stochastic stability of system Eqn. (8), an operator L narrated in [15, 17, 19, 29, 35] is introduced as follows:

$$Lm(t,p) = \frac{\partial m(t,p)}{\partial t} + a^T(h)\frac{\partial m(t,p)}{\partial p} + \frac{1}{2}Tr\left[f^T(p)\frac{\partial^2 m(t,p)}{\partial p^2}f(s)\right] \quad (9)$$

Where $\frac{\partial m}{\partial p} = \begin{pmatrix} \frac{\partial m}{\partial p_1} \\ \frac{\partial m}{\partial p_2} \\ \frac{\partial m}{\partial p_3} \end{pmatrix}, \quad \frac{\partial^2 m}{\partial p^2} = \left(\frac{\partial^2 m}{\partial p_i p_j}\right)_{ij}$

Theorem 1: Assume a function $m(t,p) \in C^{1,2}(R \times R^n)$ exists, which satisfies the following inequalities as:

$$\left.\begin{aligned} M_1|p|^v \le m(t,p) \le M_2|p|^v \\ Lm(t,p) \le -M_3|p|^v, v > 0 \end{aligned}\right\}$$

The trivial solution of Eqn. (9) is u^{th} moment exponentially stable. Given that v = 2, the trivial solution is exponentially mean square stable and the break-even p = 0 is globally asymptotically stable [33-35].

Theorem 2: Consider ${\pi_1}^2 p_1 < 2[(\beta ke^{-\alpha t} + d)p_1 - \beta kp_3e^{-\alpha t}], {\pi_2}^2 p_2 < 2[\lambda kp_2e^{-\alpha t} - p_1 ake^{-\alpha t} - gkp_3e^{-\alpha t}], {\pi_3}^2 p_3 < 2[\gamma p_3 - (\gamma + d + f)p_1]$ hold, and then the zero solution of system (4.1) is mean square stable.

Proof: Assume a function $m(t,p) = \frac{1}{2}(D_1{p_1}^2 + D_2{p_2}^2 + D_3{p_3}^2)$

Here, $\frac{\partial m(t,p)}{\partial t} = 0$

$$f^T(p)\frac{\partial m(t,p)}{\partial p} = (p_1 \quad p_2 \quad p_3)\begin{pmatrix} -\beta ke^{-\alpha t} - d & ake^{-\alpha t} & \gamma \\ 0 & -\lambda ke^{-\alpha t} - d & 0 \\ \beta ke^{-\alpha t} & gke^{-\alpha t} & -(\gamma + d + f) \end{pmatrix} \begin{pmatrix} D_1p_1 \\ D_2p_2 \\ D_3p_3 \end{pmatrix}$$

$$= (-\beta kp_1e^{-\alpha t} - dp_1 + \beta kp_3e^{-\alpha t} \quad p_1ake^{-\alpha t} - \lambda kp_2e^{-\alpha t} + gkp_3e^{-\alpha t} \quad \gamma p_1 - (\gamma + d + f)p_3) \begin{pmatrix} D_1p_1 \\ D_2p_2 \\ D_3p_3 \end{pmatrix}$$

$$= -D_1(\beta kp_1e^{-\alpha t} + dp_1 - \beta kp_3e^{-\alpha t})p_1 - D_2(\lambda kp_2e^{-\alpha t} - gkp_3e^{-\alpha t} - p_1ake^{-\alpha t})p_2 - D_3\{-\gamma p_1 + (\gamma + d + f)p_3\}p_3$$

$$\frac{\delta^2 m}{\delta p^2} = \begin{pmatrix} \pi_1 & 0 & 0 \\ 0 & \pi_2 & 0 \\ 0 & 0 & \pi_3 \end{pmatrix}$$

$$g^T(t)\frac{\delta^2 m}{\delta p^2}g(p) = \begin{pmatrix} {\pi_1}^2 D_1{p_1}^2 & 0 & 0 \\ 0 & {\pi_2}^2 D_2{p_2}^2 & 0 \\ 0 & 0 & {\pi_3}^2 D_3{p_3}^2 \end{pmatrix}$$

Therefore $\frac{1}{2}Tr\left[g^T(t)\frac{\delta^2 m}{\delta p^2}g(p)\right] = \frac{1}{2}({\pi_1}^2 D_1{p_1}^2 + {\pi_2}^2 D_2{p_2}^2 + {\pi_3}^2 D_3{p_3}^2)$ and hence

$$L\,m(t,p) = -D_1\left(\beta kp_1e^{-\alpha t} + dp_1 - \beta kp_3e^{-\alpha t} - \frac{1}{2}{\pi_1}^2 p_1\right)p_1 - D_2\left(\lambda kp_2e^{-\alpha t} - p_1ake^{-\alpha t} - gkp_3e^{-\alpha t} - \frac{1}{2}{\pi_2}^2 p_2\right)p_2 - D_3(-\gamma p_1 + (\gamma + d + f)p_3 - \frac{1}{2}{\pi_3}^2 p_3)p_3.$$

This completes the proof of the theorem and hence it can be concluded that it follows asymptotic stability.

III. RESULTS AND DISCUSSION

A. Sensitivity Analysis

To understand the contribution of each of the parameters in the Reproduction number a sensitivity analysis [32] is conducted under

Sensitivity index of the system is given as:

$$S_E^{R_0} = \frac{\partial R_0}{\partial E}.\frac{E}{R_0}$$

The index table is shown Table 1.

Interpretation: Table 1 reveals the sensitivity analysis of different parameters on the Reproduction number. α and t have additive inverse effect on R_0. 10% increase of k increases 10% in the R_0. 20% increase of γ decreases R_0 by 31%. 2.5% increase of d decreases R_0 by 3%. 42% increase of f decreases R_0 by 65%. 2% g has 0.18% positive impact on R_0.

Table 1: Sensitivity index table.

Parameters	Sensitivity index	Sensitivity index values
α	$-\alpha$	-1
β	$\frac{\beta H}{\beta H + gH_v}$	0.998
k	1	1
t	$-t$	-1
H	$\frac{\beta H}{\beta H + gH_v}$	0.998
γ	$\frac{-\gamma}{\gamma + d + f}$	-0.3101
d	$\frac{-d}{\gamma + d + f}$	-0.0388
f	$\frac{-f}{\gamma + d + f}$	-0.6512
H_v	$\frac{gH_v}{\beta H + gH_v}$	0.0018
g	$\frac{gH_v}{\beta H + gH_v}$	0.0018
J	-1	-1

B. Numerical Simulation:

In this section, the ongoing models (2.1.3) and (6) are examined graphically through numerical simulation [35-37]. For this reason, Matlab 2016a software is being used. To use this technique some initial values of the basic variables and the parameters are listed as follows: If N = 500, H = 180, H_v = 10, J = 10, β = 0.62, k = 0.84, t = 0.01, γ = 0.2, d = 0.025, = 0.25, α = 0.21, a = 0.12, b = 300, g = 0.02, f = 0.42, then the endemic equilibrium is $B_1(5.5183,1278.9934,145.2946)$ and the reproduction number is $R_0 = 14.52946$. At this endemic equilibrium $R_0 > 1$ and hence system becomes stable here.

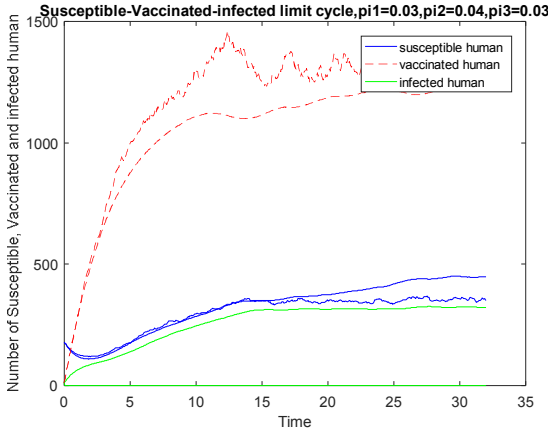

Fig. 1. Susceptible-Vaccinated-Infected limit cycle.

Interpretation: Fig. 1 shows the limit cycle of Susceptible, Vaccinated and Infective for different point of time. It shows the huge increase of vaccinated human population. Susceptible human population though at the beginning but it has started increasing. Infective population also not that much increased.

Interpretation: Fig. 2 depicts the limit cycle of infective population for various values of vaccinated population. Here it is clear that number of infective population varies for different values of g.

Interpretation: Fig. 3 unveils the phase plane of infected Vs vaccinated human population. It shows a stable behaviour at a particular point (H_v, J) as $(550,20)$ which means that whenever the vaccinated population is 550 and more then infective population become stagnant at 20.

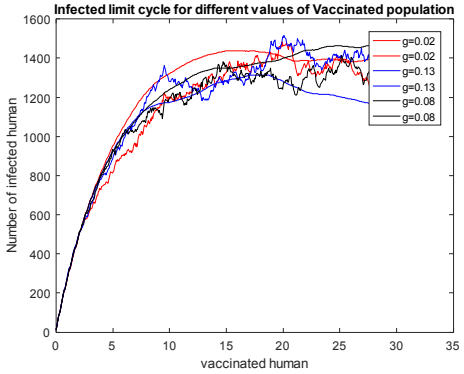

Fig. 2. Infected limit cycle for different values of *g*.

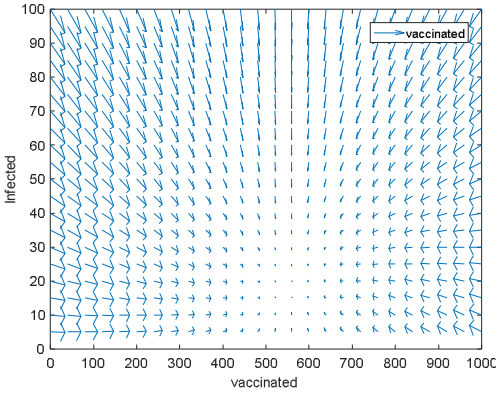

Fig. 3. Phase portrait of Vaccinated Vs Infected human.

IV. CONCLUSION

This paper is based on the control dynamics of JPEN. The study is carried out with the help of mathematical models. A robust nature is studied using deterministic model in model (3). The disease-free equilibrium shows stable behaviour under the conditions $(i)\theta_1 - \theta_3 > 0$, $(ii)\ \theta_2\lambda - a\theta_1 - g\theta_2 > 0$, $(iii)\theta_3(f + d + \gamma) - \theta_1\gamma > 0$ and $(iv)dH_v - b > 0$. The Endemic equilibrium $B_1\left(\frac{aH_vke^{-\alpha t}+\gamma J}{\beta ke^{-\alpha t}+d}, \frac{b}{\lambda ke^{-\alpha t}+d}, \frac{\beta khe^{-\alpha t}+gkH_ve^{-\alpha t}}{f+d+\gamma}\right)$ is asymptotically stable for $(i)\ \varphi_1 H - \varphi_3 J > 0$, $(ii)\ \lambda\varphi_2 H_v - a\varphi_1 H - g\varphi_3 J > 0$, $(iii)\ \varphi_3(\gamma + d + f) - \gamma\varphi_1 H > 0$ and $(iv)\ dH_v - b > 0$.

Eigen values of the Jacobian for the values taken in section (numerical results) are (-0.2686, -0.9211, -0.2346). It shows the stability of the model.

The stochastic model (8) is mean square stable under the conditions

$\pi_1{}^2 p_1 < 2[(\beta ke^{-\alpha t} + d)p_1 - \beta kp_3 e^{-\alpha t}], \pi_2{}^2 p_2 < 2[\lambda kp_2 e^{-\alpha t} - p_1 ake^{-\alpha t} - gkp_3 e^{-\alpha t}], \pi_3{}^2 p_3 < 2[\gamma p_3 - (\gamma + d + f)p_1]$.

The reproduction number obtained here is $R_0 = \frac{\beta khe^{-\alpha t}+gkH_ve^{-\alpha t}}{(f+d+\gamma)J} = 14.52946$ for the endemic equilibrium is $B_1(5.5183, 1278.9934, 145.2946)$. The phase portrait depicted in Fig. 3 shows the stability at the point $(H_v, J) = (550,20)$. Sensitivity analysis is done in Table 1. Increase of α, the death rate of carrier mosquitoes, γ, natural recovery rate of the infective, d, the natural death rate and f, the disease-induced death rate affect

the reproduction number inversely. β, the contact rate of carrier mosquitoes and human and g, the rate of vaccinated-infected rate are positively correlated with the reproduction number.

V. FUTURE SCOPE

The model can be verified with the actual data. From the sensitivity analysis, it is understood that carrier mosquitoes play a sensitive role in the reproduction number. So controlling of the mosquitoes can further be studied.

ACKNOWLEDGEMENTS

Authors are thankful to Prof. A. K. Mishra for his valuable suggestion and comment on the value of the reproduction number.

Conflict of Interest. There is no such interest.

REFERENCES

[1]. Misra, A. K., Sharma, A., & Shukla, J. B. (2011). Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases. *Mathematical and Computer Modelling, 53*, 1221-1228.

[2]. Bailey, N. T. (1975). Mathematical theory of infectious diseases and its applications. Geneva: WHO.

[3]. Burke, D. S., & Leake, C. J. (1988). Japanese Encephalitis., *In: Monath TP, ed.:The Arboviruses: Epidemiology and Ecology*, *3*, 63-92.

[4]. Saxena, S. K., Tiwari, S., Saxena, R., Mathur, A., & Nair, M. P. (2011). Japanese encephalitis: an emerging and spreading arbovirosis. *Flavivirus Encephalitis. Rijeka, Croatia: InTech*, 295-316.

[5]. Jacobson, J., Halstead, S. B., & Dubischar-Kastner, K. (2007). Japanese Encephalitis Vaccines. Saunders, Philadelphia, Pa, USA., 1410-1411.

[6]. Halstead, S. B, Jacobson J., & Dubischar-Kastner K. (2018). Japanese encephalitis. In *Greenbook: Immunisation against infectious diseases*. DHSSPS.

[7]. Campbell, G. L., Hills, S.L., Fischer, M., Jacobson, J. A., Hoke, C. H., & Hombach, J. M. (2011). Estimated global incidence of Japanese encephalitis: a systematic review. *Bulletin of World Health Organisation , 89*(10), 766-774.

[8]. Biswas, S. (1995). *Applied Stochastic Processes*. New Age International limited.

[9]. Tapaswi, P. K., Ghosh, A. K. and Mukhopadhayay, B. B., (1995). Transmission of Japanese Encephalitis in a 3-population model, *Ecological Modelling*, *83*, 295-309.

[10]. Tapaswi, P. K. & Ghosh, A. K. (1999). Dynamics of Japanese encephalitis—A stundy in mathematical epidemiology, *Mathematical Medicine and Biology: A Journal of the IMA*, *16*(1), 1-27.

[11]. Naresh, R., & Pandey, S. (2009). Modeling and Analysis of the Spread of Japanese Encephalitis with Environmental Effects, *Applications and Applied Mathematics: An International Journal (AAM), 4*(1), 155 – 175.

[12]. Ghosh, D., & Basu, A. (2009). Japanese encephalitis—a pathological and clinical perspective. *PLoS neglected tropical diseases*.

[13]. Kalita, B., & Devi, A. (2020). Control Model of Transmission of Japanese Encephalitis through Media Awareness. *International Journal of Advanced Science and Technology, 29*(5), 7645-7656.

[14]. Capasso, V. (1993). Mathematical Structures of Epidemic Systems. *Springer*.

[15]. Denu, D. (2017). Analysis of stochastic vector-host epidemic models.

[16]. Frederic, P., & Miller, A. F. (2010). *Geometric Brownian Motion.* VDM Publishing.

[17]. Mukherjee, D. (2003). Stability Analysis of a Stochastic Model for Prey-Predator System with Disease in the Prey, *Nonlinear Analysis: Modelling and Control*, *8*(2), 83–92.

[18]. Ghosh, M., Chandra, P., Sinha, P., & Shukla, J. B. (2004). Modeling the Spread of Carrier Dependent Infectious Diseases with Environmental Effect. *Applied Mathematics. Computation,152*, 385-402.

[19]. Afanasiev, V. N., Kolmanovskii, V., & Nosov**,** V. R. (1989). *Mathematical Theory of Control Systems Design*. Moscow: Springer.

[20]. Liying, X. (2006). *A Geometric Brownian Motion Oil Price Model.*: University of Texas at Austin.

[21]. Boogaard, H. F. P. V. D., Serafy, G. Y. E., Weerts, A. H., & Gerritsen, H. (2005). *Conversion of Deterministic Models into Stochastic Models*.

[22]. Anderson, H., & Britton, T. (2000). Stochastic Epidemic Models and Their Statistical Analysis. Springer.

[23]. Dubischar-Kastner, K., Halstead, S. B., & Jacobson, J. (2018). Japanese encephalitis. In *Greenbook: Immunisation against infectious diseases.* DHSSPS.

[24]. Gilden, D., Cohrs, R. J., Halstead**,** S. B., & Jacobson, J. (2008). Japanese Encephalitis Vaccines. Saunders, Philadelphia, Pa, USA.

[25]. Hee, J. Y. (2009). *A Geometric Brownian Motion for Commodity Prices.* Universiti Teknologi Malaysia.

[26]. Kapur, J. N. (1998). *Mathematical modelling.* New Age International (P) Limited, Publishers.

[27]. Kermack, W. O., & McKendrick, A. G. (1932). Contributions to the mathematical theory of epidemics, ii. *Proceedings of the Royal Society, Edinbarg, Section A. Mathematics, 138*, 55–83.

[28]. Kermack, W. O., & McKendrick, A. G. (1927). Contributions to the mathematical theory of epidemics, part-i. *Proceedings of the Royal Society of Edinburgh. Section A. Mathematics*, *115*, 700–721.

[29]. Khasminskii, R. (2012). Stochastic Stability of Differential Equations. Springer.

[30]. Leake, D. B. ((1988)). Japanese Encephalitis. In: Monath TP, ed.:The Arboviruses: *Epidemiology and Ecology, 3*, 63-92.

[31]. Mao, X. (2007). Stochastic Differential Equations and Applications. Woodhead Publishing.

[32]. Kamuhanda, A. E., Mary, W. & Shaibu, O. (2018). Mathematical Modelling andAnalysis of the Dynamics of Cholera. *Global Journal of Pure and Applied Mathematics*, *14*, 1259–1275.

[33]. Øksendal, B. (2000). *Stochastic Differential Equations*. New York: Springer-Verlag.

[34]. Anderson, H., & Britton, T. (2000). Stochastic Epidemie Models and Their Statistical Analysis.

[35]. Zhu, Q. (2014). Asymptotic Stability in the pth Moment for Stochastic Differential Equations with Levy Noise. *Journal of Mathematical Analysis and Applications*, *416*, 126-142.

[36]. Eustace, K. A., Osman, S., & Mary, W., Mathematical Modelling andAnalysis of the Dynamics of Cholera. *Global Journal of Pure andApplied Mathematics*, *14*, 1259–1275.

[37]. Melinik, R. (2015). Mathematical nad Computational Modeling.: Wiely.

How to cite this article: Kalita, B. and Devi, A. (2020). Mathematical Modelling of Impact of Vaccination in Controlling Japanese Encephalitis. *International Journal on Emerging Technologies*, *11*(3): 792–796.