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Stability and bifurcation of an SIVS epidemic model with treatment and age of vaccination ☆

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ABSTRACT

In this paper, we consider a two-dimensional SIS model with vaccination. It is assumed that vaccinated individuals become susceptible again when vaccine loses its protective properties with time. Here the rate at which vaccinated individual move to susceptible class again, depends upon vaccine age and hence it is assumed to be a variable. This SIVS model with treatment exhibits backward bifurcation under certain conditions on treatment which complicate the criteria for the success of the treatment by making it possible to have stable endemic states. We also show how the infectivity and the recovery function affect the existence of backward bifurcation.

1. Introduction

In recent years, the main aim of researchers is to analysis and predict the consequences of the strategies designed to control infections diseases, particularly TB and AIDS. Attention has been given to vaccination and treatment policies both in terms of the different vaccine classes, vaccine efficacy (e.g. [1–4]) and also to application schedules and associated costs (e.g. [5–9]). The study of vaccination, treatment and associated behavioral changes related to disease transmission has been the subject of intense theoretical analysis.

The application of a vaccination and treatment programs has the likely effect of inducing behavioral changes in those individuals subjected to it.

In particular, in the case of HIV, risk behavior can be combined with vaccination or treatment resulting in a possible harmful effect in terms of disease prevalence.

Blower and McLean [10] have argued that a mass vaccination campaign can increase the severity of disease, if the vaccination is applied to only 50% of the population and vaccine efficacy is 60%. Velasco–Hernandez and Hsieh [11,12] confirmed this result in a theoretical mathematical model of disease transmission. They showed that a too-large case treatment rate combined with lengthening of the infectious period could result in the increase of the treatment reproduction number, i.e, treatment would contribute to the spread of disease rather than its elimination. Of course, these are theoretical investigation on the plausible effects of vaccination and treatment programs. However, in our model, we investigate the impact of a perfect vaccine, which means that vaccine confers a full protection against disease.

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Many population dynamics models have been developed considering age-structure. A simple model was first proposed by Lotka and Von Foerster [13,14], where the birth and the death processes were independent of the total population size and so the limitation of the resources were not taken into account. To overcome this deficiency, Gurtin and MacCamy [15], in their pioneering work considered a nonlinear age-dependent model, where birth and death rates were function of the total population.

The age-structure is a very important consideration in the models which describe the evolution of infection diseases. In 1974, a quite general age-dependent epidemic model was analyzed (see the pioneering work of Hoppensteadt [16,17]). Since then, the interest in studying the effects of the age factor on the epidemic models arises. Many works have been carried out on these models leading to quite complete epidemic models, for example, series of deterministic compartment models such as SI, SIS, SIR, SIRS, SEIR etc. have been proposed and analyzed [18–22], and also the different modes of disease transmission, vertical and horizontal are considered. However, the introduction of the age makes their analysis very complex, because of this, it is supposed habitually that the age only affects the vaccinated population [23].

We know that, in simple epidemic models, typically when the reproduction number is below one, only the disease-free equilibrium exists. This equilibrium is locally and globally stable, which implies that the disease will disappear from the population. Recommendations for disease control can be made based on that observation. In particular, measures which act to reduce the reproduction number below one will lead to the disease disappearance. Recently, it has been observed in theoretical considerations that nontrivial equilibria can be present even when the reproduction number of the disease is smaller than one. One way for this situation to occur is through a phenomenon called backward bifurcation. In the case of backward bifurcation, the endemic equilibrium which bifurcates from the disease-free equilibrium at the critical value one of the reproduction number, exists for values of the reproduction number smaller than one. In fact, for values of the reproduction number between some minimal value, called the minimal transition value, and one there are two or more endemic equilibria. If backward bifurcation occurs, it is not sufficient to reduce the reproduction number below one to eradicate the disease; instead, it is necessary to reduce it below a much lower value, the minimal transition value. Although this phenomenon is not as readily observed in data as oscillations, it plays a significant role in the dynamics of the disease and in our ability to combat it effectively. In recent years, the presence of backward bifurcation in epidemic models has led to significant interest. In many cases backward bifurcation seems to be caused by the presence of several classes with different susceptibilities to the disease (see, for example, [23,24] and the references therein).

In [25], wang studied an SIR model with treatment of the form $T(I)$ given by:

$$T(I) = \begin{cases} rI & \text{if } 0 \leqslant I \leqslant I_0, \\ k & \text{if } I > I_0, \end{cases}$$

where $k = rI_0$. We also consider the same form of treatment, mainly to analysis the effect of vaccination on the reproduction number.

2. Model formulation

We introduce an SIS model with vaccination. We consider the total population size as $N(t)$ whose demography is regulated by a constant birth/recruitment rate A and a natural mortality rate μ. The susceptible population is subjected to a vaccination campaign with the rate of vaccination as ψ. After vaccination, individuals move to the vaccinated class where they are completely protected from the infection. However, the vaccine loses its protective properties with time and eventually vaccinated individuals become susceptible again. We call the time individuals spend in the vaccinated class as vaccine-age and denote it by θ. The newly vaccinated individuals enter the vaccinated class $V(\theta, t)$ with vaccine-age equal to zero. The rate at which the vaccine wanes is denoted by $\alpha(\theta)$. The rate of transmission of the disease is β and the recovery rate is γ. Under these assumption, the model takes the following form:

$$\begin{aligned} &\frac{dS}{dt} = A - \beta SI - \mu S - \psi S + \gamma I + T(I) + \int_0^\infty \alpha(\theta) V(\theta, t) d\theta, \\ &\frac{dI}{dt} = \beta SI - \mu I - \gamma I - T(I), \\ &\frac{\partial V}{\partial \theta} + \frac{\partial V}{\partial t} = -\mu V(\theta, t) - \alpha(\theta) V(\theta, t), \\ &V(0, t) = \psi S(t), \end{aligned} \tag{1.1}$$

where

$$N(t) = S(t) + I(t) + \int_0^\infty V(\theta, t) d\theta$$

and initial conditions are

$$S(0) = S_0, \quad I(0) = I_0, V(\theta, 0) = V_0(\theta).$$

We note that the equation for the total population is $\frac{dN}{dt} = A - \mu N$. Thus, $N \to \frac{A}{\mu}$ as $t \to \infty$ i.e., the size of the population reaches its limiting value $N = \frac{A}{\mu}$.

Thus, we can assume that the initial value is $N_0 = S_0 + I_0 + \int_0^\infty V_0(\theta)d\theta = \frac{A}{\mu}$ in order to have a population of constant size $N = \frac{A}{\mu}$. Furthermore, integrating the third equation in (1.1) along the characteristic line $t - \theta =$ constant, we get the following formula:

$$V(\theta, t) = \begin{cases} V_0(\theta - t)\frac{K_0(\theta)}{K_0(\theta - t)} & \text{for } \theta \geqslant t, \\ \psi S(t - \theta)K_0(\theta) & \text{for } \theta < t, \end{cases} \tag{1.2}$$

where

$$K_0(\theta) = e^{-\mu\theta - \int_0^\theta \alpha(\tau)d\tau}.$$

Substituting (1.2) into the first equation in (1.1) we have

$$\frac{dS}{dt} = A - \beta SI - \mu S - \psi S + \gamma I + T(I) + \int_0^t K_1(\theta)S(t - \theta)d\theta + F_1(t), \tag{1.3}$$

where

$$\begin{aligned} K_1(\theta) &= \psi\alpha(\theta)K_0(\theta), \\ F_1(t) &= \int_t^\infty \alpha(\theta)\frac{K_0(\theta)V_0(\theta - t)}{K_0(\theta - t)}d\theta \end{aligned} \tag{1.4}$$

and $F_1(t)$ satisfies

$$\lim_{t\to\infty} F_1(t) = 0.$$

Thus it is sufficient to consider the following system of equations:

$$\begin{aligned} \frac{dS}{dt} &= A - \beta SI - \mu S - \psi S + \gamma I + T(I) + \int_0^t K_1(\theta)S(t - \theta)d\theta + F_1(t), \\ \frac{dI}{dt} &= \beta SI - \mu I - \gamma I - T(I). \end{aligned} \tag{1.5}$$

Here we have ignored the last equation of (1.1) as other equations in (1.1) do not contain $V(\theta, t)$ and once we solve the system of Eq. (1.5), we can use (1.2) to obtain $V(\theta, t)$. When $T(I) = rI$, (1.5) becomes

$$\begin{aligned} \frac{dS}{dt} &= A - \beta SI - \mu S - \psi S + \gamma I + rI + \int_0^t K_1(\theta)S(t - \theta)d\theta + F_1(t), \\ \frac{dI}{dt} &= \beta SI - \mu I - \gamma I - rI. \end{aligned} \tag{1.6}$$

When $T(I) = k$, (1.5) becomes

$$\begin{aligned} \frac{dS}{dt} &= A - \beta SI - \mu S - \psi S + \gamma I + k + \int_0^t K_1(\theta)S(t - \theta)d\theta + F_1(t), \\ \frac{dI}{dt} &= \beta SI - \mu I - \gamma I - k. \end{aligned} \tag{1.7}$$

This paper is organized as follows: in the next section, we present a qualitative analysis of the system (1.5). In Section 4, we study the global analysis of disease-free equilibrium and the local asymptotic stability of the endemic equilibria. In Section 5, using numerical simulation we demonstrate all theoretical results established in earlier sections. Finally we conclude our results in Section 6.

3. Equilibria

According to [26], any equilibrium (S, I) of the system (1.5), if it exists, must be a constant solution of the following limiting system associated with (1.5):

$$\begin{aligned} \frac{dS}{dt} &= A - \beta SI - (\mu + \psi)S + \gamma I + T(I) + \int_0^\infty K_1(\theta)S(t - \theta)d\theta, \\ \frac{dI}{dt} &= \beta SI - (\mu + \gamma)I - T(I). \end{aligned} \tag{2.1}$$

Thus we have to look for the solutions of the following system:

$$A = \beta SI + (\mu + \psi)S - \gamma I - T(I) - \Re_1 S,$$
$$\beta SI = (\mu + \gamma)I + T(I),$$

where

$$\Re_1 = \int_0^\infty K_1(\theta)d\theta = \psi - \psi\mu\Re_0 \quad \text{and} \quad \Re_0 = \int_0^\infty K_0(\theta)d\theta.$$

System (2.1) always has the disease-free equilibrium $E_0 = (S_0, 0)$, where

$$S_0 = \frac{A}{\mu(1 + \psi\Re_0)}.$$

When $0 < I \leqslant I_0$, system (2.1) becomes

$$\begin{cases} A = \beta SI + (\mu + \psi)S - \gamma I - rI - \Re_1 S, \\ \beta SI = (\mu + \gamma + r)I. \end{cases} \tag{2.2}$$

When $I > I_0$, system (2.1) becomes

$$\begin{cases} A = \beta SI + (\mu + \psi)S - \gamma I - k - \Re_1 S. \\ \beta SI = (\mu + \gamma)I + k. \end{cases} \tag{2.3}$$

Let

$$R_0(\psi) = \frac{A\beta}{\mu(\mu + \gamma + r)(1 + \psi\Re_0)}.$$

Then $R_0(\psi)$ is a basic reproduction number of (1.5). If $R_0(\psi) > 1$, (2.2) admits a unique positive solution $E^* = (S^*, I^*)$, where

$$S^* = \frac{\mu + \gamma + r}{\beta},$$
$$I^* = \frac{A\beta - (\mu + \psi)(\mu + \gamma + r) + \Re_1(\mu + \gamma + r)}{\mu\beta} = \frac{(R_0(\psi) - 1)(\mu + \gamma + r)(1 + \psi\Re_0)}{\beta}.$$

Clearly, E^* is an endemic equilibrium of (1.6) if and only if

$$1 < R_0(\psi) \leqslant 1 + \frac{\beta I_0}{(\mu + \gamma + r)(1 + \psi\Re_0)}. \tag{2.4}$$

In order to obtain positive solutions of system (2.3), we solve first equation of (2.3) for S to obtain

$$S = \frac{A + \gamma I + k}{\mu(1 + \psi\Re_0) + \beta I}.$$

Substituting it into the second equation, we have

$$\mu\beta I^2 + BI + \mu k(1 + \psi\Re_0) = 0, \tag{2.5}$$

where

$$B = \mu(\mu + \gamma)(1 + \psi\Re_0) - A\beta.$$

If $B \geqslant 0$, it is clear that (2.5) does not have a positive solution. Let us consider the case where $B < 0$. If $\Delta = B^2 - 4\mu^2\beta k(1 + \psi\Re_0)$. It is easy to obtain

$$\Delta = [\mu(1 + \psi\Re_0)(\mu + \gamma - R_0(\psi)(\mu + \gamma + r))]^2 - 4\mu^2\beta k(1 + \psi\Re_0). \tag{2.6}$$

It follows that $\Delta \geqslant 0$ is equivalent to

$$R_0(\psi) \leqslant -\frac{2\sqrt{\beta k/(1 + \psi\Re_0)}}{\mu + \gamma + r} + \frac{\mu + \gamma}{\mu + \gamma + r}, \tag{2.7}$$

or

$$R_0(\psi) \geqslant \frac{2\sqrt{\beta k/(1 + \psi\Re_0)}}{\mu + \gamma + r} + \frac{\mu + \gamma}{\mu + \gamma + r} \equiv p_0 \quad \text{(say)}. \tag{2.8}$$

It is noted that $B < 0$ is equivalent to

$$R_0(\psi) > \frac{\mu + \gamma}{\mu + \gamma + r}.$$

It follows that if the inequality (2.8) holds then $B < 0$ as well as $\Delta \geqslant 0$. Let us assume that (2.8) holds, then (2.5) has two positive solutions I_1 and I_2, where

$$I_1 = \frac{-B - \sqrt{\Delta}}{2\mu\beta}, \quad I_2 = \frac{-B + \sqrt{\Delta}}{2\mu\beta}. \tag{2.9}$$

Let

$$S_i = \frac{A + \gamma I_i + k}{\mu(1 + \psi\Re_0) + \beta I_i}$$

and $E_i = (S_i, I_i)$ for $i = 1, 2$. Then E_i is an endemic equilibrium of (1.5) if $I_i \geqslant I_0$. Let us consider the conditions under which $I_1 > I_0$. By the definitions, we see that it is equivalent to

$$-B - \sqrt{\Delta} > 2\mu\beta I_0. \tag{2.10}$$

This implies that

$$2\mu\beta I_0 + B < 0. \tag{2.11}$$

It follows from the definition of B that

$$R_0(\psi) > \frac{\mu + \gamma}{\mu + \gamma + r} + \frac{2\beta I_0}{(\mu + \gamma + r)(1 + \psi\Re_0)} \equiv p_1 \quad \text{(say)}. \tag{2.12}$$

Further, (2.10) implies that

$$(2\mu\beta I_0 + B)^2 > \Delta. \tag{2.13}$$

By direct calculations, we see that (2.13) is equivalent to

$$R_0(\psi) \leqslant 1 + \frac{\beta I_0}{(\mu + \gamma + r)(1 + \psi\Re_0)} \equiv p_2 \quad \text{(say)}. \tag{2.14}$$

Hence, $I_1 > I_0$ holds if and only if (2.12) and (2.14) are valid. Moreover, if $R_0(\psi) \leqslant p_1$ or $R_0(\psi) \geqslant p_2$, we have $I_1 \leqslant I_0$. By similar arguments as above, we see that $I_0 < I_2$ if (2.12) holds or

$$p_2 < R_0(\psi) \leqslant p_1. \tag{2.15}$$

Furthermore, $I_2 \leqslant I_0$ if

$$R_0(\psi) \leqslant min\{p_1, p_2\}. \tag{2.16}$$

Summarizing the discussions above, we have the following conclusions.

Theorem 2.1. *$E^* = (S^*, I^*)$ is an endemic equilibrium of (1.6) if and only if $1 < R_0(\psi) \leqslant p_2$. Furthermore, E^* is the unique endemic equilibrium of (1.6) if $1 < R_0(\psi) \leqslant p_2$ and one of the following conditions is satisfied: (i) $R_0(\psi) < p_0$, (ii) $p_0 \leqslant R_0(\psi) < p_1$.*

Theorem 2.2. *Endemic equilibria E_1 and E_2 do not exist if $R_0(\psi) < p_0$. Further, if $R_0(\psi) \geqslant p_0$, we have the following:*

(i) *if $p_1 < p_2$, then both E_1 and E_2 exist when $p_1 < R_0(\psi) < p_2$.*
(ii) *if $p_1 < p_2$, then E_1 does not exist but E_2 exists if $R_0(\psi) \geqslant p_2$.*
(iii) *Let $p_1 \geqslant p_2$. Then E_1 does not exist. Further, E_2 exists when $p_2 < R_0(\psi)$, and E_2 does not exist when $R_0(\psi) \leqslant p_2$.*

From Theorem 2.2(i), we can easily get the following corollary.

Corollary 2.3. *The system (1.7) has a backward bifurcation with endemic equilibria when $R_0(\psi) < 1$ if $p_1 < p_2$ and $p_0 < 1$.*

4. Stability analysis

Theorem 3.1. *The disease free equilibrium point E_0 is locally asymptotically stable if $R_0(\psi) < 1$ and is unstable if $R_0(\psi) > 1$.*

Proof. It is easy to see that the linearizing system of (2.1) at the equilibrium (S^*, I^*) takes the following form (here we take $T(I) = rI$)

$$\frac{ds}{dt} = -\beta S^* i(t) - \beta I^* s(t) - (\mu + \psi)s(t) + (\gamma + r)i(t) + \int_0^\infty K_1(\theta)s(t - \theta)d\theta,$$
$$\frac{di}{dt} = \beta S^* i(t) + \beta I^* s(t) - (\mu + \gamma + r)i(t).$$

In order to get the characteristic equation of the disease free equilibrium $E_0(S_0, 0)$, we linearize the system about E_0 and look for the following kind of solution of the system:

$$s(t) = e^{\lambda t}s, \quad i(t) = e^{\lambda t}i.$$

We get the following characteristic equation corresponding to the equilibrium point E_0:

$$\begin{vmatrix} \lambda + (\mu + \psi) - \widehat{K}_1(\lambda) & \beta S_0 \\ 0 & \lambda + \mu + \gamma + r - \beta S_0 \end{vmatrix} = 0, \tag{3.1}$$

where $\widehat{K}_1(\lambda)$ denotes the Laplace transform of $K_1(\theta)$. Now noting the expression of S_0 and $R_0(\psi)$, the Eq. (3.1) can be written as follows:

$$(\lambda + \mu + \psi - \widehat{K}_1(\lambda))(\lambda + (\mu + \gamma + r)(1 - R_0(\psi))) = 0.$$

If $R_0(\psi) > 1$, then the above characteristic equation has at least one positive root $\lambda = -(\mu + \gamma + r)(1 - R_0(\psi))$ implying E_0 is unstable. If $R_0(\psi) < 1$, then, obviously, all the solution of the characteristic equation have negative real parts provided all the roots of the equation

$$\lambda + \mu + \psi - \widehat{K}_1(\lambda) = 0 \tag{3.2}$$

have negative real parts.

In fact, if we assume $Re\lambda \geqslant 0$, then we have following two inequalities:

$$|\widehat{K}_1(\lambda)| = \left|\psi \int_0^\infty \alpha(\theta) e^{-\lambda\theta} e^{-\mu\theta} e^{-\int_0^\theta \alpha(\tau)d\tau} d\theta\right| < \psi \int_0^\infty \alpha(\theta) e^{-\int_0^\theta \alpha(\tau)d\tau} d\theta = \psi$$

and

$$|\lambda + \mu + \psi| \geqslant \mu + \psi > \psi$$

implying λ cannot be a root of Eq. (3.2). From which we conclude that all the roots of (3.2) have negative real parts. This completes the proof. □

Theorem 3.2. *The disease free equilibrium point E_0 is a global attractor if $R_0(0) < 1$.*

Proof. Note that

$$I' = \beta SI - (\mu + \gamma + r)I \leqslant \frac{A\beta}{\mu} I - (\mu + \gamma + r)I = (\mu + \gamma + r)\left(\frac{A\beta/(\mu + \gamma + r) - 1}{\mu}\right) I.$$

Also $R_0(0) = \frac{A\beta}{\mu(\mu+\gamma+r)} < 1$ implies $I(t) \to 0$ as $t \to \infty$.

Let $S_\infty = \liminf_{t\to\infty} S(t), S^\infty = \limsup_{t\to\infty} S(t)$. As in [23, Theorem 2], we choose the sequence $t_n^1 \to \infty$, $t_n^2 \to \infty$ such that $S(t_n^1) \to S^\infty, S(t_n^2) \to S_\infty$, and $S'(t_n^1) \to 0, S'(t_n^2) \to 0$. Then, from the first equation of (1.5), noticing that $I(t)$ and $F_1(t)$ go to 0 as $t \to \infty$, it follows that

$$0 \leqslant A - (\mu + \psi)S^\infty + (\psi - \psi\mu\Re_0)S^\infty,$$
$$0 \geqslant A - (\mu + \psi)S_\infty + (\psi - \psi\mu\Re_0)S_\infty.$$

So, we get $S^\infty = S_\infty = \frac{A}{\mu(1+\psi\Re_0)}$, and this completes the proof. □

Theorem 3.3. *The endemic equilibrium point E^* is locally asymptotically stable if the equation*

$$\lambda(1 + \psi\hat{K}_0(\lambda)) + \beta I^* = 0$$

has no roots with non-negative real parts. If $\alpha(\theta) = \alpha$ (constant), then E^* is always locally asymptotically stable.

Proof. The linearization of (1.6) at E^* gives the following characteristic equation:

$$\begin{vmatrix} \lambda + \mu + \psi - \widehat{K}_1(\lambda) + \beta I^* & \beta S^* - \gamma - r \\ -\beta I^* & \lambda + \mu + \gamma + r - \beta S^* \end{vmatrix} = 0. \tag{3.3}$$

Note that $\beta S^* = \mu + \gamma + r$ and

$$\widehat{K}_1(\lambda) = \psi - (\lambda + \mu)\psi\widehat{K}_0(\lambda),$$

where $\widehat{K}_0(\lambda)$ denotes the Laplace transform of $K_0(\theta)$. Eq. (3.3) is equivalent to

$$(\lambda+\mu)\begin{vmatrix} 1+\psi\widehat{K}_0(\lambda) & 1 \\ -\beta I^* & \lambda \end{vmatrix} = 0.$$

Hence we have one root as $\lambda = -\mu$, and if the following equation:

$$\lambda(1+\psi\hat{K}_0(\lambda)) + \beta I^* = 0$$

has no roots with non-negative real parts, then E^* is locally asymptotically stable. If $\alpha(\theta) = \alpha$ (*constant*), then above equation reduces to

$$\lambda^2 + (\mu+\alpha+\psi+\beta I^*)\lambda + (\alpha+\mu)\beta I^* = 0.$$

Obviously, this equation has no roots with nonnegative real parts. This completes the proof of the theorem. □

Theorem 3.4. *The nontrivial equilibrium point E_1 is unstable whenever it exists. And the nontrivial equilibrium point E_2 is locally asymptotically stable provided all roots of the following equation has negative real parts.*

$$\lambda = -(\mu+\gamma-\beta S_2) - \frac{\beta I_2}{1+\psi\widehat{K}_0(\lambda)}.$$

Especially, when $\alpha(\theta) = \alpha$, the equilibrium point E_2 is always locally asymptotically stable.

Proof. The linearization of (1.7) at E_i gives the following characteristic equation:

$$\begin{vmatrix} \lambda+(\mu+\psi)-\widehat{K}_1(\lambda)+\beta I_i & \beta S_i-\gamma \\ -\beta I_i & \lambda+\mu+\gamma-\beta S_i \end{vmatrix} = 0, \tag{3.4}$$

which is equivalent to

$$(\lambda+\mu)\begin{vmatrix} 1+\psi\widehat{K}_0(\lambda) & 1 \\ -\beta I_i & \lambda+\mu+\gamma-\beta S_i \end{vmatrix} = 0. \tag{3.5}$$

Clearly, one root of the last equation is $-\mu$. Now we just need to analyze roots distribution of the following equation

$$\lambda = -(\mu+\gamma-\beta S_i) - \frac{\beta I_i}{1+\psi\widehat{K}_0(\lambda)}. \tag{3.6}$$

Let us first consider the stability of the equilibrium point E_1. Let

$$F(\lambda) = -(\mu+\gamma-\beta S_1) - \frac{\beta I_1}{1+\psi\widehat{K}_0(\lambda)} - \lambda.$$

If λ is a real number, then

$$F'(\lambda) = -1 + \frac{\psi\widehat{K_0}'(\lambda)\beta I_1}{(1+\psi\widehat{K}_0(\lambda))^2} < 0$$

implying $F(\lambda)$ is a monotonic decreasing function for real λ. Also we note that

$$F(0) = -(\mu+\gamma-\beta S_1) - \frac{\beta I_1}{1+\psi\Re_0} = \frac{k}{I_1} - \frac{\beta I_1}{1+\phi\Re_0} = \frac{2\mu k(1+\psi\widehat{K}_0)+BI_1}{\mu I_1(1+\psi\widehat{K}_0)} > 0.$$

Hence the equation $F(\lambda) = 0$ must have a positive root, implying that the equilibrium E_1 is unstable.

Secondly, for the equilibrium E_2, if

$$\lambda = -(\mu+\gamma-\beta S_2) - \frac{\beta I_2}{1+\psi\widehat{K}_0(\lambda)}$$

has all roots with negative real parts, then this equilibrium point E_2 is locally asymptotically stable.

In the following, we discuss a special cease, $\alpha(\theta) = \alpha(constant)$, i.e., the rate at which the vaccine wanes is a constant. Then Eq. (3.6) reduces to

$$\lambda^2 + (\mu+\alpha+\psi+\mu+\gamma+\beta I_2-\beta S_2)\lambda + (\mu+\alpha+\psi)(\mu+\gamma-\beta S_2) + \beta I_2(\mu+\alpha) = 0.$$

Let

$$A = \mu+\alpha+\psi+\mu+\gamma+\beta I_2-\beta S_2, \quad B = (\mu+\alpha+\psi)(\mu+\gamma-\beta S_2)+\beta I_2(\mu+\alpha).$$

It is easy to see that

$$
\begin{aligned}
A &= \mu+\alpha+\psi+\mu+\gamma+\beta I_2 - R_0(\mu+\gamma+r)+\frac{\beta I_2}{1+\psi\Re_0}\\
&> \mu+\alpha+\psi-\frac{2\beta I_0}{1+\psi\Re_0}+\frac{\beta I_2}{1+\psi\Re_0}+\beta I_2>0 \quad (I_2>I_0),\\
B &= (\mu+\alpha+\psi)\left[\mu+\gamma-\frac{\beta A}{\mu(1+\psi\Re_0)}+\frac{\beta I_2}{1+\psi\Re_0}\right]+\beta I_2(\mu+\alpha)\\
&= (\mu+\alpha+\psi)\left[\mu+\gamma-R_0(\mu+\gamma+r)+\frac{\beta I_2}{1+\psi\Re_0}\right]+\beta I_2(\mu+\alpha)\\
&> (\mu+\alpha+\psi)\left[-\frac{2\beta I_0}{1+\psi\Re_0}+\frac{\beta I_2}{1+\psi\Re_0}+\beta I_2(\mu+\alpha)\right]\\
&= -2\beta I_0(\mu+\alpha)+2\beta I_2(\mu+\alpha)>0 \quad (I_2>I_0).
\end{aligned}
$$

So eigenvalues are either negative or have negative real parts, implying asymptotic stability of the equilibrium point E_2 when $\alpha(\theta)=\alpha(constant)$. This completes the proof. □

5. Simulation

The system (2.1) is simulated by considering α as constant i.e., the vaccine wanes at a constant rate. Simulation is performed using the package XPP (see [27]). The simulation results are consistent with the analytical results. In Figs. 1–8, (S,I) phase planes including nullclines are drawn which confer the existence and the stability of different equilibria of the system (2.1). In each of these figures, the red curve represents the S-nullcline and the green one represents the I-nullcline and obviously the intersection of these two curves gives the equilibria of the system. Here the Fig. 1, shows the local asymptotic stability of the infection-free equilibrium $E_0(258.0645,0)$ when the reproduction number $R_0(\psi)=0.1281171$ for the parameter values as follows: $r=1.2, A=80, \beta=0.0007, \mu=0.01, \psi=0.9, \gamma=0.2, \alpha=0.02, I_0=150$. Here, Fig. 2, is describing the situation when the equilibrium $E^*(163.75,61.25)$ is locally asymptotically stable and the infection-free equilibria E_0 is unstable where $R_0(\psi)=1.0178117$ for the parameter values $r=1.2, A=35, \beta=0.008, \mu=0.01$, $\psi=0.8, \gamma=0.1, \alpha=0.03, I_0=1000$. and all conditions stated in Theorem 2.1(i) are satisfied. The equilibria E_1 and E_2 do not exist in this case.

Fig. 3 is describing the situation when the equilibrium $E^*(54.0001170.286)$ is locally asymptotically stable and the infection-free equilibria E_0 is unstable where $R_0(\psi)=2.194517$ and all conditions stated in Theorem 2.1(ii) are satisfied. The equilibria E_1 and E_2 do not exist in this case. Bi-stability is shown in Figs. 4, 5. Fig. 4 is describing the situation when $1<R_0(\psi)=p_0=1.3888889$ and $p_1<R_0(\psi)<p_2$ where Theorem 2.2 (i) holds. Here it is found that both the equilibria $E_1=E_2=(45.249,676.01)$ and $E^*=(63,392)$ are locally asymptotically stable. Here Fig. 5 too describes the same situation

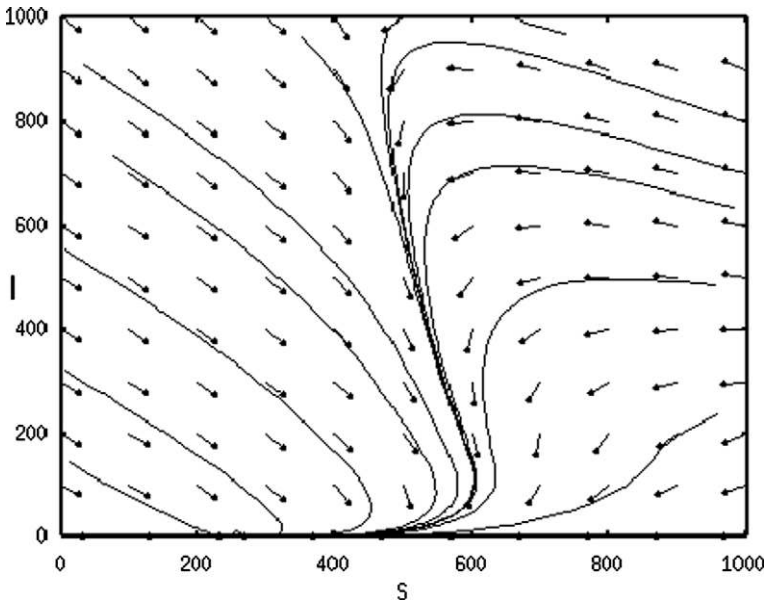

Fig. 1. Phase plot of I verses S including nullclines showing infection free equilibrium to be stable when $R_0(\psi)<1$.

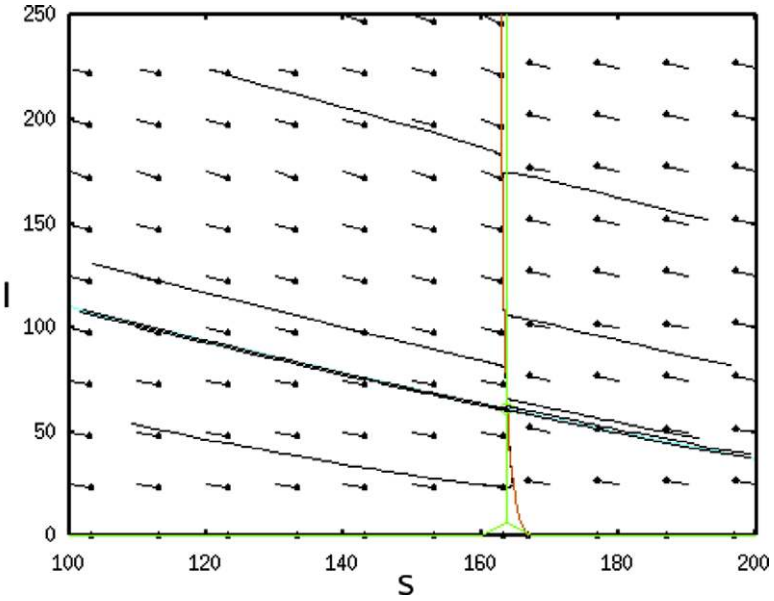

Fig. 2. Phase plot of I verses S including nullclines showing the existence of only E^* which is globally stable when $1 < R_0(\psi) < p_2$ and $R_0(\psi) < p_0$.

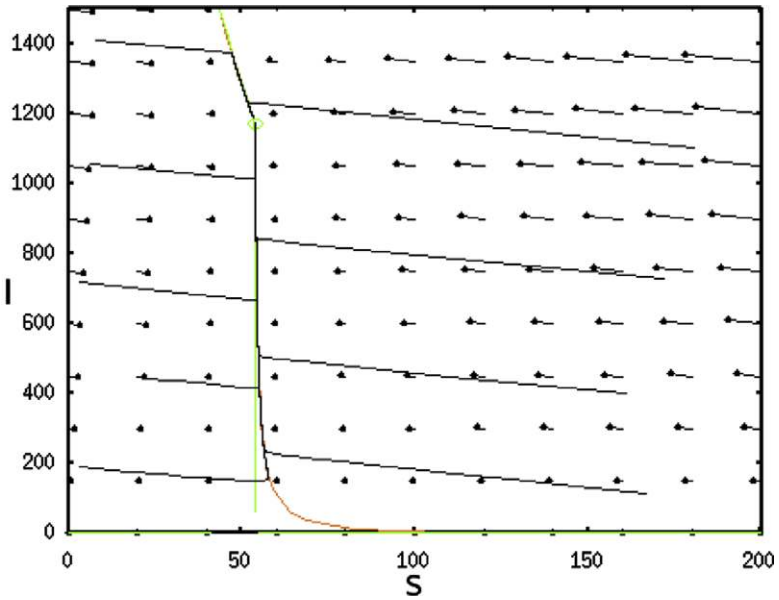

Fig. 3. Phase plot of I verses S including nullclines showing the existence of only E^* which is globally stable when $1 < R_0(\psi) < p_2$ and $p_0 \leqslant R_0(\psi) < p_1$ for the parameter values $r = 0.7, A = 21.5, \beta = 0.015, \mu = 0.01, \psi = 0.6, \gamma = 0.1, \alpha = 0.025, I_0 = 1175$.

except in this case the equilibria E_1 and E_2 are distinct. It is observed that the equilibrium $E^*(63, 392)$ and the equilibrium $E_2(29.516, 927.75)$ are stable and the equilibrium $E_1(60.98412, 424.254)$ is unstable which lies in between E^* and E_2.

In Fig. 6, existence of E_1, E_2 and the infection free equilibrium E_0 is shown. Here $R_0(\psi) < 1$ which implies E^* does not exist. It is found that the equilibria $E_0(276.5957, 0)$ and $E_2(188.7547, 2064.264)$ are stable and the equilibrium $E_1(237.841, 910.7364)$ is unstable. Fig. 7 describes the situation where Theorem 2.2(ii) holds, i.e. in this case the equilibrium $E_2(75.9514, 1779.379)$ exists and is asymptotically stable. Obviously the infection-free equilibrium $E_0(688.52, 0)$ exists but is unstable as $R_0 > p_2 > 1$. Finally, the Fig. 8 describes the situation where the Theorem 2.2 (iii) holds, i.e., when the equilibrium $E_2(145.3867, 1167.062)$ exists and is stable. Obviously from all these plots, it is clear that if E_2 exists, the number of infectives i.e. I_2 is always greater than the number of infectives corresponding to other equilibria when they exist. These facts are demonstrated in the following bifurcation diagrams (see Figs. 9–11). The first bifurcation diagram (see Fig. 9) is obtained by taking the constant recruitment rate A as the critical parameter. The other parameter values are as follows:

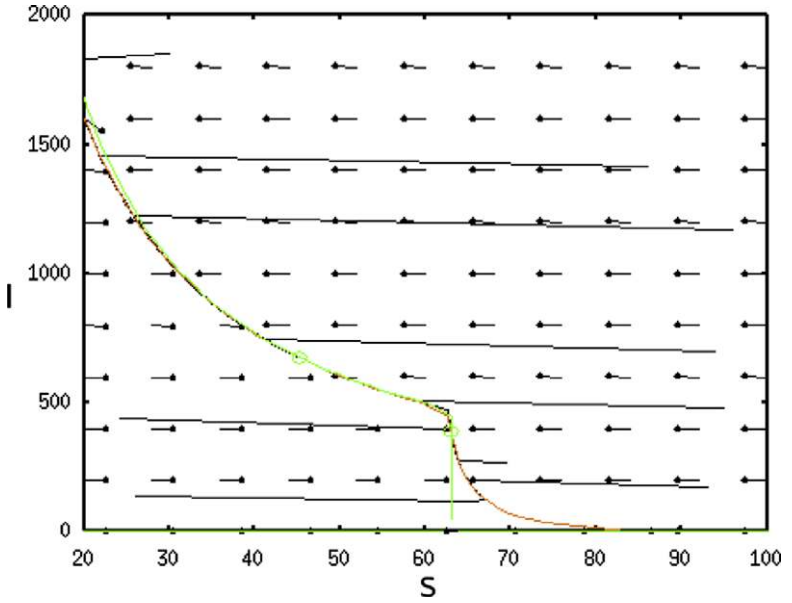

Fig. 4. Phase plot of I verses S including nullclines showing bi-stability when $R_0(\psi) = p_0$ and $p_1 < R_0(\psi) < p_2$ for the parameter values $r = 1.2, A = 70, \beta = 0.02, \mu = 0.05, \psi = 0.9, \gamma = 0.01, \alpha = 0.01, I_0 = 476.0166665$.

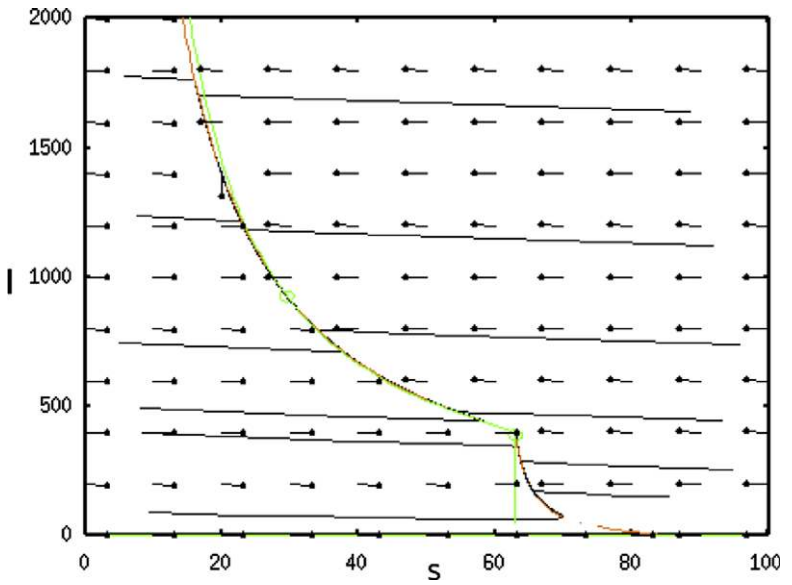

Fig. 5. Phase plot of I verses S including nullclines showing bi-stability when $R_0(\psi) = p_0$ and $p_1 < R_0(\psi) < p_2$ for the parameter values $r = 1.2, A = 70, \beta = 0.02, \mu = 0.05, \psi = 0.9, \gamma = 0.01, \alpha = 0.01, I_0 = 410$.

$$r = 1.2, \quad \beta = 0.02, \quad \mu = 0.05, \quad \psi = 0.9, \quad \gamma = 0.01, \quad \alpha = 0.01, \quad I_0 = 476.0166665.$$

The horizontal axis is labelled with the appropriate value of the reproduction number R_0 corresponding to this bifurcation parameter A. It is observed that when the reproduction number is between 0 and 1, we have a stable infection free equilibrium. At the point where $R_0(\psi) = 1$ which corresponds to $A = 50.4$, we have forward bifurcation and this onwards, endemic equilibrium E_* is stable until $R_0(\psi)$ reaches to $R_0(\psi) = 1.389$ which corresponds to $A = 70$. Here we get backward bifurcation which leads to the existence of multiple endemic equilibria. Thus we get bistability for $1.389 < R_0(\psi) < 1.468254$ where the equilibria E^* and E_2 are stable and the equilibrium E_1 is unstable. For $R_0(\psi) > 1.468254$, there exists only one equilibrium point E_2 which is stable. Fig. 10, is also showing the backward bifurcation from the endemic equilibrium. This diagram is drawn by considering the contact rate β as the bifurcation parameter and other parameter values are as follows:

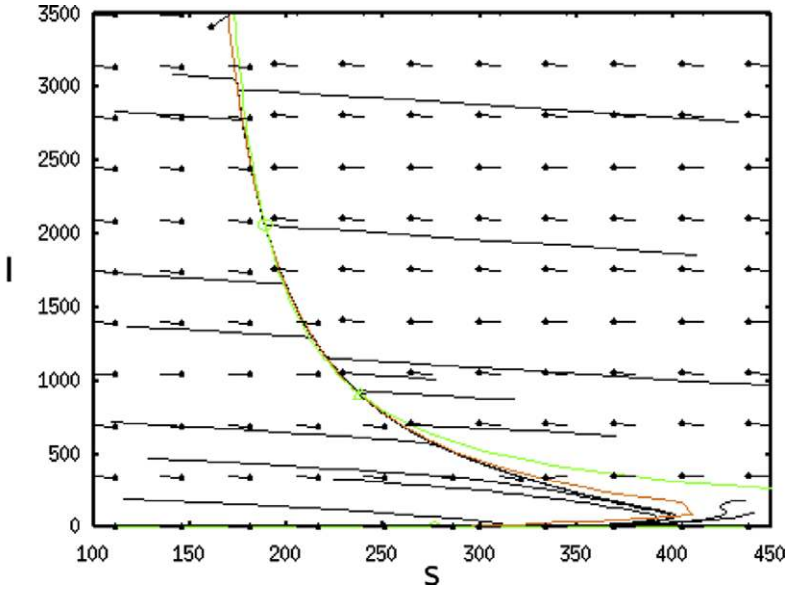

Fig. 6. Phase plot of I verses S including nullclines showing the existence of E_1 and E_2 and the stability of E_2 and the infection free equilibrium E_0 when $p_0 < R_0 < 1$ and $p_1 < R_0 < p_2$ for the parameter values $r = 1.2, A = 130, \beta = 0.0018, \mu = 0.02, \psi = 0.9, \gamma = 0.25, \alpha = 0.02, I_0 = 120$.

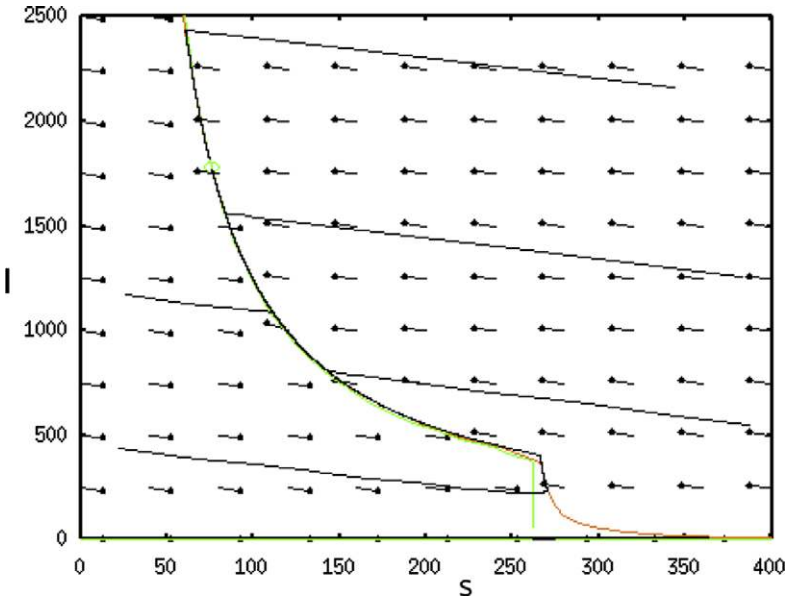

Fig. 7. Phase plot of I verses S including nullclines showing the existence of only E_2 which is stable when $p_1 < p_2, p_0 < R_0$ and $R_0 > p_2 > 1$ for the parameter values $r = 1.2, A = 20, \beta = 0.005, \mu = 0.01, \psi = 0.4, \gamma = 0.1, \alpha = 0.2, I_0 = 400$.

$$r = 1.2, \quad A = 130, \quad \mu = 0.02, \quad \psi = 0.9, \quad \gamma = 0.25, \quad \alpha = 0.02, \quad I_0 = 120.$$

As in the previous diagram, the horizontal axis is labelled with the appropriate value of the reproduction number R_0 corresponding to this bifurcation parameter β. Here we see that even for $R_0(\psi) < 1$, we get stable endemic equilibria E^* and E_2 and unstable equilibrium E_1 in between. This shows that by reducing the reproduction number $R_0(\psi)$ below one is not sufficient to eliminate the disease from the population. Hence we need to study the dynamics of the disease carefully before applying any control measures which cause the decrease in $R_0(\psi)$. As in the situation described in Fig. 1, where there is a forward bifurcation at $R_0(\psi) = 1$ it is sufficient to decrease $R_0(\psi)$ to 1 but in the case of backward bifurcation described in Fig. 10, we need to make $R_0(\psi)$ well below 1. In Fig. 11, bifurcation is performed by taking the threshold value of the infective population I_0 as the critical parameter. Here it is noted that this parameter is involved in the treatment. As it is assumed that

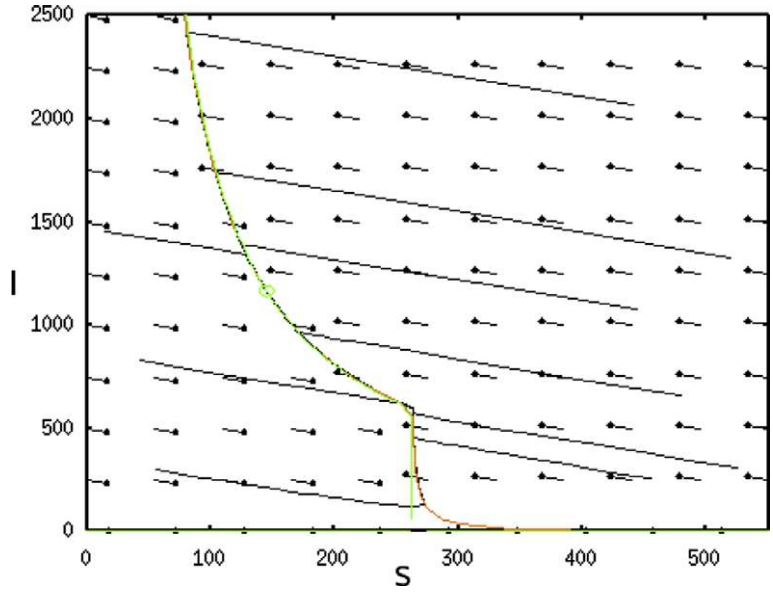

Fig. 8. Phase plot of I verses S including nullclines showing the existence of only E_2 which is stable when $R_0 > p_0$, $p_1 > p_2$ and $p_2 < R_0$ for the parameter values $r = 1.2, A = 15, \beta = 0.005, \mu = 0.01, \psi = 0.4, \gamma = 0.1, \alpha = 0.3, I_0 = 600$.

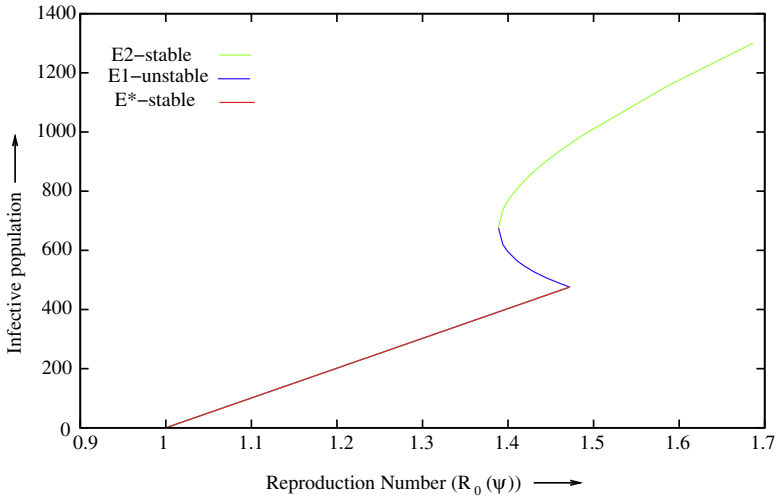

Fig. 9. The variation of equilibrium level of the infective population with the reproduction number showing the backward bifurcation from an endemic equilibrium at $R_0(\psi) = 1.389$ where Theorems 2.1 and 2.2 hold.

treatment is proportional to the number of infective until the infective population reaches a threshold value I_0 and after that the treatment function is a constant. Due to this fact in this figure we see that the equilibrium level of the infective population decreases with the increase in I_0 until it comes to a saturation point (in our case $I_0 = 392$) where increasing it further gives bistability until I_0 reaches to 476.0166 and after that the equilibrium level of the infective population stabilizes i.e, there is no effect of increasing I_0 further. This is the situation when the medical facilities are more than sufficient and hence there is no effect of improving it further. Now we summarize our simulation results in Table 1.

From Table 1, we observe that when α i.e., the rate at which vaccine wanes is high, only E_2 exists which is locally asymptotically stable and in this case other equilibria do not exist. The equilibrium E^* is stable when I_0 is very large and in this case E_1 and E_2 do not exist. When I_0 is somewhat reasonable neither very high nor very low, there is a possibility of existence of all nontrivial equilibria and in this case E^* and E_2 become stable and E_1 always remains unstable. When the recovery rate γ is very high, then $R_0(\psi)$ becomes small and in this case E^* do not exist but E_1 and E_2 can exist and E_0 and E_2 can be stable.

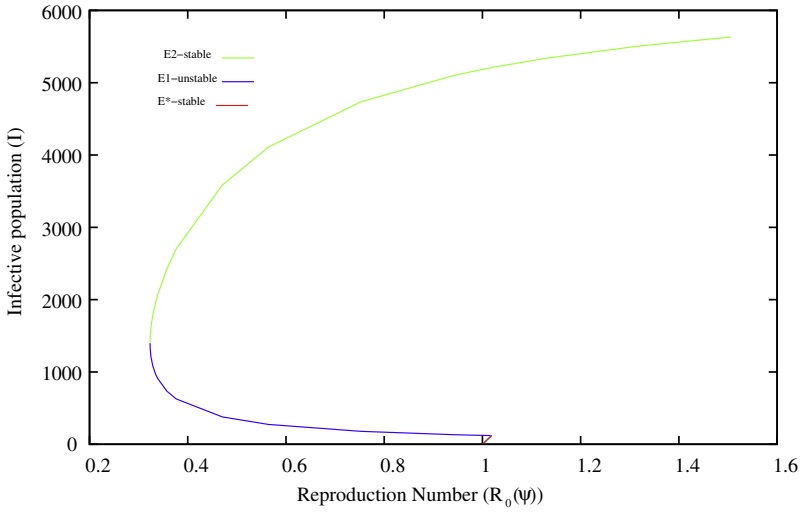

Fig. 10. The variation of equilibrium level of the infective population with the reproduction number showing the backward bifurcation from an endemic equilibrium at $R_0(\psi) = 1.0187002$ where Corollary 2.3 holds.

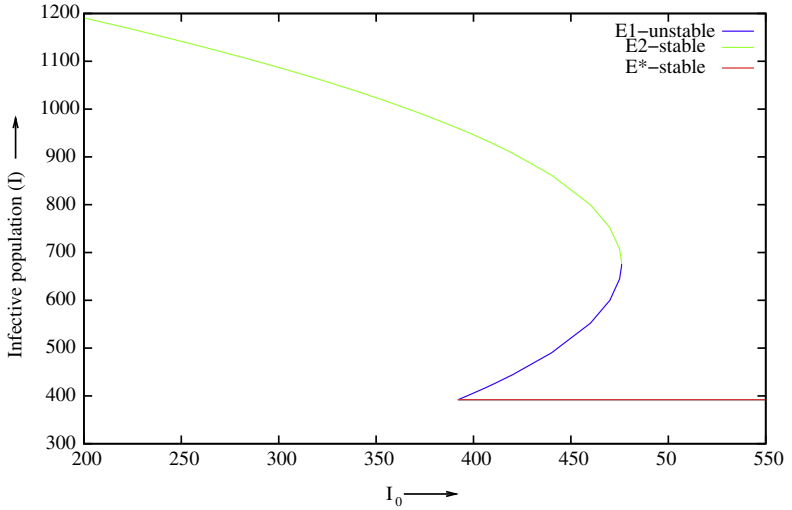

Fig. 11. The variation of equilibrium level of the infective population with I_0 showing the bifurcation at $I_0 = 392$ from where we have bistability until $I_0 = 476.0166$ for the parameters $r = 1.2, A = 70, \beta = 0.02, \mu = 0.05, \psi = 0.9, \gamma = 0.01, \alpha = 0.01$.

Table 1
Simulation results.

r	A	β	μ	ψ	γ	α	I_0	$R_0(\psi)$	p_0	p_1	p_2	Result
1.2	80	0.0007	0.01	0.9	0.2	0.02	150	0.128	0.239	0.153	1.0024	E_0 stable
1.2	35	0.008	0.01	0.8	0.1	0.03	1000	1.0178	1.116	0.665	1.29	E^* stable and E_1, E_2 do not exist
0.7	21.5	0.015	0.01	0.6	0.1	0.025	1175	2.194	2.172	2.534	2.199	E^* stable and E_1, E_2 do not exist
1.2	70	0.02	0.05	0.9	0.01	0.01	476.0167	1.388	1.388	0.992	1.472	$E_1 = E_2$ and E^* are stable
1.2	70	0.02	0.05	0.9	0.01	0.01	410	1.388	1.292	0.861	1.406	E^* and E_2 are stable and E_1 is unstable
1.2	130	0.0018	0.02	0.9	0.25	0.02	120	0.338	0.326	0.196	1.006	E_0 and E_2 are stable E_1 is unstable, E^* does not exist
1.2	20	0.005	0.01	0.4	0.1	0.2	400	2.628	1.471	1.135	1.525	Only E_2 exists and is stable E_0 is unstable
1.2	15	0.005	0.01	0.4	0.1	0.3	600	2.628	1.471	1.135	1.525	Only E_2 exists and is stable E_0 is unstable

6. Concluding remarks

In this paper, we have proposed an age-structured model by incorporating the vaccine age in the model. The local and global stability of the equilibria of the model are discussed. In epidemiological modelling, the sensitivity of the results to

the parameter's values are very important in planning the control strategies. This model is simulated for the various set of parameters and results are consistent with the analytical results. It is found that the model shows backward bifurcation for various critical parameters such as the rate of recruitment in the population, the contact rate, the rate of vaccination and the rate at which vaccine wanes. As the treatment depends upon the threshold value of the infective population, we get bifurcation and bistability by considering this as the critical parameter. Here it is found that initially the equilibrium level of the infectives decreases with the increase in the I_0 that means providing more facilities/beds reduces the epidemic until this threshold I_0 coincides with the equilibrium level of the epidemic. Then for a small range of the threshold value I_0 we get bistability and after that increasing I_0 further does not affect the equilibrium level of the epidemic i.e. a saturation arrives where by increasing the medical facilities/beds one cannot reduce the severity of the epidemic. This is easy to visualize as when the medical facilities are more than sufficient then there is no use of improving it further.

Hence in the case of limited medical facilities, one should try to lower the level of initial infectious invasion to the threshold I_0 to minimize the number of infectives at the endemic steady state. From all these simulation results we conclude that one need to plan the control strategies depending upon the situation as just lowering the reproduction number to one is not always sufficient to eliminate the disease from the population.

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