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Dynamics of Vaccination Model with Holling Type II Functional Response

Sumit Kaur Bhatia* and Sudipa Chauhan
Amity Institute of Applied Sciences, Amity University, U.P 201-313, India
e-mail : sumit2212@gmail.com *and* sudipachauhan@gmail.com

Umama Nasir
Amity Institute of Applied Sciences, Amity University, U.P 201-313, India
e-mail : umamanasir.93@gmail.com

Abstract. We propose a mathematical model with Holling type II functional response, to study the dynamics of vaccination. In order to make our model more realistic, we have incorporated the recruitment of infected individuals as a continuous process. We have assumed that vaccination cannot be perfect and there is always a possibility of re-infection. We have obtained the existence of a disease free and endemic equilibrium point, when the recruitment of infective is not considered and also obtained the existence of at least one endemic equilibrium point when recruitment of infective is considered. We have proved that if $R_v < 1$, disease free equilibrium is locally asymptotically stable, which leads to the elimination of the disease from the population. The persistence of the model has also been established. Numerical simulations have been done to establish the results obtained.

1. Introduction

Mathematical modelling is increasingly used to study the behaviour of diseases. It studies the factors that play major role in the development of diseases; factors like transmission and recovery rate [16]. Modelling helps in understanding the dynamics of spreading of disease [2]. Many realistic models have been developed to study the transmission dynamics of infectious diseases [6]. In these models, the population is divided into classes based on various conditions and these classes are represented by ordinary differential equations [14]. The main focus in the study of an epidemic model is to analyse the steady states and their stability [3, 12].

* Corresponding Author.
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Many infectious diseases can be prevented by vaccination [10]. Disease can reoccur in some individuals due to short term immunity against re-infection. Hence, it is important to include the effects of immunity into realistic mathematical models [11]. The immunity period varies from disease to disease. Some diseases provide life long immunity while others provide short lived susceptibility. In measles, vaccines provides less immunity than natural immunity. The Hepatitis B vaccine gives 10–15 years of immunity and after that a booster is required for immunity to be effective. Also, influenza has a very short lived immunity [18]. Vaccination is a strategy to control infectious diseases and so it should also be included in order to make a model more realistic.

In the modeling of infectious diseases, the rate of infection plays a crucial role. Factors like density of population and life style affects the rate directly or indirectly. The rate can determine the spread or fall of an epidemic [17]. Bilinear incidence rates [9] and standard incidence rates $\frac{\beta SI}{N}$ are frequently used in epidemic models. These rates are derived from the law of mass action. This contact law is applicable to communicable diseases such as influenza, but not for sexually transmitted diseases. It is noted that standard incidence rates may be a good approximation if the population is large and the probability of contact is modest. After studying the spread of the cholera epidemic in the Italian town of Bari in 1973, Capasso and Serio [13] introduced a saturated incidence rate $g(I)S$ into epidemic models [15], where $g(I)$ tends to a fixed saturation level when I gets large, i.e., $g(I) = \frac{\beta I}{1+\alpha I}$ where βI measures the infection force of the disease and $1/(1 + \alpha I)$ measures the inhibition effect from the behavioral change of the susceptible individuals when their number increases, or from the crowding effect of the infective individuals [1, 4, 5]. This incidence rate seems more reasonable than the bilinear incidence rate βIS because it includes the behavioral change and crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters. It is more appropriate to consider a saturated incidence rate $g(I)S$ for the study of sexually transmitting disease. Hepatitis B is an illness caused by the hepatitis B virus (HBV) and transmitted via contact with infectious bodily fluids. It can be spread sexually, through the sharing of drug needles, in birth from an infected mother, through contact with open sores or wounds of an infected person, and through sharing of razors or toothbrushes with an infected person. Symptoms of hepatitis B infection include fever, abdominal pain, and jaundice, among others. Human papillomaviruses (HPV) belong to a large family of viruses, only some of which are sexually transmitted. Most people who contract HPV have no symptoms, and they quickly clear the virus from their bodies. However, in other people certain types of HPV cause genital warts. Other HPV types are the main cause of cervical cancer, and some are associated with anal, penile, mouth, and throat cancers.

Based on above factors, we have proposed a SVEIR model (Susceptible, Vaccinated, Exposed, Infected, Recovered), in Section 2. In order to make our model more realistic we have assumed that the recruitment of infected individuals is a continuous process, and that re-infection is possible. In Section 3, we obtain the existence of disease free and endemic equilibrium points. In this section we also find

the reproduction numbers and prove the local stability of a disease free equilibrium. In Section 4, we prove the persistence of the model, and in the final section we give the results of a numerical simulation.

2. Development of the Model

In this section, we present our mathematical model. Total population is divided into five parts: susceptible, vaccinated, infected, exposed and recovered i.e., $N(t) = S(t) + V(t) + E(t) + I(t) + R(t)$, at time t. The population increases at a constant rate π in the absence of disease and can be described by the following population model:

$$\frac{dS}{dt} = \pi \tag{2.1}$$

The model is developed with the following assumptions:

1. The susceptible population will increase by the loss of vaccine induced immunity and the recruitment of individual through previous vaccination. It will decrease from infection, vaccination and death rate. Susceptible population will get infected through contact of population with infected and exposed population i.e., βIS. Hence, the equation is:

$$\frac{dS}{dt} = (1-\epsilon)\pi - \frac{\beta IS}{1+hI} - \frac{\beta\eta ES}{1+hE} - \xi S - \mu S + \omega V \tag{2.2}$$

2. The vaccination of the susceptible population will increase the population of vaccinated individuals. The vaccination is assumed to be imperfect, so vaccinated individuals can acquire immunity to infection at a reduced rate $(1-\sigma)\frac{\beta IS}{1+hI}$. The vaccinated population is decreased by the infection and natural death rate μ. The vaccine is effective only if $\sigma = 1$ and ineffective if $\sigma = 0$. Thus, the equation of the vaccinated population is as follows:

$$\frac{dV}{dt} = \xi S - \frac{(1-\sigma)\beta IV}{1+hI} - \frac{(1-\sigma)\beta\eta EV}{1+hE} - (\mu+\omega)V \tag{2.3}$$

3. The population of exposed will increase with increase of the infected and exposed population. It decreases as the disease spreads and natural death rate increases. Thus, the equation is:

$$\frac{dE}{dt} = \frac{\beta IS}{1+hI} + \frac{\eta\beta ES}{1+hE} + \frac{(1-\sigma)\beta IV}{1+hI} + \frac{(1-\sigma)\beta\eta EV}{1+hE} - \kappa E - \mu E \tag{2.4}$$

4. The infected population increases with recruitment of infected individuals and due to rise of infection in exposed individuals. It decreases as the natural death and rate of recovery increases. The infected population is given as:

$$\frac{dI}{dt} = \epsilon\pi + \kappa E - (\gamma+\mu)I \tag{2.5}$$

5. The population of recovered individual decreases with increase of natural death and it increases as infected individuals recover:

$$\frac{dR}{dt} = \gamma I - \mu R \tag{2.6}$$

The biological meaning of the parameters in the above model is as follows:

Parameters	Meaning
π	Recruitment of Individuals
ξ	Rate of vaccination in susceptible individual
σ	Vaccine efficacy
μ	death rate
β	Probability of infection on contact with infected individual
κ	Transfer rate of exposed into infected
η	Modification parameter
γ	Rate of recovery
ω	Loss of immunity due to vaccine
ϵ	Recruitment of infected individual

Now, we shall prove some basic dynamical properties of the model:

Theorem 2.1. *The system is positively invariant and uniformly bounded in X with the following property:*

$$X = \left((S, V, E, I, R) : S, V, E, I, R > 0; S + V + E + I + R \leq \frac{\pi}{\mu} \right)$$

Proof. Adding all the above equations (2.2-2.6), we get,

$$\frac{dN}{dt} = \pi - \mu N \tag{2.7}$$

From equation (2.7) the feasible region is:

$$X = \left((S, V, E, I, R) : S, V, E, I, R > 0; S + v + E + I + R \leq \frac{\pi}{\mu} \right)$$

and it can be easily seen that X is positively invariant. □

In the following section, we will be studying the dynamics of two models: the first model is when there is no recruitment of infected individuals in the population i.e., $\epsilon = 0$, and the second model is when recruitment of infected individual in the population is considered, i.e., $\epsilon \neq 0$.

3. Stability Analysis

In this section, we will obtain the existence of disease free and endemic equilibrium points. We will calculate the reproduction number with the help of next generation method [7] and discuss the local stability of disease free equilibrium point based on reproduction number.

3.1. Equilibrium Points at ($\epsilon = 0$)

The mathematical model for this case is as follows:

$$\frac{dS}{dt} = \pi - \frac{\beta IS}{1+hI} - \frac{\beta\eta ES}{1+hE} - \xi S - \mu S + \omega V \tag{3.1}$$

$$\frac{dV}{dt} = \xi S - \frac{(1-\sigma)\beta IV}{1+hI} - \frac{(1-\sigma)\beta\eta EV}{1+hE} - (\mu+\omega)V \tag{3.2}$$

$$\frac{dE}{dt} = \frac{\beta IS}{1+hI} + \frac{\eta\beta ES}{1+hE} + \frac{(1-\sigma)\beta IV}{1+hI} + \frac{(1-\sigma)\beta\eta EV}{1+hE} - \kappa E - \mu E \tag{3.3}$$

$$\frac{dI}{dt} = \kappa E - (\gamma+\mu)I \tag{3.4}$$

$$\frac{dR}{dt} = \gamma I - \mu R \tag{3.5}$$

Equilibrium point for the above model can be obtained by solving the following equations:

$$\pi - \frac{\beta IS}{1+hI} - \frac{\beta\eta ES}{1+hE} - \xi S - \mu S + \omega V = 0 \tag{3.6}$$

$$\xi S - \frac{(1-\sigma)\beta IV}{1+hI} - \frac{(1-\sigma)\beta\eta EV}{1+hE} - (\mu+\omega)V = 0 \tag{3.7}$$

$$\frac{\beta IS}{1+hI} + \frac{\eta\beta ES}{1+hE} + \frac{(1-\sigma)\beta IV}{1+hI} + \frac{(1-\sigma)\beta\eta EV}{1+hE} - \kappa E - \mu E = 0 \tag{3.8}$$

$$\kappa E - (\gamma+\mu)I = 0 \tag{3.9}$$

$$\gamma I - \mu R = 0 \tag{3.10}$$

The disease free equilibrium (DFE) point can be obtained by taking $E = I = R = 0$ in the above equations and then using equations (3.6-3.7). We get the following value of V:

$$V = \frac{\pi\xi}{\mu(\mu+\xi+\omega)}, \tag{3.11}$$

and the following value of S:

$$S = \frac{\pi(\mu+\omega)}{\mu(\mu+\xi+\omega)}. \tag{3.12}$$

Thus the DFE is $\left(\frac{\pi(\mu+\omega)}{\mu(\mu+\xi+\omega)}, \frac{\pi\xi}{\mu(\mu+\xi+\omega)}, 0, 0, 0\right)$.

3.2. Reproduction Number

Let $x = (S, V, E, I)^t$. The system can be written as:

$$\frac{dx}{dt} = F(x) - V(x), \tag{3.13}$$

where

$$F = \begin{bmatrix} \beta S + (1-\sigma)\beta V & \eta\beta S + (1-\sigma)\beta\eta V \\ 0 & 0 \end{bmatrix}$$
$$V = \begin{bmatrix} 0 & (\kappa+\mu) \\ (\gamma+\mu) & -\kappa \end{bmatrix}$$
$$V^{-1} = \frac{1}{(\kappa+\mu)(\gamma+\mu)} \begin{bmatrix} \kappa & (\kappa+\mu) \\ (\gamma+\mu) & 0 \end{bmatrix}$$

FV^{-1} is the next generation matrix for the model and the spectral radius of matrix FV^{-1} is the reproduction number for the system. Thus,

$$R_v = FV^{-1} = \beta(S + (1-\sigma)V)(\frac{\kappa}{(\kappa+\mu)(\gamma+\mu)} + \frac{\eta}{(\kappa+\mu)}).$$

Now, we will study the local stability of the DEF point based on the reproduction number.

3.3. Local Stability

We will show that the disease free equilibrium is locally asymptotically stable if $R_v < 1$ and unstable if $R_v > 1$. The characteristic equation corresponding to disease free equilibrium is given by $|J - \lambda I| = 0$. Thus,

$$|J-\lambda I| = \begin{bmatrix} \pi - (\xi+\mu+\lambda) & \omega & -\beta\eta S & -\beta S \\ \xi & -(\mu+\omega) - \lambda & -(1-\sigma)\beta\eta V & -(1-\sigma)\beta V \\ 0 & 0 & \beta\eta S + (1-\sigma)\beta\eta V - (\kappa+\mu) - \lambda & \beta S + (1-\sigma)\beta V \\ 0 & 0 & \kappa & -(\gamma+\mu) - \lambda \end{bmatrix}$$

Therefore, the characteristic equation of disease free equilibrium point is given by the product of the following two quadratic equations.

$$\lambda^2 - \lambda((\pi-\mu) - (\xi+\mu+\omega)) - (\pi(\mu+\omega) - \mu(\xi+\mu+\omega)) = 0 \tag{3.14}$$

$$\begin{aligned} \lambda^2 &- \lambda(\beta\eta S + (1-\sigma)\beta\eta V - (\kappa+\mu) - (\gamma+\mu)) \\ &- ((\beta\eta S + (1-\sigma)\beta\eta V - (\kappa+\mu))(\gamma+\mu) + \beta\kappa S + (1-\sigma)\beta\kappa V) = 0 \end{aligned} \tag{3.15}$$

It can be easily seen that if $R_v < 1$ then the roots of (3.14) are negative. The other two roots of the characteristic equation are given by roots of equation (3.15). In

order to check the sign of roots of (3.15), we evaluate the sum and product of roots of (3.15). Indeed, the sum of the roots is:

$$\eta R_v\left(\frac{(\kappa+\mu)(\gamma+\mu)}{\kappa+\eta(\gamma+\mu)} - ((\kappa+\mu)+(\gamma+\mu)\right) \tag{3.16}$$

and the product of the roots is:

$$(\kappa+\mu)(\gamma+\mu)(1-R_v). \tag{3.17}$$

The roots of equation (3.15) are negative as their sum is negative and their product is positive if $R_v < 1$. Thus, we have the following theorem.

Theorem 3.3.1. *Disease free equilibrium is locally asymptotically stable if $R_v < 1$ and locally asymptotically unstable if $R_v > 1$.*

3.4 Endemic Equilibrium Point

Case 1: Endemic Equilibrium point when $\epsilon = 0$

From equation (3.8) we get:

$$I = \frac{\lambda\kappa R_v}{\beta(\kappa+\eta(\gamma+\mu))} \tag{3.18}$$

From equation (3.7) we obtain:

$$E = \frac{\lambda(\gamma+\mu)R_v}{\beta(\kappa+\eta(\gamma+\mu))} \tag{3.19}$$

$$\lambda = \left(\frac{I}{1+hI} + \frac{\eta E}{1+hE}\right) \tag{3.20}$$

$$\lambda = \beta\left(\frac{I(1+hE)+\eta E(1+hI)}{(1+hI)(1+hE)}\right) \tag{3.21}$$

Solving the above equation, we get,

$$\lambda h^2 IE - h\beta(IE+\eta IE) + \lambda(hE+hI) - \beta(I+\eta E) + \lambda = 0 \tag{3.22}$$

Above equation can also be written as:

$$X\lambda^2 + Y\lambda + Z = 0 \tag{3.23}$$

where,

$$X = h^2\kappa(\gamma+\mu)R_v \tag{3.24}$$

$$Y = h\beta R_v((\kappa+\gamma+\mu)(\kappa+\eta(\gamma+\mu)) - \kappa(\gamma+\mu)(1+\eta)R_v) \tag{3.25}$$

$$Z = \beta^2(\kappa+\eta(\gamma+\mu))^2(1-R_v) \tag{3.26}$$

If $R_v > 1$ then $X > 0$ and $Z < 0$. By Descrate's rule of signs, we can clearly see that there is precisely one endemic equilibrium when $R_v > 1$. On the other hand if $R_v < 1$ then $X > 0$, $Z > 0$. Consider,

$$\begin{aligned}&((\kappa+\gamma+\mu)(\kappa+\eta(\gamma+\mu))-\kappa(\gamma+\mu)(1+\eta)R_v)\\&>((\kappa+\gamma+\mu)(\kappa+\eta(\gamma+\mu))-\kappa(\gamma+\mu)(1+\eta))\\&>\kappa^2+(\gamma+\mu)^2>0\end{aligned}$$

Thus, $Y > 0$. Hence, endemic equilibrium does not exist if $R_v < 1$ and thus, we have the following theorem.

Theorem 3.4.1. *Unique endemic equilibrium exists if* $R_v > 1$.

Case 2: Endemic Equilibrium Point when $\epsilon \neq 0$

Now, we consider the case $\epsilon \neq 0$ i.e, recruitment of infected individuals is allowed at the rate ϵ.

$$I = \left(\frac{\lambda\kappa R_v}{\beta(\kappa+\eta(\gamma+\mu))} + \frac{\epsilon\pi}{(\gamma+\mu)}\right) \tag{3.27}$$

$$E = \frac{\lambda(\gamma+\mu)R_v}{\beta(\kappa+\eta(\gamma+\mu))} \tag{3.28}$$

$$\lambda = \beta\left(\frac{I}{(1+hI)} + \frac{\eta E}{(1+hE)}\right) \tag{3.29}$$

Solving this equation, we get,

$$\lambda(1+hI)(1+hE) = \beta\left(I(1+hI)+\eta E(1+hI)\right) \tag{3.30}$$

$$\lambda h^2 IE - \beta hIE(1+\eta) - \beta(I+\eta E) + h\lambda(I+E) + \lambda = 0 \tag{3.31}$$

Now substituting value of I and E from equation (3.27) and (3.28) to equation (3.30), we get:

$$P\lambda^3 + Q\lambda^2 + R\lambda + S = 0 \tag{3.32}$$

where,

$$P = h^2\kappa(\gamma+\mu)R_v^2 \tag{3.33}$$

$$Q = \beta hR_v(\epsilon\pi h(\kappa+\eta(\gamma+\mu))+(\kappa+\gamma+\mu)(\kappa+\eta(\gamma+\mu))-\kappa(\gamma+\mu)(1+\eta)R_v) \tag{3.34}$$

$$R = \beta^2(\kappa+\eta(\gamma+\mu))((\frac{h\epsilon\pi}{(\gamma+\mu)}+1-R_v)(\kappa+\eta(\gamma+\mu))+h\epsilon\pi(1+\eta)) \tag{3.35}$$

$$S = -\frac{\epsilon\pi\beta^3(\kappa + \eta(\gamma+\mu))^2}{(\gamma+\mu)} \tag{3.36}$$

We have $P > 0$ and $S < 0$. Clearly, we see that at least one endemic equilibrium always exists. Thus we have the following theorem.

Theorem 3.4.2. *For $\epsilon \neq 0$ endemic equilibrium always exists.*

4. Persistence

Let Y be a locally compact metric space with metric d and let F be a closed subset of Y with boundary ∂F and interior in F. Let π be a semi dynamical system defined on F. We say that π is persistent if for all $u \in \operatorname{int} F$, $\liminf_{t\to\infty} d(\pi(u,t)\partial F) > 0$, and that π is uniformly persistent if there is $\epsilon > 0$ such that for all $u \in \operatorname{int} F$, $\liminf_{t\to\infty} d(\pi(u,t), \partial F) > \epsilon$. A result about persistence is given by Fonda [8] in terms of repellers. A subset Θ of F is said to be a uniform repeller if there is there exists an $\epsilon > 0$ such that for each $u \in \frac{F}{\Theta}$, $\liminf_{t\to\infty} d(\pi(u,t), A) > \epsilon$. A semi flow on a closed subset F of a locally compact metric space is uniformly persistent if the boundary of F is repelling. The result of Fonda is given below:

Lemma 4.1. *Let Θ be a compact subset of Y such that $\frac{Y}{\Theta}$ is positively invariant. A necessary and sufficient condition for Θ to be a uniform repeller is that there exists a neighbourhood U of Θ and a continuous function $P : Y \to R^+$ satisfying:*

(1) *$P(u) = 0$ if and only if $u \in \Theta$.*

(2) *For all $u \in \frac{U}{\Theta}$ there is a T_u such that $P(\pi(u, T_u)) > P(u)$.*

For any $u_0 = (S(0), V(0), E(0), I(0)) \in \Omega$, there is a unique solution $\pi(u_0, t) = (S, V.E, I)(t; u_0)$ of which is defined in R^+ and satisfies $\pi(u_0, 0) = (S(0), V(0), E(0), I(0))$. Since Ω is a positively invariant set of the system, we have that $\pi(u_0, t)$ is in Ω for $t \in R^+$ and is a semi-dynamical system in Ω.

Theorem 4.1. *The set Θ is uniform repeller and π is uniformly persistent in Ω if $R_v > 1$.*

Proof. We see that Θ is positively invariant as $I(t) > 0$ for $t > 0$ if $I(0) > 0$. The set Θ is also a compact subset of Ω. Let $P : \Omega \to R^+$ be defined as, $P(S, V, E, I) = I$ and $U = \{(S, V, E, I) \in \Omega : P(S, V, E, I) < \xi\}$ for $t > 0$. From the first two equations of the system, we have,

$$\frac{dS(t)}{dt} \geq A - (\phi + \mu)S \tag{4.1}$$

$$\frac{dV(t)}{dt} \geq \phi S - \mu V \tag{4.2}$$

then $\liminf_{t\to\infty} S(t,\tilde{u}) \geq \frac{A}{\phi+\mu}$, $\liminf_{t\to\infty} V(t\tilde{u}) \geq \frac{\phi A}{\mu(\mu+\phi)} - \epsilon$ for $t > T$, where $\epsilon > 0$ is a sufficiently small constant. Define the auxiliary function:

$$L(t) = \frac{\kappa(1-\xi)}{\kappa+\mu}E(t) + I(t) \tag{4.3}$$

where $\xi(0 < \xi < 1)$ is a sufficiently small constant and since $R_v > 1$,therefore,

$$\frac{a\kappa(1-\xi)}{(\kappa+\mu)}\left(\beta\left((\frac{A}{\phi+\mu}-\epsilon\right)+\beta(1-\sigma)\left((\frac{\phi A}{\mu(\phi+\mu)}-\epsilon\right)\right)-(d-r+\delta)>0 \tag{4.4}$$

The derivative of L(t) along $\pi(u,t)$ is as follows:

$$\begin{aligned}\frac{dL}{dt} &= \frac{a\kappa(1-\xi)}{(\kappa+\mu)}\left(\beta(I+\eta E)\left(\frac{A}{\phi+\mu}-\epsilon\right)\right. \\ &\left.+\beta(I+\eta E)(1-\sigma)\left(\frac{\phi A}{\mu(\phi+\mu)}-\epsilon\right)+\kappa E-(d-r+\delta)I\right)\end{aligned} \tag{4.5}$$

$$\begin{aligned}\frac{dL}{dt} &\geq \frac{a\kappa(1-\xi)}{(\kappa+\mu)}\left(a\beta(I+\eta E)\left(\frac{A}{\phi+\mu}-\epsilon\right)\right. \\ &\left.+a\beta(I+\eta E)(1-\sigma)\left(\frac{\phi A}{\mu(\phi+\mu)}-\epsilon\right)\right)+\kappa\xi E-(d-r+\delta)\end{aligned} \tag{4.6}$$

$$\geq \frac{a\kappa(1-\xi)}{(\kappa+\mu)}\left(a\beta\left(\frac{A}{\phi+\mu}-\epsilon\right)+a\beta(1-\sigma)\left(\frac{\phi A}{\mu(\phi+\mu)}-\epsilon\right)-(d-r+\delta)\right)I(t) \tag{4.7}$$

$$+\frac{a\kappa(1-\xi)}{(\kappa+\mu)}\left(a\beta\eta\left(\frac{A}{\phi+\mu}-\epsilon\right)+a\beta\eta(1-\sigma)\left(\frac{\phi A}{\mu(\phi+\mu)}-\epsilon\right)+\kappa\xi\right)E(t) \tag{4.8}$$

Let

$$\tau = \min\left\{\left(a\beta\eta(\frac{A}{\phi+\mu}-\epsilon)+a\beta\eta(1-\sigma)(\frac{\phi A}{\mu(\phi+\mu)}-\epsilon)+\frac{a\kappa(1-\xi)}{(\kappa+\mu)}\kappa\xi\right),\right. \tag{4.9}$$

$$\left.\frac{\kappa(1-\xi)}{(\kappa+\mu)}\left(a\beta(\frac{A}{\phi+\mu}-\epsilon)+a\beta(1-\sigma)(\frac{\phi A}{\mu(\phi+\mu)}-\epsilon)-(d-r+\delta)\right)\right\} \tag{4.10}$$

then,

$$\frac{dL}{dt} \geq \tau L(t) \tag{4.11}$$

From equation (4.11) we can see that $L(t) \to \infty$ as $t \to \infty$. But, $L(t)$ is bounded on the set Ω, so the assumption above is not true. Thus, we have proved that for each $u \in \Omega$, with u belonging to suitably small neighbourhood of Θ, there is some T_u such that $P(\pi(u,T_u)) > P(u)$.

Therefore by the lemma, we conclude the theorem. □

5. Numerical Analysis

We have done numerical simulations and obtained the following results:

1. For parameters $\epsilon = 0, \omega = 0.01, \beta = 0.04, \eta = 0.03, \xi = 0.01, \sigma = 0.06, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.9$, the equilibrium point is $S = 13.2386, V = 0.2092, E = 10.2344, I = 4.7326$. Thus, trajectories are approaching to endemic equilibrium point (Figure 5).

2. For parameters $\epsilon = 0, \omega = 0.01, \beta = 0.004, \eta = 0.03, \xi = 0.01, \sigma = 0.06, \kappa = 0.0002, h = 0.04, \mu = 0.2, \pi = 0.0001, \gamma = 2$, the equilibrium point is $S = 0.0003, V = 0.0002, E = 0.0000, I = 0.0000$. Here, we can see that $R_v = 0.00000131065 < 1$ and trajectories are approaching to disease free equilibrium point (Figure 6).

3. For parameters $\epsilon = 0.95, \omega = 0.01, \beta = 0.05, \eta = 0.03, \xi = 0.01, \sigma = 0.6, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.1$ infected will always be greater than susceptible. The values are $S = 0.3589, V = 0.0085, E = 0.8017, I = 15.8868$ (Figure 1). For the same set of parameters and $\xi = 0.8, \sigma = 0.9$, we get, $S = 0.1718, V = 0.5266, E = 0.4955, I = 15.8664$ (see Figure 2), thus, we see that if the recruitment rate of the infected population is large, then infected will always be greater than susceptible and disease cannot be controlled even by increasing vaccination parameter to a large extent.

4. For parameters $\epsilon = 0.1, \omega = 0.01, \beta = 0.04, \eta = 0.03, \xi = 0.01, \sigma = 0.06, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.9$, the estimated values are $S = 19.4772, V = 0.6585, E = 1.9406, I = 0.4899$ (Figure 3). For the same parameters and $\xi = 0.8, \sigma = 0.9$, we get, $S = 4.5853, V = 17.1482, E = 0.5301, I = 0.4643$ (Figure 4). Hence, the susceptible population will always be greater than infective, i.e., if the recruitment rate of the infected population is small, then the disease can be controlled and it can be further reduced by increasing the vaccination parameter.

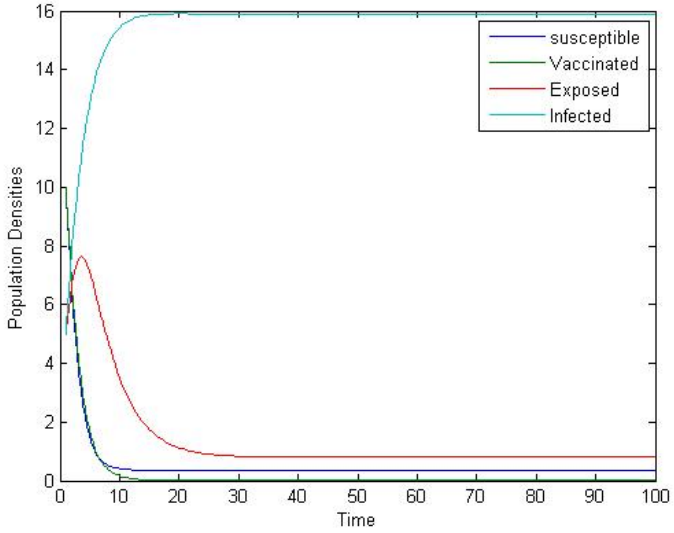

Figure 1: $\epsilon = 0.95, \omega = 0.01, \beta = 0.05, \eta = 0.03, \xi = 0.01, \sigma = 0.6, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.1$

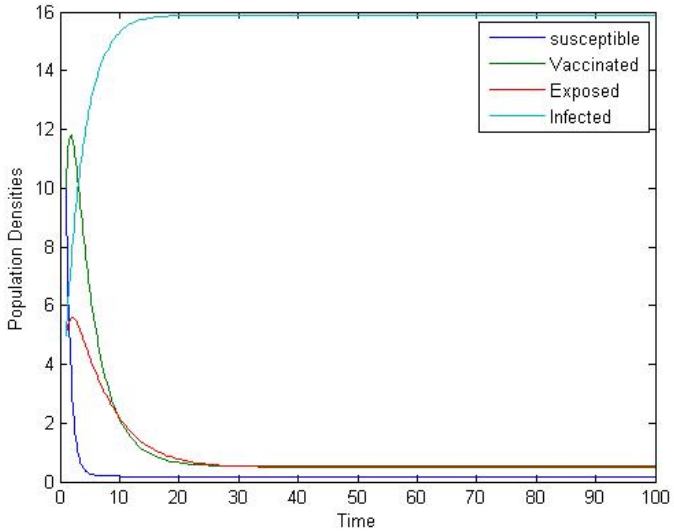

Figure 2: $\epsilon = 0.95, \omega = 0.01, \beta = 0.05, \eta = 0.03, \xi = 0.8, \sigma = 0.9, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.1$

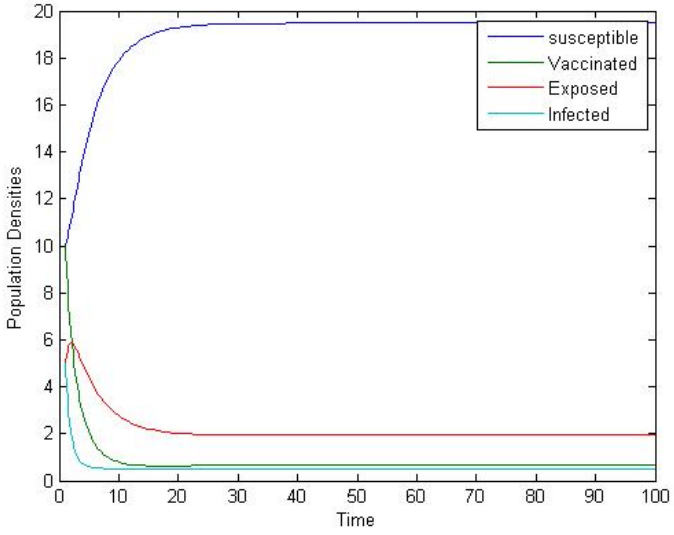

Figure 3: $\epsilon = 0.1, \omega = 0.01, \beta = 0.04, \eta = 0.03, \xi = 0.01, \sigma = 0.06, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.9$

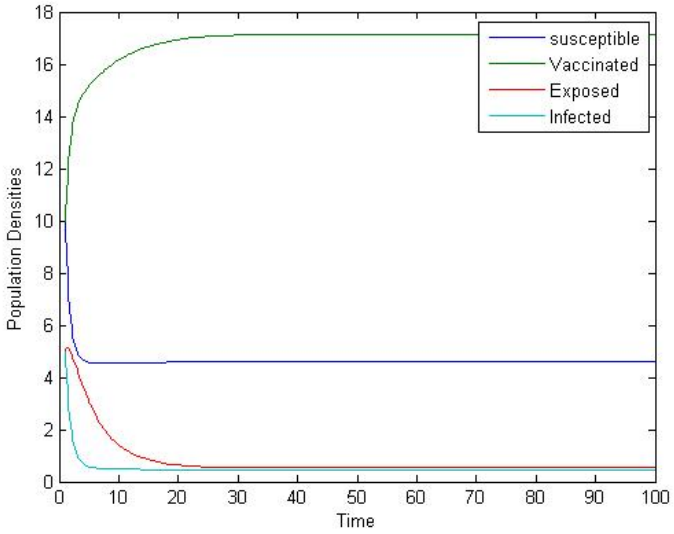

Figure 4: $\epsilon = 0.1, \omega = 0.01, \beta = 0.04, \eta = 0.03, \xi = 0.8, \sigma = 0.9, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.9$

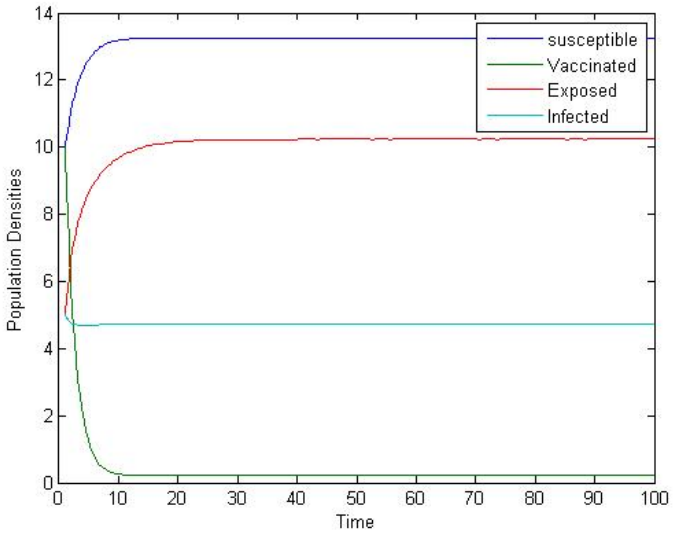

Figure 5: $\epsilon = 0, \omega = 0.01, \beta = 0.04, \eta = 0.03, \xi = 0.01, \sigma = 0.06, \kappa = 0.02, h = 0.04, \mu = 0.2, \pi = 5, \gamma = 0.9$

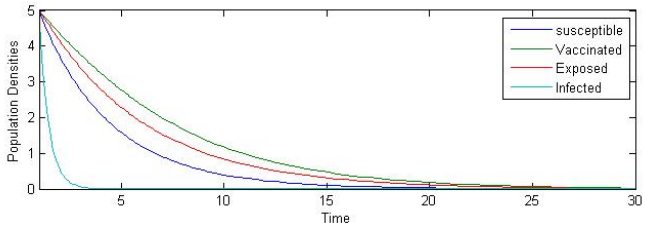

Figure 6: $\epsilon = 0, \omega = 0.01, \beta = 0.004, \eta = 0.03, \xi = 0.01, \sigma = 0.06, \kappa = 0.0002, h = 0.04, \mu = 0.2, \pi = 0.0001, \gamma = 2$

6. Conclusion

In this paper, we have studied an SVEIR model, and we have discussed the dynamics of the system. Two cases have been studied, one when the recruitment of infected is not considered i.e., $\epsilon = 0$ and another case when recruitment of infected is considered i.e., $\epsilon \neq 0$. For $\epsilon = 0$, we have obtained the existence of a disease free equilibrium point and endemic equilibrium point. Also, we have proved that disease free equilibrium point is locally stable for $R_v < 1$. Further, we have proved that if $R_v > 1$, unique endemic equilibrium point exists. For $\epsilon \neq 0$, we obtained the existence of at least one endemic equilibrium point. Uniform persistence of the system has been studied. We have further verified our results obtained with the help of numerical simulations for both the cases. It has been proved that recruitment rate of infective plays an important role in shaping the dynamics of the model, for higher value of recruitment rate of infective, disease can not be controlled even by increasing the vaccination parameter to a large extent whereas if the recruitment rate of infective is low then disease can be controlled and it can be further reduced by increasing the vaccination parameter.

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