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ORIGINAL PAPER

Dynamics of an *SVEIRS* Epidemic Model with Vaccination and Saturated Incidence Rate

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Abstract
Measles and influenza are two major diseases–caused an epidemic in India. Therefore, in this paper, a *SVEIRS* epidemic mathematical model for measles and influenza is proposed and analyzed, where pre and post vaccinations are considered as control strategies with waning natural, vaccine-induced immunity and saturation incidence rate. The dissection of the proposed model is conferred in terms of the associated reproduction number $\mathcal{R}_v$, which is determined by the next-generation approach and obtained that if $\mathcal{R}_v \leq 1$, the disease-free equilibrium exists and it is locally as well as globally asymptotically stable. Further for $\mathcal{R}_v > 1$, a unique endemic equilibrium exists and it is also locally as well as globally asymptotically stable under certain conditions, which shows the prevalence and persistence of the disease in the population.

Keywords Pre and post vaccinations · Reproduction number · Saturated incidence rate · Global stability

Introduction

Mathematical modeling has become an essential tool to analyze the spread and control of several infectious diseases [1,2]. In recent years, many attempts have been made to develop some sensible mathematical models for investigating the dynamics and asymptotic behaviors [3–6]. Mathematical models take into account foremost factors that govern improvement of a disease, such as vaccination, transmission, recovery etc. The most important factor in the epidemic is asymptomatic or unapparent, where the infectious diseases spread incessantly throughout the population without showing symptoms. Individuals with asymptomatic infection are still infectious and supply to the dissemination of the disease from one human to a different [7,8]. Asymptomatic diseases can decline the immune system and make individuals susceptible to other diseases. When a subclinical infection is not noticed by the infected indi-

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vidual, it is dangerous not only for infected individuals but also for other people around him. These infections are very risky for certain association of people such as pregnant women. The asymptomatic situation may not be identified until the patient undergoes medical tests (X-rays or other inquisition). Therefore, some people may remain asymptomatic for a remarkably long period of time and even they also died. This type of infection exists for many diseases, like measles, influenza etc. [5,7].

Vaccines play a significant role in keeping us vigorous as they defend us from serious and sometimes fatal diseases, e.g., influenza and measles. There is a vast variability in the construction of vaccination models in epidemiology which entirely depends on the disease and the type of vaccines. Some vaccines may be greatly effective against infection while others may be unsuccessful in various respects like degree, duration, infectiousness, and progression etc [9]. The most frequent vaccination plan is to immunize all individuals, where the spread of various infectious diseases can be prevented by giving vaccination at a regular rate to the susceptible population on pre or post level [10–12]. The pre-vaccination of susceptible may reduce the level of an epidemic, which was studied by the several researchers [9,13,14]. The cohort (or pre-vaccination) is considered in [9], where a proportion of newly recruited individuals are vaccinated. While the impact of post vaccination is studied by a number of researchers [15,16], and obtained that, to eliminate the disease from the population, vaccination rate must be larger than the critical vaccination coverage otherwise the disease persists in the population. But only a few numbers of researchers studied both vaccination strategies simultaneously at the same time [17,18].

In modeling of the infectious diseases, the incidence function play a key role as it can determine the rise and fall of epidemics and represents the number of new cases per unit time. Some factors, such as density and lifestyle of the population could affect the incidence rate openly or circuitously. Many forms of incidence rates are possible. In the conventional epidemic disease models, bilinear incidence rate βSI and standard incidence rate $\frac{\beta SI}{N}$ have been studied. The bilinear incidence rate is based on the law of mass action and is a tremendous type, which can't elucidate enhanced the sophisticated phenomena of disease conduction [19–21]. Moreover, the standard incidence rate may be a good approximation if the number of existing partners is huge enough and each one could not make more contacts than is basically reasonable [9,22–24]. The saturated incidence rate $\frac{\beta SI}{1+\alpha I}$, which tends to a saturation level when I gets large, where βI measures the infection force when the disease is entering a fully susceptible population, and $\frac{1}{1+\alpha I}$ measures the inhibition effect from the behavioral change of susceptible individuals when their number increases or from the crowding effect of the infective individuals [25]. Capasso and Serio introduced a saturation incidence rate $\frac{\beta SI}{1+\beta\delta I}$ $(\delta > 0)$ in [26], for the cholera epidemic model, where the infection force for a very large number of infective may decrease as the number of infective individuals increases. Saturation incidence rate seems more sensible than the bilinear incidence rate because of it includes the behavioral change and crowding effect of the infective individuals and prevents the unboundedness of the contact rate by choosing suitable parameters [15,27,28]. Longini Jr et al. [29,30] and Germann et al. [31], proposed models by including two classes of infective persons, namely, symptomatic infective with clinical symptoms and asymptomatic infective with no or negligible symptoms. A model to include the asymptomatic stage in a standard $SARS$ epidemic model to study the treatment and chemoprophylaxis strategies and their effects on enlarging of the disease is proposed by Stilianakis et al. [32]. According to our knowledge, the role of asymptomatic infection on the transmission dynamics of influenza was never analytically explored. It is thought that, asymptomatic cases and asymptomatic infection of influenza occur regularly [33,34].

Several studies have indicated that asymptomatic infections account for about one-third of infections [35–38]. Through its character, asymptomatic flu cases are difficult to diagnose. Therefore, it is impossible to specifically define the number of asymptomatic cases. As a result, clinical evidence of asymptomatic infection is enormously limited and the size of its donation to spread of flu is tough to determine. Longini Jr et al. [29], assumed that the chance that a person will be symptomatic given that person has been infected is 0.67, based on population-level influenza cohort studies in the U.S. Furthermore, they assumed that an asymptomatic infection is only 50% as infectious as a symptomatic infection. Intuitively, there might be a considerable difference in the respective transmission probabilities from asymptomatic and symptomatic person to susceptible [8]. Obviously, an infected person with clinical illness sheds additional virus than does one with sub-clinical symptoms. Alternatively, infected persons with clinical symptoms may show reduced contacts if they are sufficiently ill to be restricted to bed. For clear discussion, the readers are referred to Hsu and Hsieh [8] and references therein. The aforementioned modeling studies with asymptomatic stage utilize complex models and simulation studies to explore the role of sub-clinical infection in intervention strategies.

The present study is more reasonable than the existing literature as it includes the impact of asymptomatic individuals with both pre and post vaccinations, which has not been given much prominence in the past. Motivated with the work of Sahu and Dhar [15], in this paper we consider a *SVEIRS* epidemic model with asymptomatic stage and saturated incidence rate. The intent of this paper is to propose an epidemic model, to examine the global dynamics and ensures the conditions for the eradication of the disease from the population.

This article is organized as follows: in "Formulation of the Mathematical Model" section, we develop a mathematical model as system of ODEs and describe all parameters used in the model. In section "Steady States and the Control Reproduction Number", we determine all possible steady states and associated reproduction number. In section "Stability Analysis of the Equilibria", stability analysis is carriedout for both the equilibriua. Finally, numerical simulations and discussions are given in the last section.

Formulation of the Mathematical Model

An SVEIS epidemic model for an infectious disease that spread in the host population through horizontal transmission was investigated by Sahu and Dhar [15]. They proposed the following mathematical model:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda - \mu S - \omega S + \theta V - \frac{\beta SI}{1+aI} + (1-q)\gamma I,\\
\frac{dV}{dt} &= \omega S - \theta V - \mu V + \xi E + q\gamma I,\\
\frac{dE}{dt} &= \frac{\beta SI}{1+aI} - \mu E - \xi E - \sigma E,\\
\frac{dI}{dt} &= \sigma E - \mu I - \gamma I,
\end{aligned}$$

where Λ is the recruitment rate of susceptible; μ is the natural death rate; ω is the rate at which susceptible individuals are vaccinated; θ is the rate at which vaccine wanes; β is the transmission coefficient of exposed individuals; $\frac{1}{a}$ is the half-saturation constant of infected individuals; q is the fraction recovered individuals getting disease acquired immunity; ξ is

the rate of recovery from exposed class due to natural immunity; $\frac{1}{\gamma}$ is the average length of infection of the infected individuals and $\frac{1}{\sigma}$ is the average time-span of infected individuals in exposed class.

In [15], the authors assumed that constant vaccination is applied continuously and the population is mixing and interacting homogeneously. The disease induced death rate is negligible and infection is spread due to the interaction of infected and susceptible individuals with nonlinear saturation incidence rate. Another model was proposed by Samsuzzoha et al. [41] and studied uncertainty and sensitivity of the basic reproduction number of a vaccinated epidemic model of influenza. From these models, it is observed that the model is given in [15] is not applicable in the case if the symptoms of infectious diseases are not shown (e.g., influenza and measles diseases). Also, in the model proposed by Samsuzzoha et al. [41], the authors do not consider the case in which the infection is asymptomatic and the effect of vaccination on the infected or infectious individuals is not discussed. Hence, the model is extended by incorporating a new compartment for the infectious individuals, when they will remain in an asymptomatic stage without showing any symptoms and also consider pre-vaccination and post-vaccination strategies.

Therefore, in this paper, we propose a mathematical model with vaccination and saturation incidence rate. Here, the susceptible-vaccinated-exposed-infected- asymptomatic-recovered-susceptible ($SVEIRS$) model, to be described. The model divides the total population size at time t, say $N(t)$, into seven mutually exclusive subpopulations, called compartments, e.g. susceptible ($S(t)$), pre-vaccinated ($V_1(t)$), post-vaccinated ($V_2(t)$), exposed ($E(t)$), asymptomatic ($A(t)$) and recovered ($R(t)$), such that $N(t) = S(t) + V_1(t) + V_2(t) + E(t) + I(t) + A(t) + R(t)$. Here, the population is mixing, interacting equivalently and the disease induced death rate is negligible. The transmission of diseases is taken to be horizontally and vertically and the movement of population is not considered in this paper. The basic assumptions of the model are as follows:

(A1) The model assumes a simple demographic progression in which recently recruited individuals (such as newborns or pre and early-adolescents) come into the population at a rate Π and go out (e.g., due to natural death) at the same rate μ.
(A2) Infectious persons can be divided into two compartments namely, $I(t)$ (with symptoms) and $A(t)$(without any symptoms called asymptomatic). $A(t)$ is assumed to be less infective than $I(t)$, i.e., $0 < b < 1$, where b is the proportion at which the exposed individuals will join the infectious class.
(A3) The disease induces momentary protection in a fraction of recovered population and rest of the recovered population rejoin to the susceptible class.
(A4) Instead of being infected, a small part of exposed individuals recovers and develops disease acquired short-term immunity and hence joins to vaccinated class.
(A5) After becoming infected, one part of the population becomes infected with the rate b and remaining part becomes asymptomatic.
(A6) Vaccination is assumed to be temporary effective and continuous vaccination is given to the population with constant vaccination rate.

Keeping in mind the above assumptions and the transition diagram shown in Fig. 1, our proposed *SVEIRS* epidemic model is given by the following system of ordinary differential equations:

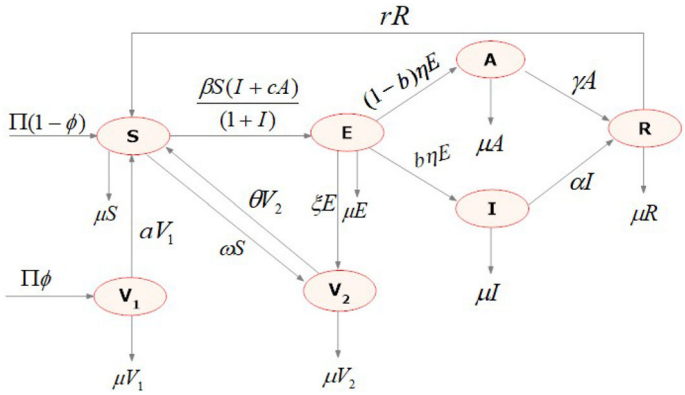

Fig. 1 Flow chart of an *SVEIRS* epidemic model with demographic effect

$$
\begin{aligned}
\frac{dS}{dt} &= \Pi(1-\phi) + aV_1 - \frac{\beta S(I+cA)}{(1+I)} + \theta V_2 + rR - (\omega+\mu)S,\\
\frac{dV_1}{dt} &= \Pi\phi - (a+\mu)V_1,\\
\frac{dV_2}{dt} &= \omega S + \xi E - (\theta+\mu)V_2,\\
\frac{dE}{dt} &= \frac{\beta S(I+cA)}{(1+I)} - (\xi+\eta+\mu)E,\\
\frac{dI}{dt} &= b\eta E - (\alpha+\mu)I,\\
\frac{dA}{dt} &= (1-b)\eta E - (\gamma+\mu)A,\\
\frac{dR}{dt} &= \alpha I + \gamma A - (r+\mu)R,
\end{aligned}
\tag{2.1}
$$

where the descriptions of parameters used in above model is given in the Table 1.All the parameters of the model (2.1) are assumed to be positive. The model has following initial conditions:

$$
\begin{aligned}
&S(0) = S_0 \geq 0, \quad V_1(0) = V_{10} \geq 0, \quad V_2(0) = V_{20} \geq 0, \quad E(0) = E_0 \geq 0,\\
&A(0) = A_0 \geq 0, \quad I(0) = I_0 \geq 0 \quad \text{and} \quad \mathrm{R}(0) = \mathrm{R}_0 \geq 0.
\end{aligned}
$$

Steady States and the Control Reproduction Number

Now, we appraise all feasible steady states and the associated reproduction number $\mathcal{R}_v$ for the model system (2.1).

Positive Invariance and Boundedness of the Solution Set

It is easy to prove that all the solution sets of model (2.1) are non-negative for all $t \geq 0$. Here, the total population size $N(t)$ satisfies, $\frac{dN}{dt} = \Pi - \mu N$. So, $N(t) \to \frac{\Pi}{\mu}$, as $t \to$

Table 1 Definition of the parameters

Parameter	Description	Unit
Π	Recruitment rate of susceptible individuals	Individuals· time^{-1}
N	Total population size	Individuals
ϕ	Proportion that the newly recruited individuals are vaccinated	None
μ	Natural death rate of the population	Time^{-1}
a	Loss of pre-vaccination rate	Time^{-1}
β	Disease transmission rate from infectious individuals to susceptible	Time^{-1}
c	Number of successful contacts from asymptomatic to susceptible	none
ω	Vaccination rate given to the susceptible populations (post vaccination)	Time^{-1}
b	The rate at which exposed individuals join the infectious class	None
γ	Recovery rate of asymptomatic individuals	Time^{-1}
α	Recovery rate of infectious individuals	Time^{-1}
θ	Loss rate of vaccine induced immunity	Time^{-1}
ξ	Recovery rate of exposed individuals due to natural immunity	Time^{-1}
η	Per capita rate of becoming infectious	Time^{-1}
r	The rate at which the recovered individuals become susceptible again	Time^{-1}

∞. Therefore, the region of attraction for the model (2.1), where the system has biological and feasible meanings, is given by

$$\Gamma = \left\{(S, V_1, V_2, E, A, I, R) \in \mathbb{R}^7_+ : 0 \leq S + V_1 + V_2 + E + A + I + R \leq \frac{\Pi}{\mu}\right\}.$$

This is the positive invariant set because all solutions of the model (2.1) with initial conditions in Γ remain in Γ for all time $t > 0$. This proves the boundedness of system (2.1).

The Existence of Disease Free Equilibrium (DFE)

The disease free equilibrium exists if and only if $I = 0$. Hence, the unique disease free equilibrium is $\mathcal{E}_0 = (S^0, V_1^0, V_2^0, E^0, A^0, I^0, R^0)$, where

$$S^0 = \frac{(\theta+\mu)\Pi\{a+\mu(1-\phi)\}}{\mu(a+\mu)(\theta+\omega+\mu)}, \quad V_1^0 = \frac{\Pi\phi}{a+\mu}, \quad V_2^0 = \frac{\omega\Pi\{a+\mu(1-\phi)\}}{\mu(a+\mu)(\theta+\omega+\mu)},$$
$$E^0 = 0, \quad A^0 = 0, \quad I^0 = 0, \quad R^0 = 0.$$

The Existence of Endemic Equilibrium

The endemic equilibrium point exists and is unique only if the associated reproduction number $\mathcal{R}_v > 1$ and the details are given in "Existence of Endemic Equilibrium" appendix section.

The Associated Reproduction Number

Here, we derive the associated reproduction number (or control reproduction number) for the model system (2.1) by using Next-generation approach which is formulated in [7,39].

Let $m_1 = (\omega + \mu)$, $m_2 = (a + \mu)$, $m_3 = (\theta + \mu)$, $m_4 = (\xi + \eta + \mu)$, $m_5 = (\alpha + \mu)$, $m_6 = (\gamma + \mu)$, $m_7 = (r + \mu)$ and $x = (E, A, I)^T$, then model (2.1) follows that,

$$\frac{dx}{dt} = \mathcal{F} - \mathcal{V},$$

where

$$\mathcal{F} = \begin{pmatrix} \frac{\beta S(I+cA)}{1+I} \\ 0 \\ 0 \end{pmatrix} \quad \text{and} \quad \mathcal{V} = \begin{pmatrix} m_4 E \\ m_6 A - (1-b)\eta E \\ m_5 I - b\eta E \end{pmatrix}.$$

Now, we define $\mathbf{F}$ and $\mathbf{V}$ such that

$$\mathbf{F} = \text{Jacobian of } \mathcal{F} \; at \; DFE = \begin{pmatrix} 0 & \frac{\beta c S^0}{1+I^0} & \frac{\beta S^0(1-cA^0)}{(1+I^0)^2} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

and

$$\mathbf{V} = \text{Jacobian of } \mathcal{V} \; at \; DFE = \begin{pmatrix} m_4 & 0 & 0 \\ -(1-b)\eta & m_6 & 0 \\ -b\eta & 0 & m_5 \end{pmatrix}.$$

Hence, the Next-generation matrix is given by the matrix K, where

$$K = \mathbf{F}\mathbf{V}^{-1} = \begin{pmatrix} \frac{\beta c S^0(1-b)\eta}{m_4 m_6} + \frac{b\beta S^0 \eta}{m_4 m_5} & \frac{\beta c S^0}{m_6} & \frac{\beta S^0}{m_5} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}.$$

Now, the associated reproduction number of the model (2.1) is denoted by $\mathcal{R}_v$ and is given by the spectral radius of the next-generation matrix $K = \mathbf{F}\mathbf{V}^{-1}$ i.e., $\mathcal{R}_v = \rho(K)$. Therefore,

$$\mathcal{R}_v = \frac{b\eta\beta(\theta+\mu)\Pi\{a+\mu(1-\phi)\}}{\mu(\alpha+\mu)(a+\mu)(\theta+\omega+\mu)(\xi+\eta+\mu)}(\mathcal{R}_0+1),$$

where $\mathcal{R}_0 = \frac{c(\alpha+\mu)(1-b)}{b(\gamma+\mu)}$, denotes the basic reproduction number and $\mathcal{R}_v$ denotes control reproduction number for the model (2.1), which measures the average number of new infections generated by a typical infectious individual in a population, where a fraction of the susceptible individuals is vaccinated [1,9]. Both $\mathcal{R}_v$ and $\mathcal{R}_0$ are used to determine the severity of an endemic.

Stability Analysis of the Equilibria

Local Stability of DFE

For the local stability of the disease free equilibrium point, first we calculate the jacobian of model system (2.1) at the DFE, i.e.,

$$J(\mathcal{E}) = \begin{pmatrix} -\frac{\beta(I+cA)}{1+I} - (\omega+\mu) & a & \theta & 0 & \frac{\beta S(1-cA)}{(1+I)^2} & \frac{-\beta cS}{1+I} & r \\ 0 & -(a+\mu) & 0 & 0 & 0 & 0 & 0 \\ \omega & 0 & -(\theta+\mu) & \xi & 0 & 0 & 0 \\ \frac{\beta(I+cA)}{1+I} & 0 & 0 & -(\xi+\eta+\mu) & \frac{\beta S(1-cA)}{(1+I)^2} & \frac{\beta cS}{1+I} & 0 \\ 0 & 0 & 0 & b\eta & -(\alpha+\mu) & 0 & 0 \\ 0 & 0 & 0 & (1-b)\eta & 0 & -(\gamma+\mu) & 0 \\ 0 & 0 & 0 & 0 & \alpha & \gamma & -(r+\mu) \end{pmatrix},$$

which implies that

$$J(\mathcal{E}_0) = \begin{pmatrix} -(\omega+\mu) & a & \theta & 0 & \beta S^0 & -\beta cS^0 & r \\ 0 & -(a+\mu) & 0 & 0 & 0 & 0 & 0 \\ \omega & 0 & -(\theta+\mu) & \xi & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\xi+\eta+\mu) & \beta S^0 & \beta cS^0 & 0 \\ 0 & 0 & 0 & b\eta & -(\alpha+\mu) & 0 & 0 \\ 0 & 0 & 0 & (1-b)\eta & 0 & -(\gamma+\mu) & 0 \\ 0 & 0 & 0 & 0 & \alpha & \gamma & -(r+\mu) \end{pmatrix}.$$

Here, four eigenvalues of the above matrix are $-(a+\mu)$, $-(r+\mu)$, $-\mu$, $-(\omega+\mu+\theta)$ and the remaining eigenvalues are given by the following characteristic equation

$$\lambda^3 + c_1\lambda^2 + c_2\lambda + c_3 = 0,$$

where,

$$\begin{aligned} c_1 &= \xi+\eta+\alpha+\gamma+3\mu > 0, \\ c_2 &= (\alpha+\mu)(\xi+\eta+\mu) + (\gamma+\mu)(\xi+\eta+\alpha+2\mu) - b\beta\eta S^0 - (1-b)\beta\eta cS^0. \\ &= (\alpha+\mu)(\xi+\eta+\mu)\left(1-\frac{\mathcal{R}_v}{1+\mathcal{R}_0}\right) + (\gamma+\mu)(\alpha+\mu) \\ &\quad +(\gamma+\mu)(\xi+\eta+\mu)\left(1-\frac{\mathcal{R}_0\mathcal{R}_v}{1+\mathcal{R}_0}\right), \\ \text{and } c_3 &= (\gamma+\mu)\{(\alpha+\mu)(\xi+\eta+\mu) - b\beta\eta S^0\} - (1-b)\eta\beta c(\alpha+\mu)S^0. \\ &= (\gamma+\mu)(\alpha+\mu)(\xi+\eta+\mu)(1-\mathcal{R}_v). \end{aligned}$$

Now,

$$\begin{aligned} c_1c_2 - c_3 &= (\xi+\eta+\alpha+\gamma+3\mu)\left((\alpha+\mu)(\xi+\eta+\mu)\left(1-\frac{\mathcal{R}_v}{1+\mathcal{R}_0}\right) + (\gamma+\mu)(\alpha+\mu)\right. \\ &\quad \left. + (\gamma+\mu)(\xi+\eta+\mu)\left(1-\frac{\mathcal{R}_0\mathcal{R}_v}{1+\mathcal{R}_0}\right)\right) - (\gamma+\mu)(\alpha+\mu)(\xi+\eta+\mu)(1-\mathcal{R}_v) \\ &= (\xi+\eta+\mu)\left((\xi+\eta+\mu)(\alpha+\mu)\left(1-\frac{\mathcal{R}_v}{1+\mathcal{R}_0}\right) + (\alpha+\mu)^2\left(1-\frac{\mathcal{R}_v}{1+\mathcal{R}_0}\right)\right. \\ &\quad \left. +(\gamma+\mu)\left(1-\frac{\mathcal{R}_0\mathcal{R}_v}{1+\mathcal{R}_0}\right) + (\gamma+\mu)(\alpha+\mu)\left(\frac{\mathcal{R}_0\mathcal{R}_v}{1+\mathcal{R}_0}\right)\right) > 0. \end{aligned}$$

Clearly, $c_1c_2 - c_3 > 0$, if $\mathcal{R}_v \le \mathcal{R}_0$ $(0 < \mathcal{R}_0 \le 1)$. Hence, by Routh-Hurwitz criterion [7] the DFE $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_v \le 1$, otherwise it is unstable.

Global Stability of DFE

Here we will prove the global stability of disease free equilibrium using the method given in [40]. Since $\frac{dN}{dt} = \Pi - \mu N$. Then $N \to \frac{\Pi}{\mu}$ as $t \to \infty$. Taking the limiting case similar as discussed in [42], i.e., $N = \frac{\Pi}{\mu}$, then the system (2.1) reduces to

$$\frac{dV_1}{dt} = \Pi\phi - (a+\mu)V_1, \tag{4.1}$$

$$\frac{dV_2}{dt} = \omega\left(\frac{\Pi}{\mu} - (V_1 + E + I + A + R)\right) + \xi E - (\omega + \theta + \mu)V_2, \tag{4.2}$$

$$\frac{dE}{dt} = \frac{\beta(I + cA)}{(1+I)}\left(\frac{\Pi}{\mu} - (V_1 + V_2 + E + I + A + R)\right) - (\xi + \eta + \mu)E, \tag{4.3}$$

$$\frac{dI}{dt} = b\eta E - (\alpha + \mu)I, \tag{4.4}$$

$$\frac{dA}{dt} = (1-b)\eta E - (\gamma + \mu)A, \tag{4.5}$$

$$\frac{dR}{dt} = \alpha I + \gamma A - (r + \mu)R. \tag{4.6}$$

Let $X = (V_1, V_2, R)$ and $Z = (E, I, A)$. Here $U^0 = (X^0, Z^0)$, where $X^0 = \left(V_1^0, V_2^0, 0\right)$ and $Z^0 = (0, 0, 0)$. At $Z = Z^0$, $G(X, 0) = \left(V_1^0, V_2^0, 0\right)$. We have,

$$\begin{aligned}
\frac{dV_1}{dt} &= \Pi\phi - (a+\mu)V_1,\\
\frac{dV_2}{dt} &= \omega\left(\frac{\Pi}{\mu} - V_1 - R\right) - (\omega + \theta + \mu)V_2,\\
\frac{dR}{dt} &= -(r+\mu)R.
\end{aligned} \tag{4.7}$$

It is easy to show that, $V_1(t) \to V_1^0$ and $R(t) \to R^0$ as $t \to \infty$. From system (4.7), we have

$$\frac{dV_2}{dt} = \omega\left(\frac{\Pi}{\mu} - V_1 - R\right) - (\omega + \theta + \mu)V_2.$$

Which implies that,

$$\frac{dV_2}{dt} + (\omega + \theta + \mu)V_2 = \omega\left(\frac{\Pi}{\mu} - \frac{\Pi\phi}{a+\mu}\right) - \omega V_0 e^{-(a+\mu)t} - \omega R_0 e^{-(r+\mu)t}.$$

The solution of the above equation is given by

$$V_2(t) = c_1 e^{-(\omega+\theta+\mu)t} + \omega\left(\frac{\Pi}{\mu} - \frac{\Pi\phi}{a+\mu}\right) + \frac{1}{\omega+\theta-a}e^{-(a+\mu)t} + \frac{1}{\omega+\theta-r}e^{-(r+\mu)t},$$

where

$$c_1 = \omega\left(\frac{\Pi\phi}{a+\mu} - \frac{\Pi}{\mu}\right) - \frac{(2\omega + 2\theta - (a+r))}{(\omega+\theta-a)(\omega+\theta-r)}.$$

Clearly, $V_2(t) \to V^0$ as $t \to \infty$. Hence $X = X^0 (= V_1^0, V_2^0, R^0)$ is globally asymptotically stable. Thus, the condition $(H1)$ of [40] is satisfied. From Eqs. (4.3)–(4.5), we obtain

$$\frac{dZ}{dt} = G(X, Z) = PZ - \widetilde{Q}(X, Z),$$

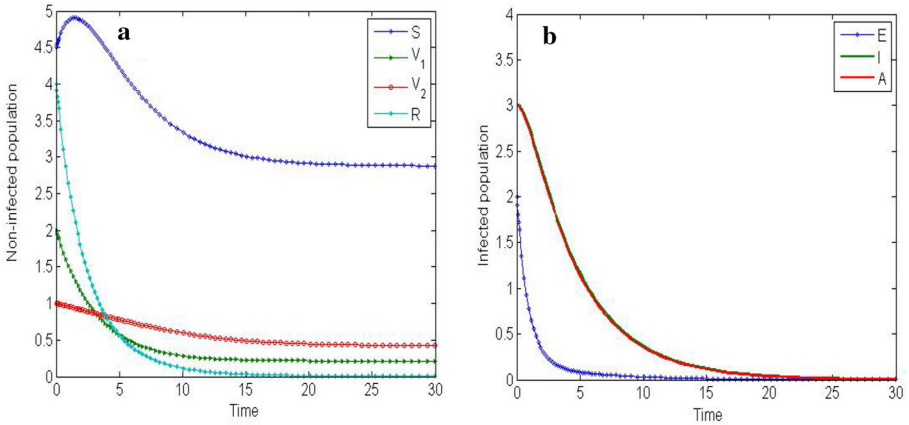

Fig. 2 Stability of disease free equilibrium $\mathcal{E}_0$, when $\mathcal{R}_v < 1$ and $\mathcal{R}_0 < 1$; **a** time series of non-infected population, **b** time series of infected population

where

$$P = \begin{pmatrix} -(\xi+\eta+\mu) & \frac{\beta S}{1+I} & \frac{\beta c S}{1+I} \\ b\eta & -(\alpha+\mu) & 0 \\ (1-b)\eta & 0 & -(\gamma+\mu) \end{pmatrix} \quad \text{and} \quad \widetilde{Q}(X,Z) = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}.$$

Hence, P is an M-matrix (since off diagonal elements of P are non-negative). Thus, both the conditions (H1) and (H2) of [40], are satisfied. Hence, the DFE is globally asymptotically stable if $\mathcal{R}_v \leq 1$.

Stability of Endemic Equilibrium

Theorem 4.1 *The endemic equilibrium $\mathcal{E}^*$ is locally asymptotically stable (or linearly stable) if the following inequalities are satisfied:*

$$L < \min\left\{\frac{2(l_1+\omega+\mu)}{21} + \frac{(\xi+\eta+\mu)}{24b\eta}, \frac{2(l_1+\omega+\mu)}{21} + \frac{b(\xi+\eta+\mu)}{6\eta}\right\} \tag{4.8}$$

$$\frac{21}{(l_1+\omega+\mu)} < \min\left\{M, \frac{3(\xi+\eta+\mu)}{b\eta l_1^2}\right\}, \tag{4.9}$$

$$\text{where } L = \frac{1701r^2}{64(r+\mu)^2(\alpha+\mu)^2(l_1+\omega+\mu)}, \quad l_1 = \frac{\beta(I^*+cA^*)}{(1+I^*)} \quad \text{and}$$

$$M = \frac{(\theta+\mu)^2\left(4(l_1+\omega+\mu)+7b\eta(\xi+\eta+\mu)\right)}{21\theta^2(\omega^2+\xi^2)}.$$

Proof The proof of theorem is given in "Local Stability of the Endemic Equilibrium" appendix section. □

Theorem 4.2 *The endemic equilibrium point $\mathcal{E}^*$ is non-linearly stable if the following inequalities are satisfied:*

$$\frac{45\beta(\mu+\Pi)}{2\mu(\xi+\eta+\mu)(1+I^*)} < \min\left\{\frac{cA^*}{I^*}, \frac{\mu^2A^*I^*}{c\Pi^2}, \frac{1}{cA^*I^*}\right\}, \tag{4.10}$$

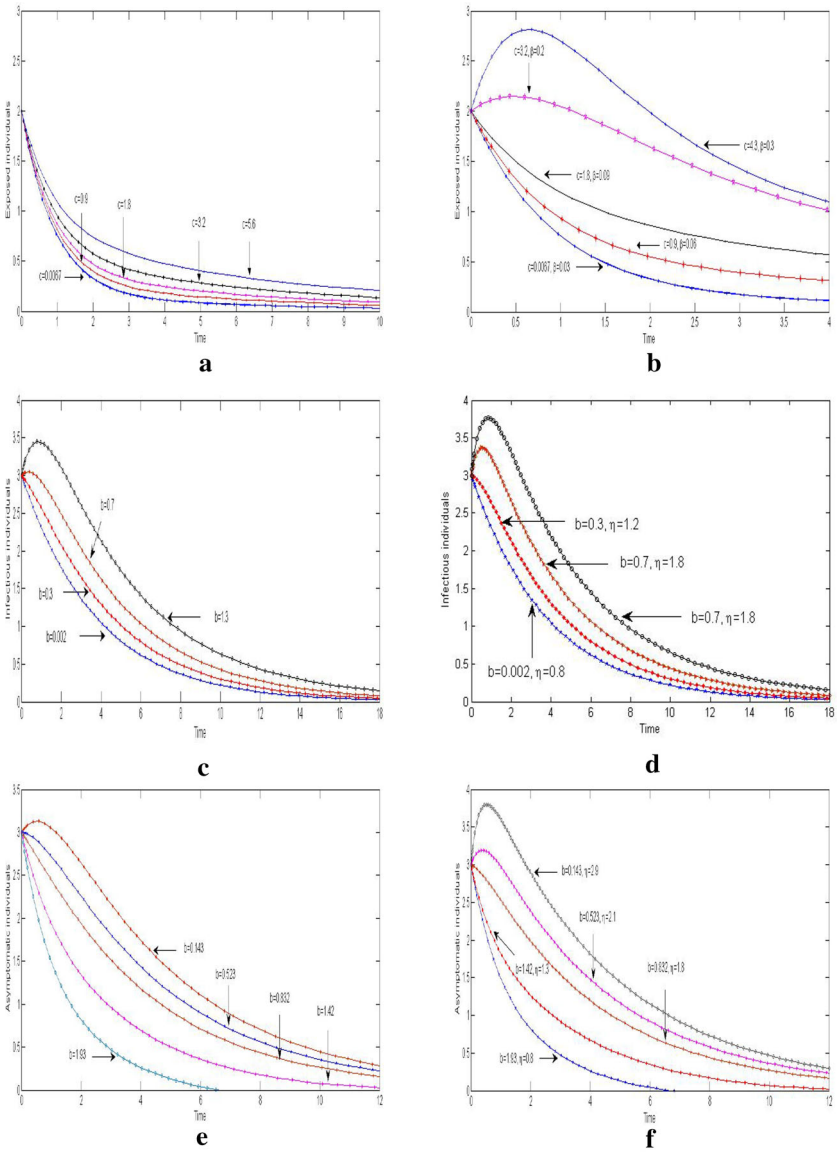

Fig. 3 Stability switching from disease free to endemic equilibrium (2.1). **a** Variation in E with respect to c. **b** Variation in E with respect to c and β. **c** Variation in I with respect to b. **d** Variation in I with respect to b and η. **e** Variation in A with respect to b. **f** Variation in A with respect to b and η

$$\frac{25\beta^2}{2(1+I^*)^2(\xi+\eta+\mu)^2} < \min\left\{\frac{2A^{*2}(\gamma+\mu)^2}{(45)^2(1-b)^2\eta^2S^{*2}}, \frac{\omega^2}{\xi^2I^{*2}}, \frac{\mu^2(\alpha+\mu)^2}{25b^2\eta^2\Pi^2}\right.$$
$$\left.\frac{(\gamma+\mu)^2}{25(1-b)^2\eta^2c^2S^{*2}}, \frac{\mu^2(\gamma+\mu)^2}{25(1-b)^2\eta^2c^2\Pi^2I^{*2}}\right\}, \tag{4.11}$$

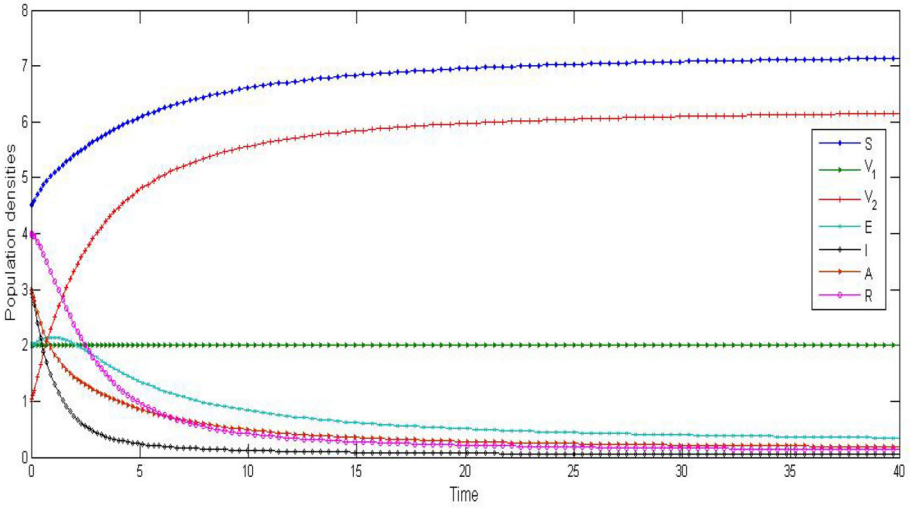

Fig. 4 Stability of endemic equilibrium $\mathcal{E}^*$, when $\mathcal{R}_v > 1$ and $\mathcal{R}_0 > 1$

$$\frac{27r^2(\mu+\Pi)(1+I^*)}{4\beta\mu cA^*I^*(r+\mu)} < p_6 < \min\left\{\frac{4(\alpha+\mu)^2(r+\mu)}{9\alpha^2}\left(\frac{2(l_1+\omega+\mu)}{21}+\frac{(\xi+\eta+\mu)}{24b\eta}\right),\right.$$
$$\left.\frac{4(\gamma+\mu)^2(r+\mu)}{9\gamma^2}\left(\frac{2(l_1+\omega+\mu)}{21}+\frac{b(\xi+\eta+\mu)}{6\eta}\right)\right\}. \quad (4.12)$$

Proof The proof of this theorem refers to "Global Stability of Endemic Equilibrium" appendix section. □

Numerical Simulations and Discussions

A deterministic *SVEIRS* epidemic model for the transmission dynamics of a disease such as measles and influenza, subject to a defective (or imperfect) vaccine with declining natural and vaccine-induced protection is analyzed, where both pre and post vaccinations are considered simultaneously in form of control strategies. The model (2.1) can be reduced to an *SVEIRS* epidemic model, when there is no pre-vaccination given to the susceptible and all infectious individuals show symptoms, i.e., $\phi = 0$ and $b = 1$. If all the individuals obtain permanent immunity, i.e., $r = 0$, then system (2.1) is reduced to an $SVEIR$ model. If we consider $\omega = 0$ and $b = 1$, the proposed model takes the form $SIRS$. Further, in the analysis, it is obtained that the system has two equilibrium points: disease free and endemic. The disease-free equilibrium of the system (2.1) is locally as well as globally asymptotically stable for $\mathcal{R}_v \leq 1$. If $\mathcal{R}_v > 1$, the endemic equilibrium is linearly stable under some certain conditions discussed in the theorem 4.1. The theorem 4.2 determined the sufficient conditions for nonlinear stability of $\mathcal{E}^*$. These analytical results of system (2.1) can be validated by performing some numerical simulation for the following set of parametric values:

$$c = 0.0067; \quad \alpha = 0.0312; \quad \mu = 0.232; \quad b = 0.523; \quad r = 0.25; \quad \eta = 0.8;$$
$$\beta = 0.03; \quad \theta = 0.0543; \quad \Pi = 0.815; \quad a = 0.09; \quad \phi = 0.0842; \omega = 0.042;$$
$$\gamma = 0.032; \quad \xi = 0.0321.$$

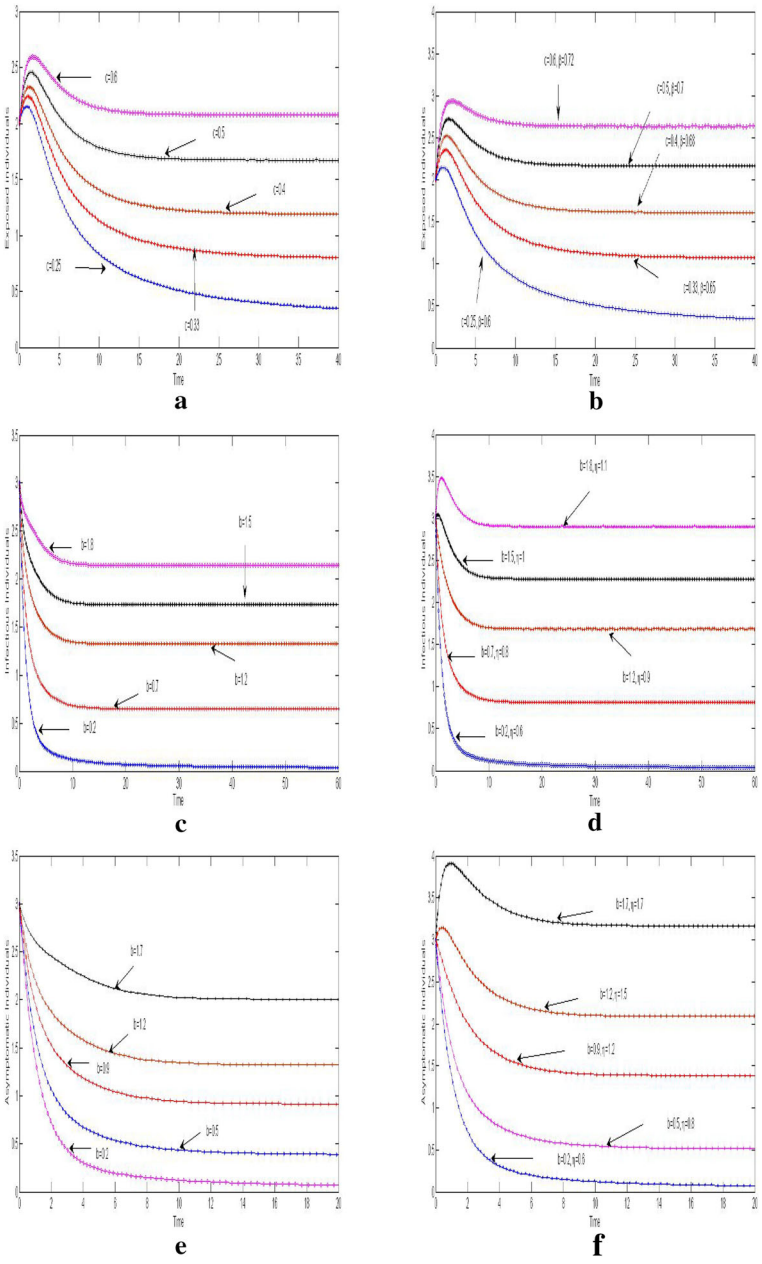

Fig. 5 Variation in E, I and A for $\mathcal{R}_0 > 1$ and $\mathcal{R}_v > 1$. **a** Variation in E with respect to c. **b** Variation in E with respect to c and β. **c** Variation in I with respect to b. **d** Variation in I with respect to b and η. **e** Variation in A with respect to b. **f** Variation in A with respect to b and η

Clearly, $\mathcal{R}_0 = 0.00609219 < 1$ and $\mathcal{R}_v = 0.129755 < 1$. Therefore, in this case, the number of infected (exposed, infectious, asymptomatic) and recovered individuals become extinct while susceptible, pre-vaccinated and post-vaccinated individuals will survive and hence the disease dies out from the population. The corresponding diagram is shown in Fig. 2.

The parameters c, β and b, η have a crucial effect on exposed, infected and asymptomatic population, respectively of system (2.1), which are responsible for switching of stability from disease free to endemic equilibrium. The corresponding diagram for the above set of parameters with different values of c, β and b, η is shown in the Fig. 3 :

On the other hand, if we choose following another set of parameters:

$$c = 0.25; \quad \alpha = 0.41; \quad \mu = 0.5; \quad b = 0.2; \quad r = 0.2; \quad \eta = 0.6; \quad \beta = 0.6; \quad \theta = 0.08;$$
$$\Pi = 8; \quad a = 0.3; \quad \phi = 0.2; \quad \omega = 0.5; \quad \gamma = 0.4; \quad \xi = 0.01,$$

then $\mathcal{R}_0 = 1.01111 > 1$ and $\mathcal{R}_v = 1.0778 > 1$. Unlike the previous case for $\mathcal{R}_v$ and $\mathcal{R}_0$, in this situation, all the individuals co-exist and hence, the disease will persist in the population. The corresponding diagram is shown in Fig. 4.

Similar to the previous case, if we change the value of the parameters like c, β, b, η then the value of state variables, e.g., E, I and A also vary with respect to time and stability switches from endemic equilibrium to disease free equilibrium, as shown in Fig. 5.

Since the basic reproduction number $\mathcal{R}_0$ is directly proportional to the successful number of contacts of asymptomatic individuals to susceptible (i.e., c), so if we make the small change in c, the value of $\mathcal{R}_0$ changes directly and hence the associated reproduction number $\mathcal{R}_v$ changes rapidly. Therefore, the parameter c plays a key role in the disease outbreak of the population. But when c is large, the disease eradication becomes a difficult task, since the associated reproduction number $\mathcal{R}_v$ will be greater than unity and hence the disease will persist in the population. Moreover, the disease spread can be minimized for small value of c. Therefore, the additional compartment, i.e., asymptomatic (A) is meaning full which contains all those individuals, which transmit the infection in the population. Hence, the number of contact of asymptomatic to susceptible c affects the dynamics of the population. Another most effective parameter in the model system (2.1) is b, which gives the number of infectious individuals with symptoms. For example, $b = 1$ gives $c = 0$, i.e., there are no such infectious individuals, which can make contact with susceptible. If $b = 0$, then all the infectious person belongs to the asymptomatic class. Also, when each infectious persons belongs to the infectious compartment, the person does not show the asymptotic behavior.

Hence, it is concluded that the pre-vaccination rate ϕ, control the associated reproduction number $\mathcal{R}_v$. If we increase the value to ϕ near about unity from the negative direction, then $\mathcal{R}_v$ may be less than or equal to one and if we take ϕ near about zero from the positive direction, then $\mathcal{R}_v$ may be greater than unity. If $\mathcal{R}_v > 1$, then the disease will persist in the population (which will depend on the selection of other parameters). The parameters b, ϕ and c can also control the outbreaks of disease, which are directly related to the asymptomatic compartment. Hence, the consideration of asymptomatic compartment is most important in transmission of several infectious diseases such as measles and influenza, which represents the more realistic situation in the region of consideration.

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Appendix A: Existence of Endemic Equilibrium

There exists a unique endemic equilibrium $\mathcal{E}^*(S^*, V_1^*, V_2^*, E^*, I^*, A^*, R^*)$, for the model system (2.1), where

$$E^* = \frac{(\alpha+\mu)I^*}{b\eta}, \quad A^* = \frac{\mathcal{R}_0 I^*}{c}, \quad R^* = \frac{(\alpha c + \gamma\mathcal{R}_0)I^*}{c(r+\mu)},$$
$$V_1^* = \frac{\Pi\phi}{(a+\mu)}, \quad S^* = \frac{(\xi+\eta+\mu)(\alpha+\mu)(1+I^*)}{b\eta\beta(1+\mathcal{R}_0)} \quad \text{and}$$
$$V_2^* = \frac{\omega(\xi+\eta+\mu)(\alpha+\mu)(1+I^*) + \xi\beta(\alpha+\mu)(1+\mathcal{R}_0)I^*}{b\eta\beta(\theta+\mu)(1+\mathcal{R}_0)}.$$

Since $E^* = \dfrac{\Pi}{\mu} - (S^* + V_1^* + V_2^* + I^* + A^* + R^*)$.
Substituting the values of E^*, S^*, V_1^*, V_2^*, A^* and R* in the above equation, we get

$$\begin{aligned}\frac{(\alpha+\mu)I^*}{b\eta} &= \frac{\Pi}{\mu} - \left(\frac{(\xi+\eta+\mu)(\alpha+\mu)(1+I^*)}{b\eta\beta(1+\mathcal{R}_0)} + \frac{\Pi\phi}{(a+\mu)}\right.\\ &\quad + \frac{\omega(\xi+\eta+\mu)(\alpha+\mu)(1+I^*) + \xi\beta(\alpha+\mu)(1+\mathcal{R}_0)I^*}{b\eta\beta(\theta+\mu)(1+\mathcal{R}_0)} + I^*\\ &\quad \left. + \frac{\mathcal{R}_0 I^*}{c} + \frac{(\alpha c+\gamma\mathcal{R}_0)I^*}{c(r+\mu)}\right).\\ &= \frac{\Pi}{\mu} - \left(\frac{(\xi+\eta+\mu)(\alpha+\mu)}{b\eta\beta(1+\mathcal{R}_0)} + \frac{\Pi\phi}{(a+\mu)} + \frac{\omega(\xi+\eta+\mu)(\alpha+\mu)}{b\eta\beta(\theta+\mu)(1+\mathcal{R}_0)}\right.\\ &\quad + \left(\frac{(\xi+\eta+\mu)(\alpha+\mu)}{b\eta\beta(1+\mathcal{R}_0)} + \frac{\omega(\xi+\eta+\mu)(\alpha+\mu) + \xi\beta(\alpha+\mu)(1+\mathcal{R}_0)}{b\beta\eta(\theta+\mu)(1+\mathcal{R}_0)}\right.\\ &\quad \left.\left. + \frac{(r+\mu)(c+\mathcal{R}_0) + (\alpha c+\gamma\mathcal{R}_0)}{c(r+\mu)}\right) I^*\right).\end{aligned}$$

This gives,

$$\begin{aligned}&I^*\left(\frac{(\alpha+\mu)}{b\eta} + \frac{\xi c(\alpha+\mu)(r+\mu) + b\eta(\theta+\mu)((r+\mu)(c+\mathcal{R}_0) + (\alpha c+\gamma\mathcal{R}_0))}{bc\eta(\theta+\mu)(r+\mu)}\right.\\ &\quad \left. + \frac{(\xi+\eta+\mu)(\alpha+\mu)(\omega+\theta+\mu)}{b\eta\beta(\theta+\mu)(1+\mathcal{R}_0)}\right)\\ &= \frac{\Pi}{\mu} - \left(\frac{(\xi+\eta+\mu)(\alpha+\mu)(\omega+\theta+\mu)}{b\eta\beta(\theta+\mu)(1+\mathcal{R}_0)} + \frac{\Pi\phi}{(a+\mu)}\right).\end{aligned}$$

Hence, we get

$$I^* = \frac{c(r+\mu)(\alpha+\mu)(\xi+\eta+\mu)(\omega+\theta+\mu)(\mathcal{R}_v-1)}{\mathcal{P}},$$

where

$$\begin{aligned}\mathcal{P} &= \beta c(\alpha+\mu)(1+\mathcal{R}_0)(r+\mu)(\xi+\theta+\mu) + c(r+\mu)(\xi+\eta+\mu)(\alpha+\mu)(\omega+\theta+\mu)\\ &\quad + b\eta\beta(1+\mathcal{R}_0)(\theta+\mu)(c(r+\mu) + \mathcal{R}_0(r+\mu) + (\alpha c+\gamma\mathcal{R}_0)) > 0.\end{aligned}$$

Hence, the endemic equilibrium point $\mathcal{E}^* = (S^*, V_1^*, V_2^*, E^*, I^*, A^*, R^*)$ exists only if $\mathcal{R}_v > 1$.

Appendix B: Local Stability of the Endemic Equilibrium

To find the local stability of endemic equilibrium $\mathcal{E}^*$, we consider the following positive definite function,

$$Z = \frac{1}{2}s_1^2 + \frac{q_1}{2}v_1^2 + \frac{q_2}{2}v_2^2 + \frac{q_3}{2}e_1^2 + \frac{q_4}{2}i_1^2 + \frac{q_5}{2}a_1^2 + \frac{q_6}{2}r_1^2,$$

where $q_1,\ q_2,\ q_3,\ q_4,\ q_5, q_6$ are positive constants to be chosen appropriately and s, v_1, v_2, e, i, a, r are the small perturbations in S, V_1, V_2, E, I, A, R respectively. Therefore, we can write $S = S^* + s_1$, $V_1 = V_1^* + v_1$, $V_2 = V_2^* + v_2$, $E = E^* + e_1$, $I = I^* + i_1$, $A = A^* + a_1$, and $R = R^* + r_1$. Now, differentiating 'Z' with respect to time 't', we get

$$\frac{dZ}{dt} = s_1\frac{ds_1}{dt} + q_1v_1\frac{dv_1}{dt} + q_2v_2\frac{dv_2}{dt} + q_3e_1\frac{de_1}{dt} + q_4i_1\frac{di_1}{dt} + q_5a_1\frac{da_1}{dt} + q_6r_1\frac{dr_1}{dt}.$$

Now, using the linearized system of model (2.1) corresponding to endemic equilibrium point $\mathcal{E}^*$, we get

$$\begin{aligned}\frac{dZ}{dt} = {} & s_1\left(av_1 - \frac{\beta S^*(1-cA^*)}{(1+I^*)^2}i_1 - \frac{\beta cS^*}{(1+I^*)}a_1 - \frac{\beta(I^*+cA^*)}{(1+I^*)}s_1 + \theta v_2 + rr_1 - (\omega+\mu)s_1\right)\\ & + q_1v_1\left(-(a+\mu)v_1\right) + q_2v_2\left(\omega s_1 + \xi e_1 - (\theta+\mu)v_2\right)\\ & + q_3e_1\left(\frac{\beta S^*(1-cA^*)}{(1+I^*)^2}i_1 + \frac{\beta cS^*}{(1+I^*)}a_1 + \frac{\beta(I^*+cA^*)}{(1+I^*)}s_1 - (\xi+\eta+\mu)e_1\right)\\ & + q_4i_1\left((b\eta)e_1 - (\alpha+\mu)i_1\right) + q_5a_1\left((1-b)\eta e_1 - (\gamma+\mu)a_1\right)\\ & + q_6r_1\left(\alpha i_1 + \gamma a_1 - (r+\mu)r_1\right).\end{aligned}$$

This implies that,

$$\begin{aligned}\frac{dZ}{dt} = {} & -\left(\frac{\beta(I^*+cA^*)}{(1+I^*)} + (\omega+\mu)\right)s_1^2 - q_1(a+\mu)v_1^2 - q_2(\theta+\mu)v_2^2 - q_3(\xi+\eta+\mu)e_1^2\\ & - q_4(\alpha+\mu)i_1^2 - q_5(\gamma+\mu)a_1^2 - q_6(r+\mu)r_1^2 + (a)s_1v_1 + (\theta)s_1v_2 + q_2(\omega)s_1v_2\\ & + q_3\left(\frac{\beta(I^*+cA^*)}{(1+I^*)}\right)s_1e_1 - \left(\frac{\beta S^*(1-cA^*)}{(1+I^*)^2}\right)s_1i_1 - \left(\frac{\beta cS^*}{(1+I^*)}\right)s_1a_1 + (r)r_1s_1\\ & + q_2(\xi)v_2e_1 + q_3\left(\frac{\beta S^*(1-cA^*)}{(1+I^*)^2}\right)e_1i_1 + q_4(b\eta)e_1i_1 + q_3\left(\frac{\beta cS^*}{(1+I^*)}\right)e_1a_1\\ & + q_5((1-b)\eta)e_1a_1 + q_6(\alpha)i_1r_1 + q_6(\gamma)a_1r_1.\end{aligned}$$

Let $q_3 = b\eta$, $q_4 = \dfrac{\beta S^*(1-cA^*)}{(1+I^*)^2} = l_2$, $q_5 = \dfrac{\beta cS^*}{(1+I^*)} = l_3$ and $l_1 = \dfrac{\beta(I^*+cA^*)}{(1+I^*)}$. Then the above equation reduces to

$$\begin{aligned}\frac{dZ}{dt} = {} & -(l_1+\omega+\mu)s_1^2 - q_1(a+\mu)v_1^2 - q_2(\theta+\mu)v_2^2 - b\eta(\xi+\eta+\mu)e_1^2 - l_2(\alpha+\mu)i_1^2\\ & - l_3(\gamma+\mu)a_1^2 - q_6(r+\mu)r_1^2 + (a)v_1s_1 + (\theta)s_1v_2 + q_2(\omega)s_1v_2 + (b\eta l_1)e_1s_1\\ & - (l_2)s_1i_1 - (l_3)a_1s_1 + (r)r_1s_1 + q_2(\xi)v_2e_1 + 2(l_2b\eta)e_1i_1 + (l_3\eta)e_1a_1 + q_6(\alpha)i_1r_1\\ & + q_6(\gamma)a_1r_1.\end{aligned}$$

Now, $\frac{dZ}{dt}$ will be a negative definite function provided that the following inequalities are satisfied,

$$a^2 < \frac{4q_1(a+\mu)(l_1+\omega+\mu)}{7} \tag{B.1}$$

$$\theta^2 < \frac{4q_2(\theta+\mu)(l_1+\omega+\mu)}{21} \tag{B.2}$$

$$q_2(\omega^2+\xi^2) < \left(\frac{4(l_1+\omega+\mu)+7b\eta(\xi+\eta+\mu)}{21}\right)(\theta+\mu) \tag{B.3}$$

$$b\eta l_1^2 < \frac{(l_1+\omega+\mu)(\xi+\eta+\mu)}{7} \tag{B.4}$$

$$l_2 < \left(\frac{2(l_1+\omega+\mu)}{21}+\frac{(\xi+\eta+\mu)}{24b\eta}\right)(\alpha+\mu) \tag{B.5}$$

$$l_3 < \left(\frac{2(l_1+\omega+\mu)}{21}+\frac{b(\xi+\eta+\mu)}{6\eta}\right)(\gamma+\mu) \tag{B.6}$$

$$r^2 < \frac{4q_6(r+\mu)(l_1+\omega+\mu)}{21} \tag{B.7}$$

$$q_6\alpha^2 < \frac{4l_2(\alpha+\mu)(r+\mu)}{9} \tag{B.8}$$

$$q_6\gamma^2 < \frac{4l_3(\gamma+\mu)(r+\mu)}{9} \tag{B.9}$$

We can select q_1 and q_2 from inequality (B.1) and (B.3) respectively, i.e., $q_1 = \frac{7a^2}{2(a+\mu)(l_1+\omega+\mu)}$ and $q_2 = \left(\frac{4(l_1+\omega+\mu)+7b\eta(\xi+\eta+\mu)}{21}\right)\cdot\frac{(\theta+\mu)}{4(\omega^2+\xi^2)}$. Hence, inequality (B.2) reduces to

$$\theta^2 < \frac{(\theta+\mu)^2(l_1+\omega+\mu)}{441(\omega^2+\xi^2)}(4(l_1+\omega+\mu)+7b\eta(\xi+\eta+\mu)). \tag{B.10}$$

Inequalities (B.5) and (B.8) gives,

$$q_6\alpha^2 < \frac{4(\alpha+\mu)^2(r+\mu)}{9}\left(\frac{2(l_1+\omega+\mu)}{21}+\frac{(\xi+\eta+\mu)}{24b\eta}\right). \tag{B.11}$$

Similarly, inequalities (B.6) and (B.9), gives

$$q_6\gamma^2 < \frac{4(\gamma+\mu)^2(r+\mu)}{9}\left(\frac{2(l_1+\omega+\mu)}{21}+\frac{b(\xi+\eta+\mu)}{6\eta}\right). \tag{B.12}$$

From inequalities (B.11) and (B.12), we have

$$q_6 < \min\left\{\frac{4(\alpha+\mu)^2(r+\mu)}{9\alpha^2}\left(\frac{2(l_1+\omega+\mu)}{21}+\frac{(\xi+\eta+\mu)}{24b\eta}\right),\right.$$
$$\left.\frac{4(\gamma+\mu)^2(r+\mu)}{9\gamma^2}\left(\frac{2(l_1+\omega+\mu)}{21}+\frac{b(\xi+\eta+\mu)}{6\eta}\right)\right\}. \tag{B.13}$$

Hence, we can select q_6 from above inequality such that (4.8) holds.

Appendix C: Global Stability of Endemic Equilibrium

To obtain the global stability of endemic equilibrium, we consider the following positive definite function

$$V = \frac{1}{2}(S-S^*)^2 + \frac{p_1}{2}(V_1-V_1^*)^2 + \frac{p_2}{2}(V_2-V_2^*)^2 + \frac{p_3}{2}(E-E^*)^2 + \frac{p_4}{2}(I-I^*)^2 + \frac{p_5}{2}(A-A^*)^2 + \frac{p_6}{2}(R-R^*)^2,$$

where p_1, p_2, p_3, p_4, p_5 and p_6 are positive constants to be chosen appropriately. Now, differentiating 'V' with respect to 't' along the solution of model (2.1) and after simple algebraic manipulations, we get

$$\begin{aligned}\frac{dV}{dt} < & -(\omega+\mu)(S-S^*)^2 - \frac{\beta I^*(S-S^*)^2}{(1+I^*)} - \frac{\beta\mu c A^* I^*(S-S^*)^2}{(\mu+\Pi)(1+I^*)} - p_1(a+\mu)(V_1-V_1^*)^2 \\ & -p_2(\theta+\mu)(V_2-V_2^*)^2 - p_3(\xi+\eta+\mu)(E-E^*)^2 - p_4(\alpha+\mu)(I-I^*)^2 \\ & -p_5(\gamma+\mu)(A-A^*)^2 - p_6(r+\mu)(R-R^*)^2 + a(S-S^*)(V_1-V_1^*) \\ & +\theta(S-S^*)(V_2-V_2^*) + p_2\omega(S-S^*)(V_2-V_2^*) + \frac{\beta I^*(S-S^*)(E-E^*)}{(1+I^*)}p_3 \\ & +\frac{\beta c\Pi(S-S^*)(E-E^*)}{\mu(1+I^*)}p_3 + \frac{\beta c A^* I^*(S-S^*)(E-E^*)}{(1+I^*)}p_3 - \frac{\beta\mu c S^*(A-A^*)(S-S^*)}{(\mu+\Pi)(1+I^*)} \\ & +r(S-S^*)(R-R^*) + p_2\xi(V_2-V_2^*)(E-E^*) + \frac{\beta\Pi(I-I^*)(E-E^*)}{\mu(1+I^*)}p_3 \\ & +\frac{\beta c S^*(A-A^*)(E-E^*)}{(1+I^*)}p_3 + \frac{\beta c\Pi I^*(A-A^*)(E-E^*)}{\mu(1+I^*)}p_3 + p_4 b\eta(E-E^*)(I-I^*) \\ & +p_5(1-b)\eta(E-E^*)(A-A^*) + p_6\alpha(I-I^*)(R-R^*) + p_6\gamma(R-R^*)(A-A^*) \\ & +\frac{\beta c A^* S^*(S-S^*)(I-I^*)}{(1+I^*)} - \frac{\beta c\mu A^* S^*(I-I^*)(E-E^*)}{(\mu+\Pi)(1+I^*)}p_3.\end{aligned}$$

For simplicity to analyze this equation we can remove first two terms from the equation. Hence, we obtain

$$\begin{aligned}\frac{dV}{dt} < & -\frac{\beta\mu c A^* I^*(S-S^*)^2}{(\mu+\Pi)(1+I^*)} - p_1(a+\mu)(V_1-V_1^*)^2 - p_2(\theta+\mu)(V_2-V_2^*)^2 \\ & -p_3(\xi+\eta+\mu)(E-E^*)^2 - p_4(\alpha+\mu)(I-I^*)^2 - p_5(\gamma+\mu)(A-A^*)^2 \\ & -p_6(r+\mu)(R-R^*)^2 + a(S-S^*)(V_1-V_1^*) + \theta(S-S^*)(V_2-V_2^*) \\ & +p_2\omega(S-S^*)(V_2-V_2^*) + \frac{\beta I^*(S-S^*)(E-E^*)}{(1+I^*)}p_3 + \frac{\beta c\Pi(S-S^*)(E-E^*)}{\mu(1+I^*)}p_3 \\ & +\frac{\beta c A^* I^*(S-S^*)(E-E^*)}{(1+I^*)}p_3 - \frac{\beta\mu c S^*(A-A^*)(S-S^*)}{(\mu+\Pi)(1+I^*)} + r(S-S^*)(R-R^*) \\ & +p_2\xi(V_2-V_2^*)(E-E^*) + \frac{\beta\Pi(I-I^*)(E-E^*)}{\mu(1+I^*)}p_3 + \frac{\beta c S^*(A-A^*)(E-E^*)}{(1+I^*)}p_3 \\ & +\frac{\beta c\Pi I^*(A-A^*)(E-E^*)}{\mu(1+I^*)}p_3 + p_4 b\eta(E-E^*)(I-I^*) \\ & +p_5(1-b)\eta(E-E^*)(A-A^*) + p_6\alpha(I-I^*)(R-R^*) + p_6\gamma(R-R^*)(A-A^*) \\ & +\frac{\beta c A^* S^*(S-S^*)(I-I^*)}{(1+I^*)} - \frac{\beta c\mu A^* S^*(I-I^*)(E-E^*)}{(\mu+\Pi)(1+I^*)}p_3.\end{aligned}$$

Now, $\frac{dV}{dt}$ will be a negative definite function provided that the following inequalities are satisfied,

$$\theta^2 < \frac{4p_2\beta\mu cA^*I^*(\theta+\mu)}{27(\mu+\Pi)(1+I^*)} \tag{C.1}$$

$$a^2 < \frac{4p_1\beta\mu cA^*I^*(a+\mu)}{9(\mu+\Pi)(1+I^*)} \tag{C.2}$$

$$p_2\omega^2 < \frac{4\beta\mu cA^*I^*(\theta+\mu)}{27(\mu+\Pi)(1+I^*)} \tag{C.3}$$

$$\frac{p_3\beta I^*}{(1+I^*)} < \frac{2\mu cA^*(\xi+\eta+\mu)}{45(\mu+\Pi)} \tag{C.4}$$

$$\frac{p_3\beta c\Pi^2}{\mu^2(1+I^*)} < \frac{2\mu A^*I^*(\xi+\eta+\mu)}{45(\mu+\Pi)} \tag{C.5}$$

$$\frac{p_3\beta cA^*I^*}{(1+I^*)} < \frac{2\mu(\xi+\eta+\mu)}{45(\mu+\Pi)} \tag{C.6}$$

$$\frac{\beta\mu cS^{*2}}{(\mu+\Pi)(1+I^*)} < \frac{4p_5A^*I^*(\gamma+\mu)}{45} \tag{C.7}$$

$$r^2 < \frac{4p_6\beta\mu cA^*I^*(r+\mu)}{27(\mu+\Pi)(1+I^*)} \tag{C.8}$$

$$p_2\xi^2 < \frac{2p_3(\xi+\eta+\mu)(\theta+\mu)}{15} \tag{C.9}$$

$$\frac{p_3\beta^2\Pi^2}{\mu^2(1+I^*)^2} < \frac{2p_4(\alpha+\mu)(\xi+\eta+\mu)}{25} \tag{C.10}$$

$$\frac{p_3\beta^2c^2S^{*2}}{(1+I^*)^2} < \frac{2p_5(\gamma+\mu)(\xi+\eta+\mu)}{25} \tag{C.11}$$

$$\frac{p_3\beta^2c^2\Pi^2I^{*2}}{\mu^2(1+I^*)^2} < \frac{2p_5(\gamma+\mu)(\xi+\eta+\mu)}{25} \tag{C.12}$$

$$p_4b^2\eta^2 < \frac{2p_3(\alpha+\mu)(\xi+\eta+\mu)}{25} \tag{C.13}$$

$$p_5(1-b)^2\eta^2 < \frac{2p_3(\gamma+\mu)(\xi+\eta+\mu)}{25} \tag{C.14}$$

$$p_6\alpha^2 < \frac{4p_4(\alpha+\mu)(r+\mu)}{15} \tag{C.15}$$

$$p_6\gamma^2 < \frac{4p_5(\gamma+\mu)(r+\mu)}{15} \tag{C.16}$$

$$\frac{\beta^2\mu^2c^2A^{*2}S^{*2}p_3}{(\mu+\Pi)^2(1+I^*)^2} < \frac{2p_4(\alpha+\mu)(\xi+\eta+\mu)}{25} \tag{C.17}$$

$$\frac{\beta cA^*S^{*2}}{(1+I^*)} < \frac{4\mu I^*p_4(\alpha+\mu)}{45(\mu+\Pi)} \tag{C.18}$$

From inequalities (C.2) and (C.3) we can select p_1 and p_2 respectively such that $p_1 = \frac{9a^2(\mu+\Pi)(1+I^*)}{2\beta\mu cA^*I^*(a+\mu)}$ and $p_2 = \frac{\beta\mu cA^*I^*(\theta+\mu)}{27\omega^2(\mu+\Pi)(1+I^*)}$. So, inequality (C.1), gives

$$\theta^2 < \frac{4(\beta\mu cA^*I^*)^2(\theta+\mu)^2}{(27)^2\omega^2(\mu+\Pi)^2(1+I^*)^2} \tag{C.19}$$

From inequalities (C.4), (C.5) and (C.6) we can select p_3, such that (4.10) holds.

In particular, if we take $p_3 = \dfrac{\mu c A^*(1+I^*)(\xi+\eta+\mu)}{45\beta I^*(\mu+\Pi)}$, then from inequalities (C.13) and (C.14), we can select p_4 and p_5 respectively such that

$$p_4 = \frac{\mu c A^*(\alpha+\mu)(1+I^*)(\xi+\eta+\mu)^2}{25\times 45 b^2\eta^2\beta I^*(\mu+\Pi)} \quad \text{and} \quad p_5 = \frac{\mu c A^*(\gamma+\mu)(1+I^*)(\xi+\eta+\mu)^2}{25\times 45\beta I^*(\mu+\Pi)(1-b)^2\eta^2}.$$

Now, using the value of p_5 in (C.7), p_2 & p_3 in (C.9), p_3 & p_4 in (C.10) and p_3 & p_5 in both (C.11) and (C.12), then respectively we have

$$\frac{\beta^2 S^{*2}}{(1+I^*)} < \frac{4A^{*2}(\gamma+\mu)^2(1+I^*)(\xi+\eta+\mu)^2}{25\times(45)^2(1-b)^2\eta^2}, \tag{C.20}$$

$$\frac{\beta^2\xi^2 I^{*2}}{27\omega^2(1+I^*)} < \frac{2(\xi+\eta+\mu)^2(1+I^*)}{15\times 45}, \tag{C.21}$$

$$\frac{\beta^2\Pi^2}{\mu^2(1+I^*)} < \frac{2(\alpha+\mu)^2(1+I^*)(\xi+\eta+\mu)^2}{(25)^2 b^2\eta^2}, \tag{C.22}$$

$$\frac{\beta^2 c^2 S^{*2}}{(1+I^*)} < \frac{2(\gamma+\mu)^2(1+I^*)(\xi+\eta+\mu)^2}{(25)^2(1-b)^2\eta^2}, \tag{C.23}$$

$$\frac{\beta^2 c^2\Pi^2 I^{*2}}{\mu^2(1+I^*)} < \frac{2(\gamma+\mu)^2(1+I^*)(\xi+\eta+\mu)^2}{(25)^2(1-b)^2\eta^2}. \tag{C.24}$$

From (C.15) and (C.16), we obtain

$$p_6 < \frac{4\mu c A^*(\alpha+\mu)^2(\xi+\eta+\mu)^2(r+\mu)(1+I^*)}{15\times 25\times 45\beta I^*\alpha^2 b^2\eta^2(\mu+\Pi)}, \tag{C.25}$$

$$p_6 < \frac{4\mu c A^*(\gamma+\mu)^2(\xi+\eta+\mu)^2(r+\mu)(1+I^*)}{15\times 25\times 45\beta I^*\gamma^2\eta^2(\mu+\Pi)(1-b)^2}, \tag{C.26}$$

respectively. Hence, we can select p_6 such that the inequality (4.12) holds.

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