Modeling Spread of Polio with the Role of Vaccination

Manju Agarwal and Archana S. Bhadauria

Department of Mathematics & Astronomy
Lucknow University
Lucknow-226007
Uttar Pradesh, India
manjuak@yahoo.com
archanasingh93@yahoo.co.in

Abstract

In this paper, we have proposed and analyzed a nonlinear mathematical model for the spread of Polio in a population with variable size structure including the role of vaccination. A threshold parameter, R, is found that completely determines the stability dynamics and outcome of the disease. It is found that if $R \leq 1$, the disease free equilibrium is stable and the disease dies out. However, if $R > 1$, there exists a unique endemic equilibrium that is locally asymptotically stable. Conditions for the persistence of the disease are determined by means of Fonda's theorem. Moreover, numerical simulation of the proposed model is also performed by using fourth order Runge - Kutta method. Numerically, it has been found that the system exhibits steady state bifurcation for some parameter values. It is concluded from our analysis that endemic level of infective population increases with the increase in rate of transmission of infection due to infective among susceptible class that further enhances because of transmission of infection due to latent hosts. A particular value of disease transmission coefficient $r\beta$ is found for which exposed and infective population dies out. It is found that periodic outbreak of the disease occurs when infection due to exposed and infective class occurs at the same rate. It is also observed from our analysis that although vaccination helps in eradicating polio by decreasing endemic equilibrium level yet careful administration of vaccination is desired because if vaccine is administered during incubation period, endemic equilibrium level increases and disease persists in the population.

1. Introduction

Polio (also called poliomyelitis) is a contagious, historically devastating disease that was virtually eliminated from the Western hemisphere in the second half of the 20th century. Although polio has plagued humans since ancient times, its most extensive outbreak occurred in the first half of the 1900s before the vaccination, created by Jonas Salk in 1952, became widely available in 1955. Poliomyelitis (polio) is a highly infectious disease caused by Poliovirus. It invades the nervous system, and can cause total paralysis in a matter of hours. It can strike at any age, but affects mainly children under three (over 50% of all cases). The virus enters the body through the mouth and multiplies in the intestine. Poliovirus mainly passes through person-to-person contact. Initial symptoms are fever, fatigue, headache, vomiting, and stiffness in the neck and pain in the limbs. However, immune and or partially immune adults and children can still be infected with poliovirus and carry the virus for long enough to take the virus from one country to another, infecting close contacts and contaminating sanitation systems. There is no cure for polio; it can only be prevented through immunization. Polio vaccine, given multiple times, usually protects a child for life.

Full immunization will markedly reduce an individual's risk of developing paralytic polio. Full immunization will protect most people (almost 99%). Immunization has enabled the global eradication of Polio. Although wild polio has not been found in the United States for more than two decades, it is still present in parts of Africa and Asia. On 23 April 2010, the World Health Organization announced the confirmation of wild poliovirus serotype 1 (WPV1) in seven samples from children with Acute Flaccid Paralysis in Tajikistan, in the context of a multi-district cluster starting in December 2009. As of 28 April, 32 of 171 reported cases were laboratory confirmed and most closely related to virus from Uttar Pradesh, India. This outbreak demonstrates the high risk that still exists for importation of wild poliovirus into polio-free regions, WHO Country Office Tajikistan (2010).

In most of the models, it is usually assumed that infection spread in susceptible population due to infective population only but some diseases like polio are contagious during incubation period so interaction of exposed population with susceptible population also has its role in the spread of disease. We, therefore, have considered interaction between susceptible and exposed population in our model. The spread of communicable diseases not only depend on interaction of population but also on the immunity of the individual. The immunity to the specific disease in the individuals can be artificially developed with the help of vaccination. But if vaccine is administered during incubation period (as symptoms of polio are not visible during incubation period) it can be produce hazardous effects also.

For example, Polio vaccine IPV can precipitate paralysis in a patient who is already in incubation period of polio. Hence, IPV or any other injection should be avoided in an unimmunized sick child with fever especially during season of polio epidemics. Keeping this in mind, we have studied the effect of vaccine administration in not only in susceptible class but also in exposed class during incubation period. Many research workers, Garfinkel (2003) and Tebbens et al. (2005) have done the modeling and analysis of polio disease, Tebbens et al. developed a dynamic disease transmission model aimed at simulating the spread of poliovirus infection after reintroduction of virus into a wild polio-free population. Michele S. Garfinkel and Daniel

Sarewitz (2003) briefly review the circumstances leading to the possibility of eradication of poliovirus. On account of proven efficiency of vaccination in controlling infectious diseases, various modeling studies have been made by Anderson (1983), C.P. Farrington (2003), Gumel (2003), Kribs-Zaleta (2000), Shulgin (1998), Struchiner (1989). In particular, Shulgin et al (1998) studied a simple *SIR* epidemic model with pulse vaccination and showed that pulse vaccination leads to epidemic eradication if certain conditions regarding the magnitude of vaccination proportion and on the period of pulses are satisfied. Kribs-Zaleta and Velasco-Hernandez (2000) presented a simple two-dimensional *SIS* model with vaccination exhibiting backward bifurcation. Farrington (2003) analyzed the impact of vaccination program on the transmission potential of the infection in large population and derived relation between vaccine efficacy against transmission, vaccine coverage and reproduction numbers. Gumel and Moghadas (2003) proposed a model for the dynamics of an infectious disease in the presence of a preventive vaccine considering non-linear incidence rate and found the optimal vaccine coverage threshold needed for disease control and eradication.

In this paper, we have analyzed an epidemic model on Polio with vaccination to determine the impact of vaccination when it is administered in susceptible and exposed population. The other main objective of our paper is to find out the way an interaction between susceptible and exposed class results in the spread of Polio. We have found two parameters R_0 and R_1 where, R_0 is the total number of secondary infections contributed by a single infective host during the infective period in an entirely susceptible population and R_1 is total number of secondary infections contributed by a single host in exposed class during the latent period. The sum of these two threshold values, denoted by R, is a sharp threshold value that completely determines the stability dynamics and the outcome of the disease. It is found that if $R \leq 1$, the disease free equilibrium is stable and the disease dies out, however if $R > 1$, there exists a unique endemic equilibrium which is locally asymptotically stable.

Persistence of the disease is shown by Fonda's theorem. This paper is organized as follows: In section 2, we outline our model. In section 3, boundedness and positivity of solutions of the system is studied. Section 4 gives a review of equilibrium points and basic reproduction ratio. Sections 5 deals with stability analysis of equilibrium points. In section 6, we have proved persistence of endemic equilibria followed by numerical simulation in section 7. Lastly, a short discussion is presented in section 8.

2.   Model Formulation

We propose a nonlinear mathematical model to study the spread of polio in a population system. The model stratifies the total population into several classes: Susceptible to infection (S), exposed (E), infective (I) and vaccinated class (V). The model assumes that an individual enters the total population N at the rate A and leaves at the rate μ, where μ is natural death rate of the population. The human population is considered to be varying in size because of the considerable degree of fatality due to the disease. As polio incubation period can be as short as 4 days or as long as 35 days, latent host is assumed to form an additional exposed class (E). After incubation period, exposed population enters into infective class (I). Susceptible population is assumed to contract disease on interaction with both exposed and infective population. Interaction between human population is modeled using bilinear interaction (law of mass action) as it is done by

Shulgin (1998), Assaad (1984), Anderson (1979). We assume that a certain fraction of individuals in susceptible class are vaccinated immediately at birth and become permanently immune, since polio vaccine is highly effective in producing immunity to poliovirus and protection from paralytic poliomyelitis. As symptoms are not shown by exposed population during incubation period it is assumed that they too are vaccinated however, numbers of individuals receiving vaccine of polio without knowing that they have polio are very less in number. Keeping in view of the above discussion, the dynamics of the transmission of polio is assumed to be governed by the following system of nonlinear ordinary differential equations:

$$\frac{dS}{dt} = A - \beta SI - r\beta SE - (\mu + v)S,$$

$$\frac{dE}{dt} = \beta SI + r\beta SE - (b + \mu + v_1)E, \tag{2.1}$$

$$\frac{dI}{dt} = (b + v_1)E - (\mu + \alpha)I,$$

$$\frac{dV}{dt} = vS - \mu V,$$

with initial conditions: $S(0) = S_0 > 0$; $E(0) = E_0 \geq 0$; $I(0) = I_0 \geq 0$; $V(0) = V_0 \geq 0$.

Here, A is the constant immigration rate of human population; β is the probability per unit time of infection transmission due to infective population and $r\beta$ $(0 < r \leq 1)$ is the probability per unit time of infection transmission due to exposed population. Here, r represents the reduction in the transmission of infection by exposed class since in case of polio although exposed population transmit infection to others yet transmission of infection by them is less than those in infective class. v is the proportion of new recruits in the susceptible class moving to vaccinated class; v_1 is the rate at which exposed population are vaccinated because of the absence of any symptoms of polio in them; b is the rate at which exposed population move to the infective class; μ and α are the natural and disease induced death rates of human population, respectively.

3.   Positivity and Boundedness of Solutions

3.1. Positivity of Solutions

Model (2.1) describes a human population, and, therefore it is very important to prove that all quantities (susceptible, exposed, infective and vaccinated population) will be positive for all times. Positivity implies that the system persists i.e. the population survives.

From the model system, we note that

$$\frac{dS}{dt}\bigg|_{S=0} = A > 0,$$

$$\frac{dE}{dt}\bigg|_{E=0} = \beta SI \geq 0,$$

$$\frac{dI}{dt}\bigg|_{I=0} = (b + v_1)E \geq 0,$$

$$\frac{dV}{dt}\bigg|_{V=0} = vS > 0.$$

Thus, the vector field is directed inwards on each bounding plane of $(R_0^+)^4$ so all solution trajectories initiating in the interior of $(R_0^+)^4$ will always remain positive.

3.2. Boundedness of Solutions

Boundedness may be interpreted as a natural restriction to grow because of limited resources. To establish the biological validity of the model system, we have to show that the solutions of system (2.1) are bounded for this we find the region of attraction in the following theorem.

Lemma 3.2.1.: All the solutions of (2.1) starting in the positive orthant $(R_0^+)^4$ either approaches, enter or remain in the subset of $(R_0^+)^4$ defined by

$$\Omega = \left\{ (S, E, I, V) \in R_+^4 : 0 < S + E + I + V \leq N \leq \frac{A}{\mu} \right\},$$

where $(R_0^+)^4$ denote the non-negative cone of R^4 including its lower dimensional faces.

Proof:

Let us consider the following function:

$$N(t) = S(t) + E(t) + I(t) + V(t).$$

From system (2.1) we get

$$\frac{dN}{dt} \le A - \mu N$$

Then, by usual comparison theorem, we get the following expression as $t \to \infty$,

$$\lim Sup_{t \to \infty} N \le \frac{A}{\mu} \ ,$$

and hence, $S(t) + E(t) + I(t) + V(t) \le \frac{A}{\mu}.$

Thus, it suffices to consider solutions in the region Ω. Solutions of the initial value problem starting in Ω and defined by (2.1) exist and are unique on a maximal interval, Hale (1980). Since solutions remain bounded in the positively invariant region Ω, the maximal interval is well posed both mathematically and epidemiologically.

4. Equilibrium Points

The two equilibrium points of the system (2.1) are as follows:

Disease-free equilibrium point: $E_0(S_0, E_0, I_0, V_0)$, where

$$S_0 = \frac{A}{\mu + v}, \ E_0 = 0, \ I_0 = 0, \ V_0 = \frac{v}{\mu} S_0.$$

The existence of disease-free equilibrium point E_1 is obvious. This equilibrium implies that if there is no exposed and infective population in the system under consideration then the equilibrium value of susceptible population will reach the value $\frac{A}{\mu + v}$ and vaccinated population will remain at its equilibrium $V_0 = \frac{v}{\mu} S_0.$

Endemic equilibrium point: $E^*(S^*, E^*, I^*, V^*)$, where

$$S^* = \frac{A}{(\mu + v)} \frac{1}{R}, \quad E^* = \frac{A}{(b + \mu + v_1)} \left(1 - \frac{1}{R} \right), \tag{4.1}$$

$$I^* = \frac{(b + v_1)}{(\mu + \alpha)} E^*, \quad V^* = \frac{v}{\mu} S^*$$

with

$$R = \frac{A\beta(b + v_1 + r(\mu + \alpha))}{(\mu + v)(b + \mu + v_1)(\mu + \alpha)} . \tag{4.2}$$

This equilibrium implies that if the exposed and infective population is present in the system, then the infection will be transmitted to the human population susceptible to polio. The equilibrium values of different variables will be given by S^*, E^*, I^*, V^*. These equilibrium values are explicitly given by equations (4.1).

Remark: It is observed that E^* and I^* exist if $R > 1$, where $R = \frac{A\beta(b + v_1 + r(\mu + \alpha))}{(\mu + v)(b + \mu + v_1)(\mu + \alpha)}$, is called the basic reproduction ratio, Anderson (1979) or the contact number, Hethcote (1976). To get the better understanding of the basic reproduction ratio R we can write

$$R = R_0 + R_1$$

$$= \frac{A\beta(b + v_1)}{(\mu + v)(b + \mu + v_1)(\mu + \alpha)} + \frac{r\beta A}{(\mu + v)((b + \mu + v_1))}.$$

where $R_0 = \beta \frac{A}{(\mu + v)} \frac{1}{(\mu + \alpha)} \frac{(b + v_1)}{(b + \mu + v_1)}$, this expression can be deciphered as follows: Over the mean infectious period $\frac{1}{(\mu + \alpha)}$, a single infective produces $\beta \frac{A}{(\mu + v)} \frac{1}{(\mu + \alpha)}$ latent hosts in a total susceptible population of size $\frac{A}{(\mu + v)}$ through direct contact, a fraction $\frac{(b + v_1)}{(b + \mu + v_1)}$ of which survive latency and become infectious. Similarly, rewrite R_1 as $R_1 = r\beta \frac{A}{(\mu + v)} \frac{1}{(b + \mu + v_1)}$ which can be interpreted as follows: $r\beta \frac{A}{(\mu + v)}$ latent hosts are produced through direct contact of a single contagious latent host from susceptible population of which $\frac{1}{(b + \mu + v_1)}$ survive latency and become infectious.

5. Stability Analysis of Equilibrium Points

To discuss the local stability of system (2.1), we compute the variational matrix of system (2.1). The signs of the real parts of the eigenvalues of the variational matrix evaluated at a given equilibria determine its stability. The entries of general variational matrix are given by differentiating the right hand side of system (2.1) with respect to S, E, I, V i.e.,

$$V(S,E,I,V) = \begin{bmatrix} -\beta I - r\beta E - (\mu+v) & -r\beta S & -\beta S & 0 \\ \beta I + r\beta E & r\beta S - (b+\mu+v_1) & \beta S & 0 \\ 0 & b+v_1 & -(\mu+\alpha) & 0 \\ v & 0 & 0 & -\mu \end{bmatrix}.$$

We denote the variational matrix corresponding to E_0 by $V(E_0)$ and corresponding to E^* by $V(E^*)$.

5.1. Local Stability Analysis of Disease-Free Equilibrium

To explore local stability disease-free equilibria we compute variational matrix of E_0. The variational matrix of disease-free equilibrium point is given as

$$V(E_0) = \begin{bmatrix} -(\mu+v) & -r\beta A/(\mu+v) & -\beta A/(\mu+v) & 0 \\ 0 & r\beta A/(\mu+v) - (b+\mu+v_1) & \beta A/(\mu+v) & 0 \\ 0 & b+v_1 & -(\mu+\alpha) & 0 \\ v & 0 & 0 & -\mu \end{bmatrix}.$$

Eigenvalues of $V(E_0)$ are given by $\lambda = -\mu, \quad \lambda = -(\mu+v)$ and the quadratic equation

$$\lambda^2 + \lambda\left((\mu+\alpha) + (b+\mu+v_1) - r\beta\frac{A}{(\mu+v)} \right)$$
$$+ \left\{ (\mu+\alpha)\left((b+\mu+v_1) - r\beta\frac{A}{(\mu+v)} \right) - \beta\frac{A(b+v_1)}{(\mu+v)} \right\} = 0$$
$$\lambda^2 + \lambda\left((\mu+\alpha) + (b+\mu+v_1)(1-R_1) \right) + (\mu+\alpha)(b+\mu+v_1)(1-R) = 0 \quad . \tag{5.1}$$

From (5.1) we observe that $V(E_0)$ has negative roots if $R_1 < 1$ and $R < 1$. But $R = R_0 + R_1$, so sufficient condition for $V(E_0)$ to have negative real part is $R < 1$. Thus disease-free equilibria E_0 is locally asymptotically stable if basic reproduction number $R < 1$ which further implies that disease will not spread and epidemic will die out.

5.2. Local Stability Analysis of Endemic Equilibria

To determine local stability of endemic equilibria we write variational matrix of E^* as given below:

$$V(E^*) = \begin{bmatrix} -\dfrac{A}{S^*} & -r\beta S^* & -\beta S^* & 0 \\ \beta I^* + r\beta E^* & -\dfrac{\beta S^* I^*}{E^*} & \beta S^* & 0 \\ 0 & b+v_1 & -(\mu+\alpha) & 0 \\ v & 0 & 0 & -\mu \end{bmatrix}.$$

The eigenvalues of $V(E^*)$ are given by $\lambda = -\mu$ and cubic equation

$$\lambda^3 + A_1 \lambda^2 + A_2 \lambda + A_3 = 0 \qquad\qquad (5.2)$$

where

$$A_1 = \frac{A}{S^*} + \mu + \alpha + \frac{\beta S^* I^*}{E^*} > 0,$$

$$A_2 = \frac{A}{S^*}(\mu+\alpha) + \frac{A\beta I^*}{E^*} + r\beta S^*(\beta I^* + r\beta E^*) > 0,$$

$$A_3 = \beta S^*(\beta I^* + r\beta E^*)\{r(\mu+\alpha) + b + v_1\} > 0, .$$

Also

$$A_1 A_2 - A_3$$

$$= \frac{A}{S^*}\left(\mu + \alpha + \frac{\beta S^* I^*}{E^*}\right)\left(\frac{A}{S^*} + \mu + \alpha + \frac{\beta S^* I^*}{E^*}\right)$$

$$+ \beta S^*(\beta I^* + r\beta E^*)\left(r\frac{A}{S^*} + \frac{r\beta S^* I^*}{E^*} - (b+v_1)\right).$$

It can be seen that all of the coefficients $A_i > 0, i = 1,2,3$ are positive. The Routh–Hurwitz criteria that are necessary and sufficient for the local asymptotic stability of the endemic equilibrium are that the coefficients are positive and the Hurwitz determinants H_i are all positive, Lancaster (1969). For a third degree polynomial, the Hurwitz determinant criteria are $H_1 = A_1 > 0$, $H_2 = A_3 > 0$ and $H_3 = A_1 A_2 - A_3 > 0$. By Routh-Hurwitz criteria, roots of equation (5.2) have negative real parts if $A_1 A_2 - A_3 > 0$. Note that the first term in

$H_3 = A_1 A_2 - A_3$ is always positive, but the third factor in second term has one negative term

$-(b + v_1)$, so that E^* may not be positive for all parameter values. Thus, the Routh–Hurwitz criteria may not be satisfied and bifurcation can occur for some parameter values.

A bifurcation occurs when a small smooth change made to the parameter values (the bifurcation parameters) of a system causes a sudden 'qualitative' or topological change in its behavior, Blanchard (2006). A bifurcation occurs when a parameter change causes the stability of an equilibrium (or fixed point) to change. In continuous systems, this corresponds to the real part of an eigenvalue of an equilibrium passing through zero. If we consider the continuous dynamical system described by the ODE

$$\dot{x} = f(x, \lambda) \quad f : \Re^n \times \Re \to \Re^n,$$

a bifurcation occurs at (x_0, λ_0) if the corresponding Jacobian matrix has an eigenvalue with zero real part. If the eigenvalue is equal to zero, the bifurcation is a steady state bifurcation, but if the eigenvalue is non-zero but purely imaginary, this is a Hopf bifurcation. We shall further show existence of steady state bifurcations in our model system (2.1) in section 7.

6. Persistence

Persistence means the survival of all populations in future time. Mathematically, persistence means that the strictly positive solution does not have omega limit points on the boundary of the non-negative cone. From the epidemiological point of view, persistence or permanence means endemicity of disease. There are two approaches to obtain persistence result, Waltman (1990). One is based on the analysis of the behavior of the dynamical system near the boundary and the other uses a Liapunov like function. Fonda (1988) follows the later one and gives a result about persistence in terms of repellers. A subset Z of F is said to be a uniform repeller if there is a $\eta > 0$ such that for each $x \in F \setminus Z, \liminf_{t \to \infty} d(\pi(x,t), Z) > \eta$. We recall some definitions in order to introduce the abstract theorem for proving the persistence. Let X is a locally compact metric space with metric d and F be a closed subset of a set X with boundary ∂F and interior $\operatorname{int} F$. Let π be a semi-dynamical system defined on F. π is said to be persistent if for all $x \in \operatorname{int} F, \liminf_{t \to \infty} d(\pi(x,t), \partial F) > 0$ and that π is uniformly persistent if there is $\xi > 0$ such that for all $x \in \operatorname{int} F, \liminf_{t \to \infty} d(\pi(x,t), \partial F) > \xi$. The result of Fonda given in Margheri (2003) is the following:

Lemma 6.1: Let Z be a compact subset of X such that $X \setminus Z$ is positively invariant. A necessary and sufficient condition for Z to be uniform repeller is that there exist a neighborhood U of Z and a continuous function $P : X \to R_0^+$ satisfying

   i. $P(x) = 0$ if and only if $x \in Z$.
   ii.   For all $x \in U \setminus Z$ there is a $T_x > 0$ such that $P(\pi(x, T_x)) > P(x)$.

Note: The result is given for a semi-dynamical system defined in a closed subset of a locally compact metric space X. However, nonlinear differential systems in epidemiology are sufficiently smooth and hence uniqueness and global existence in epidemiology are guaranteed

in some suitable domain. In this way the solutions of the system will give rise to a semi-dynamical system.

For any $x_0 = (S_0, E_0, I_0, V_0)$, there is a unique solution $\pi(x_0, t) = (S, E, I, V)(t, x_0)$ of (2.1) which is defined in $(R_0^+)^4$, lies in $(R_0^+)^4$ and satisfies $\pi(x_0, 0) = (S_0, E_0, I_0, V_0)$. In order to conclude that π is uniformly persistent , we restrict our study to the set $\Omega = \left\{ (S, E, I, V) \in (R_0^+)^4 : S + E + I + V \leq \dfrac{A}{\mu} \right\}$. If $x_0 \in \Omega$ then $\pi(x_0, t) \in \Omega$, for every $t \in (R_0^+)^4$ and hence the restriction of π to Ω, that we still denote by π, is a semi-dynamical system in Ω.

The following lemma would help us to prove that $Z = \{(S, E, I, V) \in \Omega : I = 0\}$ is a uniform repeller which implies that the semi-dynamical system π is uniformly persistent.

We are now in position to prove that $Z = \{(S, E, I, V) \in \Omega : I = 0\}$ is a uniform repeller, from which follows that the semi-dynamical system π is uniformly persistent in Ω.

Theorem 6.1: If $R_0 > 1$ and $R_1 > 1$, then the set Z is uniform repeller and hence π is uniformly persistent in Ω.

Proof:

Let us consider a compact subset Z of Ω. $\Omega \setminus Z$ is positively invariant as it is easy to show that $I(0) > 0 \Rightarrow I(t) > 0, \forall t > 0$.

Let $P : \Omega \to (R_0^+)^4$ be defined by $P(S, E, I, V) = I$ and let

$$\Sigma = \big((S, E, I, V) \in \Omega : P(S, E, I, V) < \rho \big),$$

with ρ sufficiently small so that $\dfrac{R_0}{\beta\rho + \phi} > 1$, $\dfrac{R_1}{(\beta\rho + \phi)} > 1$ where $\phi = r\beta\dfrac{A}{\mu} + \mu + v$. Then by ii of Lemma 6.1, we have to prove that for all $x \in \Sigma \setminus Z$ there is a $T_x > 0$ such that $P(\pi(x, T_x)) > P(x)$.

Let us assume by contradiction that there is a $x \in \Sigma$ such that for each $t > 0$ we have $P(\pi(x, t)) < P(x) < \rho$, which implies that $I(t, x) < \rho$ for each $t > 0$. From lemma 3.2.1 and first equation of system (2.1) we have $\dfrac{dS}{dt} \geq A - (\beta\rho + r\beta\dfrac{A}{\mu} + \mu + v)S$, then $\lim_{t \to \infty} \inf S(t) \geq \dfrac{A}{\beta\rho + \phi}$. So there is sufficiently large $T > 0$ such that $S(t) \geq \dfrac{A}{\beta\rho + \phi}$ for $t \geq T$.

In addition, consider the auxiliary function $\xi(t) = (1 - \rho^*)E(t) + I(t)$.

Differentiating $\xi(t)$ with respect to t, we obtain

$$\frac{d\xi(t)}{dt} \geq (b+v_1)E + (b+\mu+v_1)\left\{(\mu+v)(1-\rho^*)\frac{R_1}{(\beta\rho+\phi)} - 1\right\}E(t) +$$

$$(\mu+\alpha)\left\{\frac{(1-\rho^*)(b+\mu+v_1)(\mu+v)}{(b+v_1)}\frac{R_0}{(\beta\rho+\phi)} - 1\right\}I(t),$$

where $\rho^* > 0$ is a sufficiently small constant for which $(\mu+v)(1-\rho^*)\dfrac{R_1}{(\beta\rho+\phi)} > 1$ and

$$\frac{(1-\rho^*)(b+\mu+v_1)(\mu+v)}{(b+v_1)}\frac{R_0}{(\beta\rho+\phi)} > 1 .$$

$$\text{Let } \delta = \min\left[\begin{array}{c} \dfrac{(b+v_1)}{(1-\rho^*)}, \dfrac{(b+\mu+v_1)}{(1-\rho^*)}\left\{(\mu+v)(1-\rho^*)\dfrac{R_1}{(\beta\rho+\phi)} - 1\right\}, \\[2em] (\mu+\alpha)\left\{\dfrac{(1-\rho^*)(b+\mu+v_1)(\mu+v)}{(b+v_1)}\dfrac{R_0}{(\beta\rho+\phi)} - 1\right\} \end{array} \right] > 0 . \text{ Then, } \frac{d\xi(t)}{dt} \geq \delta\xi(t).$$

The last inequality implies that $\xi(t) \to \infty$ as $t \to \infty.$ However, $\xi(t)$ is bounded on the set Ω, so the assumption above is not true.

We have proved that for each $x \in \Omega \setminus Z$, with x belonging to a suitably small neighborhood Σ of Z, there is some $t = T_x$ such that $P(\pi(x,T_x)) > P(x)$. Thus, Fonda's theorem allows us to reach the conclusion.

7. Numerical Simulation

To substantiate the above analytical findings, the model is studied numerically. The system of differential equation is integrated using fourth order Runge-Kutta method under the following set of compatible parameters, which satisfy the stability conditions.

$$A = 1000, \ \beta = 0.002, \ r = 0.5, \ \mu = 0.5, \ v = 0.6, \ v_1 = 0.001, \ b = 0.9, \ \alpha = 0.6 \tag{7.1}$$

The endemic equilibrium values for this set of parameters are:

$$S^* = 531.04, \quad E^* = 296.82, \quad I^* = 243.12, \quad V^* = 637.25$$

Eigenvalues corresponding to endemic equilibria $E^*(S^*, E^*, I^*, V^*)$ are obtained as:

$$-1.1, \; -1.9262 \pm 0.6426i, \; -0.0006.$$

Since all the eigenvalues corresponding to E^* are negative, therefore E^* is locally asymptotically stable. Further, to illustrate the global stability of the equilibrium point graphically, numerical simulation is performed for different initial starts and the result is displayed in figure 1 for $I-S$ plane. All the trajectories starting from different initial starts, reach the endemic equilibrium E^*.

$$S(0) = 500 \qquad E(0) = 200 \qquad I(0) = 160 \qquad V(0) = 600$$

$$S(0) = 650 \qquad E(0) = 200 \qquad I(0) = 160 \qquad V(0) = 600$$

$$S(0) = 650 \qquad E(0) = 200 \qquad I(0) = 220 \qquad V(0) = 600$$

$$S(0) = 500 \qquad E(0) = 200 \qquad I(0) = 220 \qquad V(0) = 600$$

From the solution curve obtained in the figure, we can infer that the system is globally stable about the interior (endemic) equilibrium point $E^*(S^*, E^*, I^*, V^*)$.

The main objective of our paper is to find out the way an interaction between susceptible and exposed class results in the rapid spread of polio. For this, we have plotted figures 2 and 3, which show variation of infective and exposed population with time for different values of transmission coefficient of infection due to exposed class $r\beta$ and it is found that both the infective and exposed population increase with the increase in $r\beta$ and then become constant at their equilibrium level. Further, it is found from the graph that infective and exposed population vanish for a particular value of $r\beta$.

From this we conclude that epidemic does not spread when interaction between susceptible and exposed population is less than a particular value. In figures, 4-5 it is found that both the exposed and infective population show periodic behavior when rate of infection due to infective and exposed class are equal. This implies periodic outbreak of the disease occurs when infection due to exposed and infective class occurs at the same rate. This favours the fact that some of the countries showed a high endemicity, with periodic peaks of even higher incidence and those with small number of annual cases, experience pronounced outbreaks every few years, Assaad (1984). Figure 6 shows the variation of population in all the classes with immigration. It is found that susceptible population first increases with time and then reaches its equilibrium position. Since due to immigration, susceptible population increases continuously, therefore, infection becomes more endemic and always persists in the population.

Figure 7 shows that if rate of vaccination given to population during exposed period v_1, is raised then infective population increases. It may be because of occurrence of paralysis in a patient who is already in incubation period of polio, as can occur during polio epidemics. In order to find the variation in infective population level with basic reproduction number we have plotted figure 8. Figure 8 shows that endemic infective level first raises with the increase in basic reproduction ratio and then becomes constant. This implies that the infectious invasion will become pandemic if the threshold is greater than 1.

Thus, we see that the basic reproductive number plays an important role for the prevalence of a disease. Further to show variation of infective class size for different growth rate of human population, we have considered different values of A in figure 8 and found that increase in growth rate of human population causes an increase in endemic level of infective population implying that the disease spread fast if population is dense and endemic level of infective population remains high.

Further, we have found that A, β, μ, v and α are the bifurcation parameters. It is observed that for $A = 584$, $\beta = 0.001102$, $\mu = 0.78$, $v = 1.4$ and $\alpha = 3$ respectively and other parameters remaining same as (7.1), one of the eigenvalues corresponding to variational matrix of the endemic equilibrium point becomes zero. Hence, steady state bifurcations occur in our system for these bifurcation values of the parameters.

8. Conclusion

In this paper, an epidemic model on Polio with the effect of vaccination is considered. Two threshold parameters R_0 and R_1 corresponding to interaction of susceptible with infective and exposed class respectively are found. The sum of these two threshold values, denoted by R, is proved to be a sharp threshold value which completely determines the stability dynamics and the outcome of the disease. It is found that if $R \leq 1$, the disease free equilibrium is stable and the disease dies out, however if $R > 1$, there exists a unique endemic equilibrium which is locally asymptotically stable. Persistence of the disease is shown for $R_0 > 1$ and $R_1 > 1$. To substantiate the analytical findings, the model is studied numerically and for which the system of differential equation is integrated using fourth order Runge-Kutta method, which satisfy the stability conditions. Further, to illustrate the global stability of the equilibrium point, numerical simulation is performed for different initial starts and the results are displayed. It is found that the system exhibits steady state bifurcation for some parameter values. It is concluded that endemic level of infective population increases with the increase in rate of transmission of infection from infective to susceptible class, which can further be enhanced if transmission of infection occurs from latent hosts during incubation period. However, for a particular value of disease transmission coefficient $r\beta$, exposed and infective population die out. Variation of infective equilibrium size of the population with basic reproduction number is determined and it is found that endemic infective level first rises with the increase in basic reproduction ratio and then becomes constant.

It is found that although vaccination helps in eradicating polio by decreasing endemic equilibrium level yet careful administration of vaccination is desired because if vaccine is administered in an individual during incubation period of polio, endemic equilibrium level increases and disease spreads faster than usual pace. Like any other IM injection, it can precipitate paralysis in a patient who is already in incubation period of polio, as can occur during polio epidemics. Hence, IPV or any other IM injection should be avoided in an unimmunized sick child with fever especially during season of polio epidemics. It is found that the periodic outbreak of the disease occurs when infection due to exposed and infective class occurs at the same rate. It is pointed out from our study that population movement also contributes to the spread of disease; constant migration in human population makes the disease endemic. The

movement of infected people from areas where polio is still endemic to areas where the disease has been eradicated led to resurgence of the disease. It is further observed that transmission of infection due to population in exposed state of polio plays an important role in the spread of Polio and hence some measures must be adopted to trace the population in exposed class as it is very difficult to trace them out because of absence of symptoms of disease in them.

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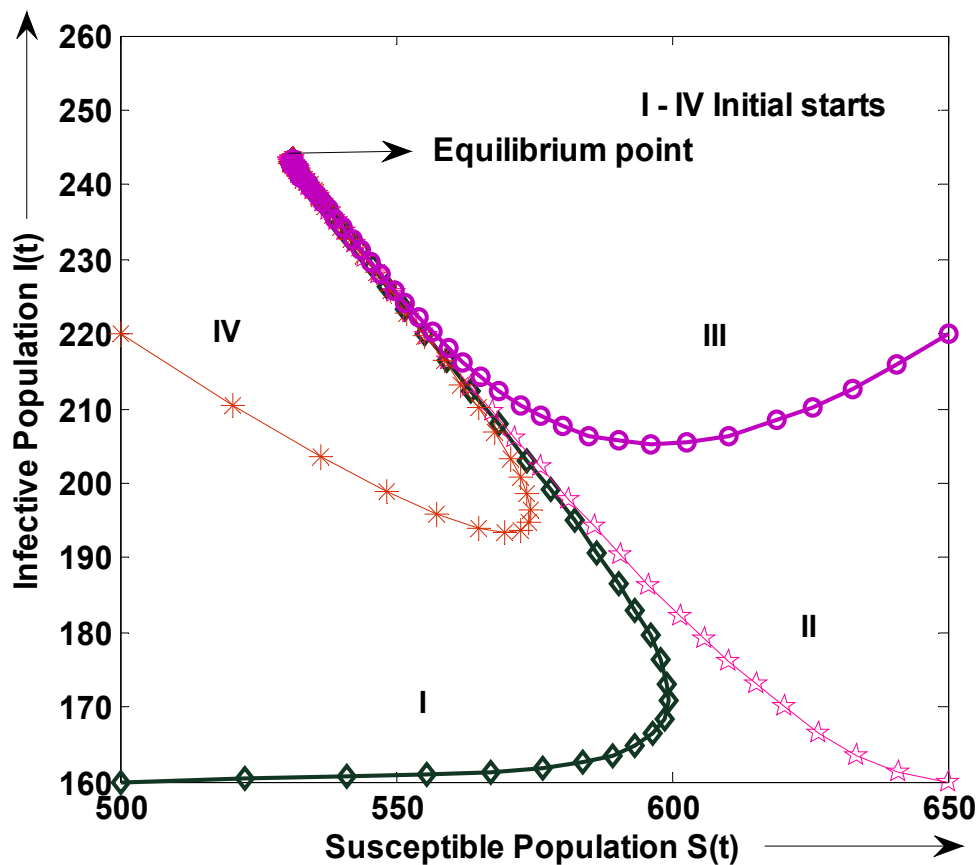
Figure 1. Variation of infective population with susceptible population for different initial starts
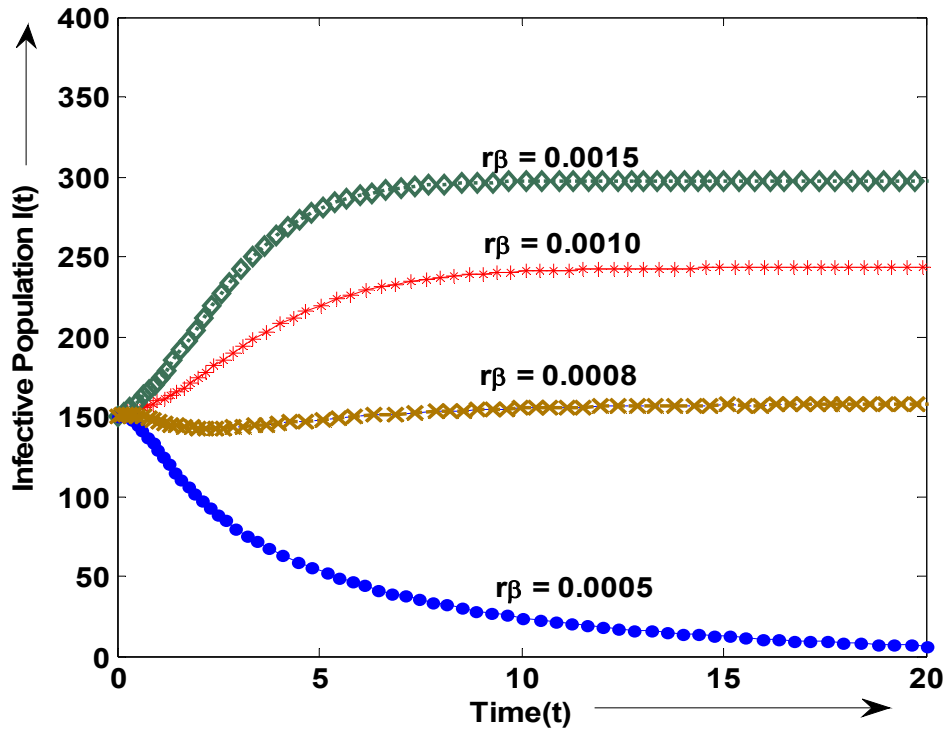
Figure 2. Variation of infective population with time for different values of transmission coefficients of infection due to exposed class.
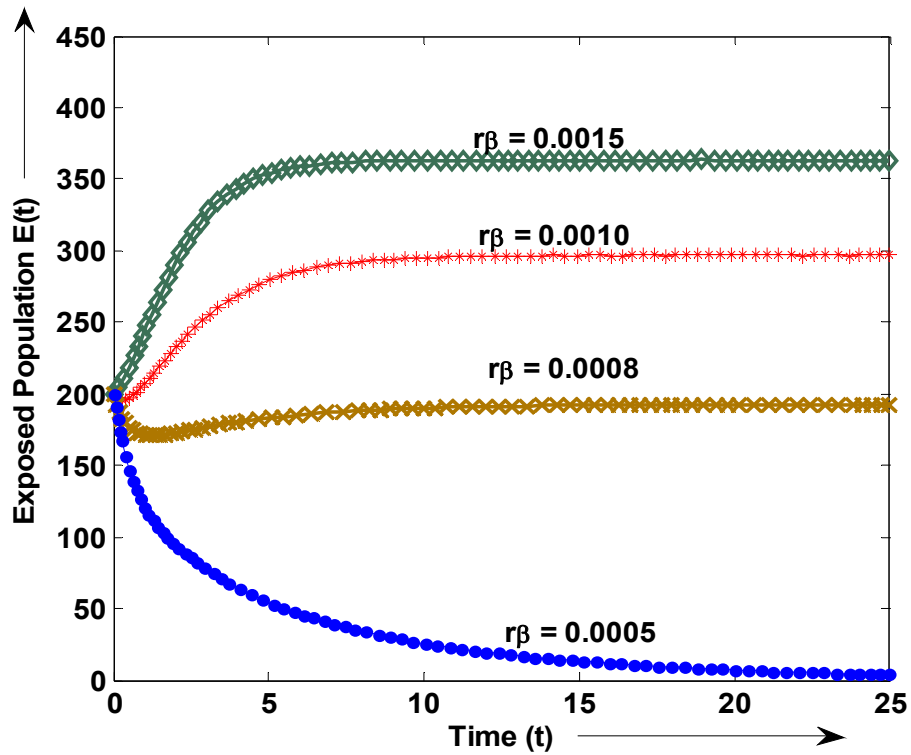

Figure 3. Variation of exposed population with time for different values of transmission coefficients of infection due to exposed class.
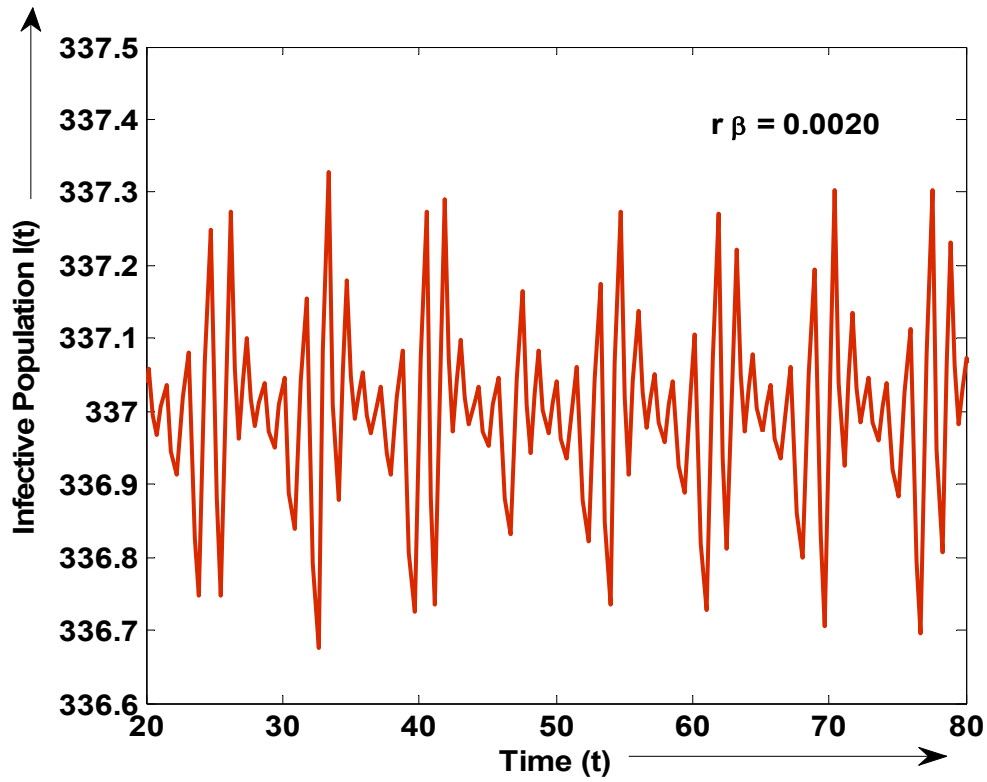
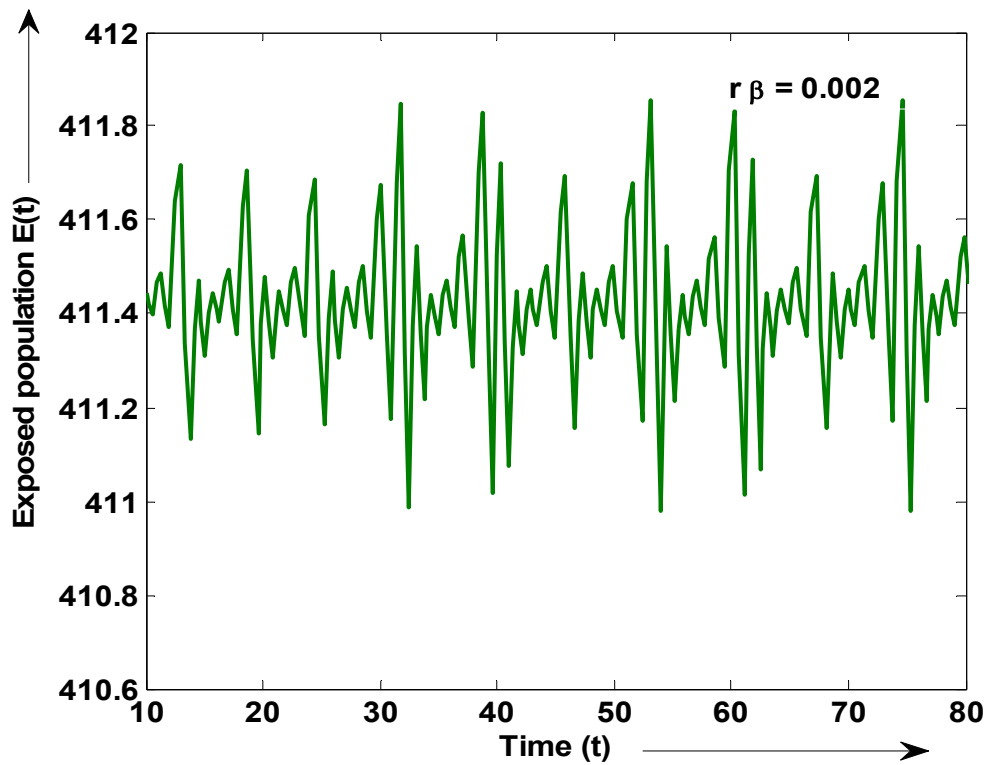**Figure 4.** Variation of infective population with time for $r\beta = 0.002$

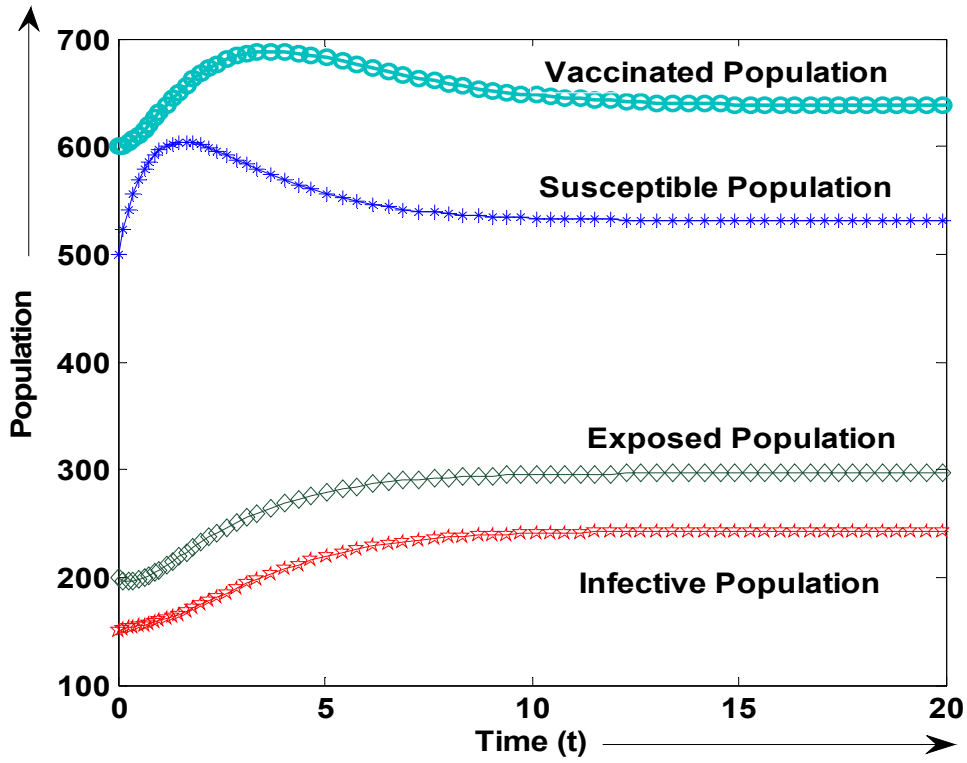**Figure 5.**   Variation of exposed population with time for $r\beta = 0.002$

Figure 6. Variation of population in different classes with immigration
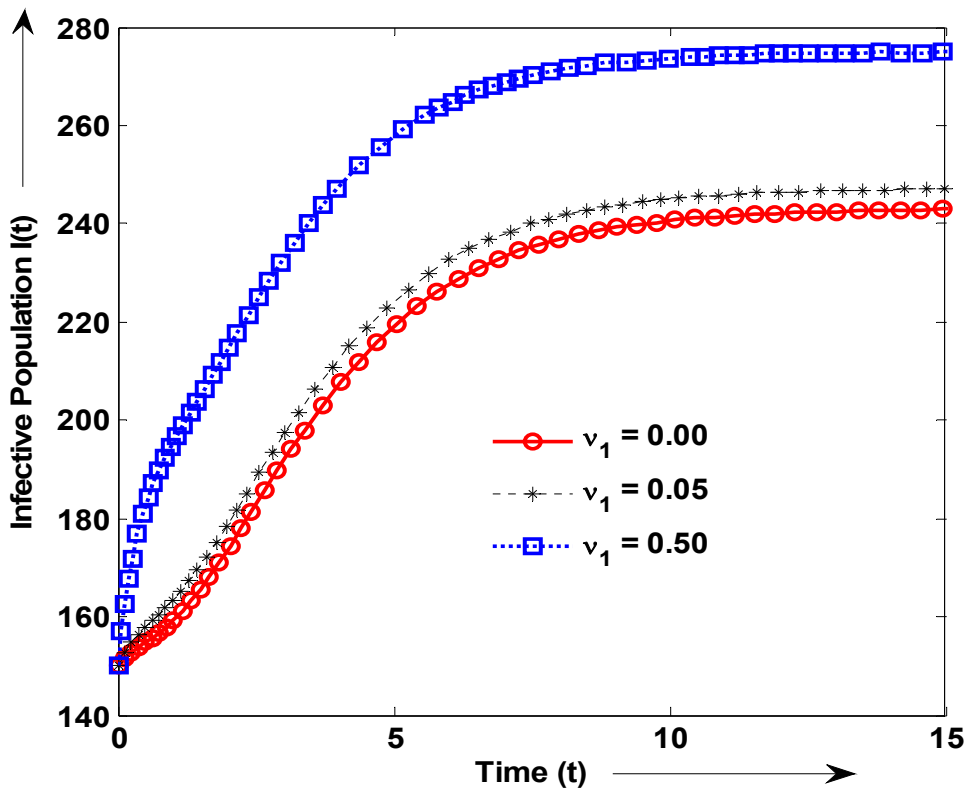

Figure 7. Variation of infective population with time for different rate of vaccination during incubation period.
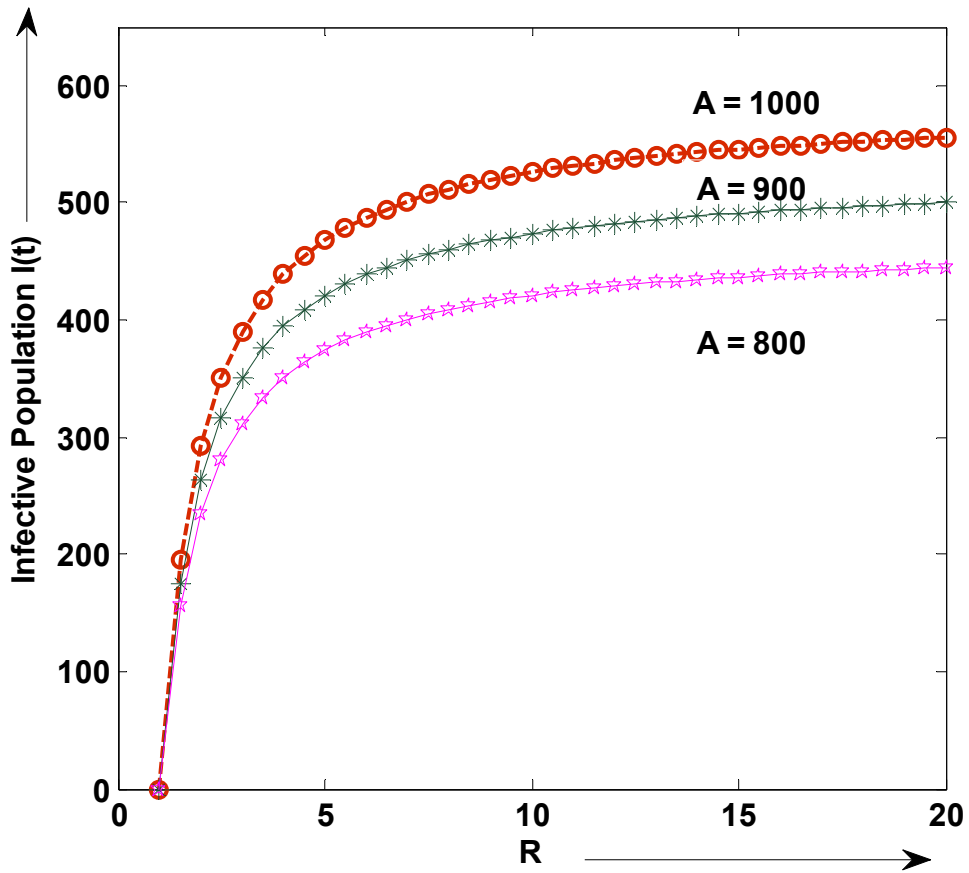
Figure 8. Variation of infective population with Basic reproduction ratio for varying immigration rate of population.