



Study of a SIR Model with Vaccination and Treatment under an Imprecise Environment

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Abstract

Presently, compartmental epidemic models are widely employed to analyze the dynamic behavior of infectious diseases and to propose effective preventive and control measures. However, many studies have been done on fixed or precisely defined parameter values, which may not accurately represent real-world scenarios. In real situations, model parameters often vary due to factors such as changes in human immunity, the genomes of disease pathogens, climatic fluctuations, situation-dependent treatment, and varying vaccination rates. Such a changing nature of parameter values is known as impreciseness in parameter values. To study the effect of the changing nature of parameter values on disease dynamics, we have developed and studied a *SIR* (Susceptible-Infected-Recovered class) model incorporating both vaccination and treatment controls within an environment of interval imprecision. To address parameter uncertainty within intervals, we have proposed a linear parametric approach for modeling interval-valued parameters. We have rigorously investigated the positivity and boundedness of model solutions and determined the basic reproduction number $R_0(s)$ using the next-generation matrix approach, where s represents the interval parameter. Furthermore, we have analyzed the local stability, global stability, and bifurcation of equilibrium points of the developed system. Our findings reveal that when values of s are low, indicating low levels of disease transmission rate, the result is a nearly disease-free system where neither vaccination nor treatment control is necessary. Additionally, we have demonstrated that relying solely on either vaccination or treatment is insufficient to control the epidemic. Through our analysis, we have identified the necessity for a comprehensive control policy for the disease.

Keywords: Epidemic model, Stability analysis, Bifurcation, Sensitivity analysis, Optimal control

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1. INTRODUCTION

Following the groundbreaking work of Kermack and McKendrick [1], the literature on SIR models has flourished, finding applications in a wide array of infectious diseases [2–5]. This expansion has seen the compartmental structure of the SIR model evolve across different domains, introducing additional compartments such as SIRV [6], SIQR [7], SEIR [8, 9], and SEIRV [10, 11], among others [12–14]. Despite this progress, many of these models rely on fixed parameters, which may not accurately reflect real-world dynamics [15–18]. This raises the pivotal question for epidemiologists: which parameters of the SIR model warrant interval assumptions rather than fixed values? Among these, the transmission rate, pre-exposure vaccination rate, and post-exposure treatment rate emerge as crucial factors, where interval uncertainty plays a vital role in defining the disease-free system.

Imprecision in epidemic models stems from several sources, including the dynamic nature of human immunity, shifts in climate, and evolving human behaviors, including awareness. Climate and weather play significant roles in the survival,

reproduction, and transmission of disease pathogens, vectors, and hosts [19]. Changes in these environmental conditions can influence infectious diseases by altering the habitats and behaviors of organisms. Research suggests that prolonged climate warming often leads to the spread of various infectious diseases across different regions, while extreme weather events can trigger localized outbreaks in unexpected locations and times [20]. For instance, warmer temperatures tend to escalate disease transmission rates and outbreak intensities [21]. Additionally, Das and Pal [22] noted the challenge of precisely determining epidemiological parameters but highlighted the feasibility of establishing parameter ranges.

Imprecise parameters can take various forms, such as fuzzy imprecise or interval imprecise settings. The exploration of compartmental models with interval uncertainty in parameters was largely overlooked until Das and Pal made a seminal contribution [22]. Das and Pal introduced interval uncertainty into SIR model parameters by integrating fuzzified and defuzzified functions. Mahato et al. [23] investigated different solution strategies for tackling epidemic models within both types of

imprecise environments. However, a drawback of this approach is the inherent fuzziness in the parameters of interest and the complexity it introduces to solving the dynamical system.

We construct a SIR model within an environment of interval imprecision, integrating both vaccination and treatment control strategies. To determine whether a system is disease-free or disease-prone, we identify a fixed parameter value (s). Given the challenge of handling parameters with interval uncertainty, we suggest employing a linear parametric approach for interval-valued model parameters, inspired by Ramezanadeh et al. [24]. Notably, the functional response of disease transmission exhibits diverse forms. Gaff and Schaefer [25] address disease transmission functional response using the standard incidence form for diseases with permanent recovery. Conversely, Yusuf and Benyah [26] propose a model applicable to permanent recovery diseases (such as Measles, Influenza, Cholera, and Tuberculosis), where the disease transmission functional response adopts a linear form. For our model of permanent recovery disease, we assume both the transmission rate and the recovery rate to be linear.

Furthermore, treatment functions play a crucial role in curbing the spread of diseases. Various types of treatment functions have been suggested, including fixed, piecewise linear, quadratic, and saturated functions [27–30]. The selection of the appropriate function depends on the unique characteristics of the disease and its treatment. In this investigation, we delve into an imprecise linear treatment rate to accommodate uncertainties in vaccination parameters, acknowledging the reciprocal influence between vaccination and treatment uncertainties.

Therefore, the primary aim of this study is to determine whether an increase in disease transmission rate invariably leads to a delay in achieving a disease-free state when interval uncertainty is considered for the three aforementioned parameters such as the transmission rate, pre-exposure vaccination rate, and post-exposure treatment rate. Additionally, we emphasize the importance of identifying optimal control strategies for disease eradication through vaccination and treatment. Previous research has employed various control measures, including vaccination and treatment, to combat vector-borne diseases [30, 32, 33, 41] such as dengue, malaria, Zika, and plague. While some studies focus solely on vaccination [34–36] or treatment control [37–39], others integrate both approaches [40–43]. Furthermore, insecticide control is commonly utilized to reduce vector populations for such diseases [44]. In this study, we implement control measures for both vaccination and treatment.

The paper's structure is organized as follows: In Section 2, we introduce the fundamental concepts of interval impreciseness and its mathematical foundations. Section 3 outlines the formulation of the proposed model within both precise and imprecise environments. Moving to Section 4, we explore the positivity and boundedness of the solution for the proposed system, accompanied by a comprehensive stability analysis of the model. Section 5 investigates the local bifurcation surrounding the disease-free equilibrium point and the forward bifurcation around the endemic equilibrium point. Following this, Section 6 conducts a numerical sensitivity analysis of parameters. Section 7 encompasses the numerical solution of the model using a hypothetical set of parameters, accompanied by a discussion on the effects of parameter variation on the model's dynamic behavior. Subsequently, optimal control strategies are introduced and their impact on the dynamical behavior of the model is discussed. Finally, conclusions are drawn in Section 8.

2. Basic Concept of Interval Impreciseness: Interval Arithmetic

In this section, we have focused on some basic concepts of interval impreciseness including interval-valued function, and interval differential equation that will be employed to explore our model. In study of epidemic models, when some model parameters have no fixed value or belong to a suitable interval then such parameters will be called interval imprecise parameters. The explicit definition of interval-valued function, and interval differential equations are given below:

i. Interval-valued function: The interval-valued function is a function whose functional values are not fixed but range in intervals [24, 45].

ii. Interval differential equation: Interval differential equations are those equations that involve rate of variations of interval-valued variables [24, 45].

Arithmetically, an interval ' E ' is defined by $E = [e_L, e_U] = \{x : e_L \leq x \leq e_U \text{ and } e_L, e_U \text{ in } \mathbb{R}\}$, and the parametric form of ' E ' is defined as:

(a) $E = \{e(s) : e(s) = e_L + s(e_U - e_L), s \in [0, 1]\}$ (Increasing form)

(b) $E = \{e(s) : e(s) = e_U - s(e_U - e_L), s \in [0, 1]\}$ (Decreasing form)

Now, interval arithmetic is accomplished in parametric forms as follows:

Let, $E = [e_L, e_U] = \{e(s) : e(s) = e_L + s(e_U - e_L), s \in [0, 1]\}$ and $F = [f_L, f_U] = \{f(s) : f(s) = f_U - s(f_U - f_L), s \in [0, 1]\}$ be two intervals in the parametric form and $\lambda \in \mathbb{R}$. Then,

Addition: $E \oplus_s F = [e_L, e_U] + [f_L, f_U] = \{e(s) + f(s) : s \in [0, 1]\}$

Subtraction/ Parametric difference: $E \ominus_s F = [e_L, e_U] - [f_L, f_U] = \{e(s) - f(s) : s \in [0, 1]\}$

Scalar multiplication: $\lambda.E = \lambda.[e_L, e_U] = \{\lambda e(s) : s \in [0, 1]\}$

Multiplication: $E \odot F = [e_L, e_U].[f_L, f_U] = \{e(s).f(s) : s \in [0, 1]\}$

Division: $E/F = [e_L, e_U]/[f_L, f_U] = \{e(s)/f(s) : s \in [0, 1] \text{ and } f(s) \neq 0\}$

Some necessary connections along with the differentiation and integration provisions in the parametric form are discussed in [45, 46].

In this work, we have studied precise epidemic model and imprecise epidemic model, whose definitions is given below:

iii. Precise epidemic model: In an epidemic model, where all included parameters have fixed values, then the model is called the precise epidemic model.

iv. Imprecise epidemic model: In an epidemic model, when values of some or all parameters changes or varies with in an interval, then the model is called the imprecise epidemic model.

3. Model Formulation

In this part, first we have introduced a precise SIR epidemic model along with the impact of awareness, vaccination, and treatment. Then, the model has been extended to an imprecise model with interval form by substituting fixed values model parameters including the disease transmission rate, the treatment rate, and the rate of vaccination by interval-valued parameters. Thereafter, we have converted the imprecise model with interval form into a parametric form. Before going to the model construction, in Tables 1 and 2, we have presented all the notations, that will be used in the model formulation.

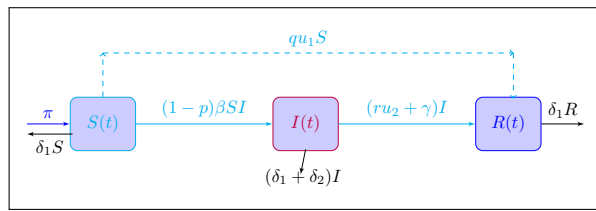


Figure 1. Flow diagram of disease of the proposed model.

3.1. Precise epidemic model

Let, $N(t)$ be the total population which is partitioned into three mutually exclusive compartments based on their disease status namely: susceptible ($S(t)$) population, infected ($I(t)$) population and recovered ($R(t)$) population. Thus, at any time t total population can be written as, $N(t) = S(t) + I(t) + R(t)$. All recruited population (π) are considered as susceptible to this infection and assumed as constant. The susceptible class is vaccinated at a rate u_1 , whereas q is the effectiveness of vaccination and p is the measure of awareness. Therefore, $(1-p)$ is the portion of unaware people. It is also considered that u_2 is the treatment rate and r being the effectiveness rate of treatment. Normal mortality rate are assumed for each compartment and disease-induced death is taken into consideration for the infected class. Based on these assumptions, the flow diagram of the disease transmission is given in Fig. 1. Under the above assumptions and using the flow diagram Fig. 1, the proposed precise model can be represented by the following system of non-linear ODEs:

$$\begin{cases} \frac{dS}{dt} = \pi - (1-p)\beta SI - qu_1 S - \delta_1 S \\ \frac{dI}{dt} = (1-p)\beta SI - ru_2 I - (\delta_1 + \gamma + \delta_2)I \\ \frac{dR}{dt} = ru_2 I - \delta_1 R + qu_1 S + \gamma I \end{cases} \quad (1)$$

with the initial conditions $S(0) > 0$, $I(0) \geq 0$, $R(0) \geq 0$. Description of the state variables and the parameters used in the model have been presented in Table-1. In this model, all parameters are assumed to have fixed values.

3.2. Imprecise SIR model in interval form

All the parameters used in (1) are deliberated precisely or static. However, among them a small number of states where parameters are not always static due to several uncertainties. In epidemic models, impreciseness arises due to the changing nature of human immunity, the genome of pathogens including viruses, and bacteria, climatic change, and the changing behavior of humans including awareness. When information about values of different model parameters cannot be appropriate. Therefore, in such situations, values of such model parameters are accepted as imprecise or flexible. In this study, we have expanded the model (1) to an uncertain condition and to formulate the imprecise model we have accepted the disease transmission rate, rate of treatment, and rate of vaccination of the model (1) as interval-valued. Considering susceptible population, infected population, and recovered population as imprecise and placing the remaining assumptions similar to (1), the imprecise version of model (1) can be introduced mathematically as

Table 1. Biological meaning of state variables and parameters of the model (1).

State Variables/ Parameters	Biological Meaning
$S(t)$:	Abundance of susceptible population at time t
$I(t)$:	Abundance of infected population at time t
$R(t)$:	Abundance of recovered population at time t
p :	Rate of awareness in susceptible population.
u_1 :	Rate of vaccination
q :	Effectiveness of vaccination
u_2 :	Rate of treatment
r :	Effectiveness of treatment
β :	Disease transmission rate
δ_1 :	The natural mortality rate of the populations
γ :	The natural recovery rate of the infected individuals
π :	Recruitment rate of the population
δ_2 :	Disease-induced death rate

$$\begin{cases} \frac{d\hat{S}}{dt} = \pi - (1-p)[\beta_L, \beta_U]\hat{S}\hat{I} - q[u_{1L}, u_{1U}]\hat{S} - \delta_1 \hat{S} \\ \frac{d\hat{I}}{dt} = (1-p)[\beta_L, \beta_U]\hat{S}\hat{I} - r[u_{2L}, u_{2U}]\hat{I} - (\delta_1 + \gamma + \delta_2)\hat{I} \\ \frac{d\hat{R}}{dt} = r[u_{2L}, u_{2U}]\hat{I} - \delta_1 \hat{R} + q[u_{1L}, u_{1U}]\hat{S} + \gamma \hat{I} \end{cases} \quad (2)$$

with the initial conditions $\hat{S}(0) > [0, 0]$, $\hat{I}(0) \geq [0, 0]$, $\hat{R}(0) \geq [0, 0]$. Here $\hat{S}$, $\hat{I}$, $\hat{R}$ are interval-valued susceptible, infected, and recovered population.

3.3. SIR model in parametric form

In epidemiology, infectious disease may be low to high infectious which implies that the disease transmission rate (β) also increase from low to high and accordingly the disease control measures including vaccination (u_1) and treatment (u_2) also need to increase for prevention the disease. In order to study this epidemiological aspect, we have linked the parameters of disease transmission rate, β and control measures u_1 and u_2 by the parameter $s \in [0, 1]$. Since parameters in the interval (Lower-upper bound) form are difficult to handle, these should be represented in the easiest form. Here, all the interval-valued model parameters are presented in linear parametric form. Employing a parametric delegation of interval and its arithmetic, the model (2) can be expressed in the following form:

$$\begin{cases} \frac{dS(t,s)}{dt} = \pi - (1-p)\beta(s)S(t,s)I(t,s) - qu_1(s)S(t,s) - \delta_1 S(t,s) \\ \frac{dI(t,s)}{dt} = (1-p)\beta(s)S(t,s)I(t,s) - ru_2(s)I(t,s) - (\delta_1 + \gamma + \delta_2)I(t,s) \\ \frac{dR(t,s)}{dt} = ru_2(s)I(t,s) - \delta_1 R(t,s) + qu_1(s)S(t,s) + \gamma I(t,s) \end{cases} \quad (3)$$

Table 2. Biological meaning of state variables and parameters in interval and parametric form of imprecise model (2) and (3) respectively.

State Variables/ Parameters	Biological Meaning
$\hat{S}(t)$:	Interval-valued abundance of susceptible population at time t
$\hat{I}(t)$:	Interval-valued abundance of infected population at time t
$\hat{R}(t)$:	Interval-valued abundance of recovered population at time t
$[u_{1L}, u_{1U}]$:	Interval-valued rate of vaccination
$[u_{2L}, u_{2U}]$:	Interval-valued rate of treatment
$[\beta_L, \beta_U]$:	Interval-valued disease transmission rate
$S(t, s)$:	Parametric form of abundance of susceptible population at time t
$I(t, s)$:	Parametric form of abundance of infected population at time t
$R(t, s)$:	Parametric form of abundance of recovered population at time t
$u_1(s)$:	Parametric form of rate of vaccination
$u_2(s)$:	Parametric form of rate of treatment
$\beta(s)$:	Parametric form of disease transmission rate

with the initial conditions $S(0, s) > 0$, $I(0, s) \geq 0$, $R(0, s) \geq 0$, where

$$\beta(s) = \beta_L + s(\beta_U - \beta_L),$$

$$u_1(s) = u_{1L} + s(u_{1U} - u_{1L}),$$

$$u_2(s) = u_{2L} + s(u_{2U} - u_{2L}), \quad s \in [0, 1].$$

The model equations (2) and (3) are equivalent which is shown below.

Theorem 3.1. *The model equations with interval coefficients (2) is equivalent to the model equations (3) in parametric form.*

Proof. : Here, using the parametric representation of interval number, the model equations (2) can be written in following form:

$$\frac{dS(t, s)}{dt} = \pi - (1 - p)(\beta(s) : s \in [0, 1])S(t, s)I(t, s) - q(u_1(s) : s \in [0, 1])S(t, s) - \delta_1 S(t, s)$$

$$\frac{dI(t, s)}{dt} = (1 - p)(\beta(s) : s \in [0, 1])S(t, s)I(t, s) - r(u_2(s) : s \in [0, 1])I(t, s) - (\delta_1 + \gamma + \delta_2)I(t, s)$$

$$\frac{dR(t, s)}{dt} = r(u_2(s) : s \in [0, 1])I(t, s) - \delta_1 R(t, s) + q(u_1(s) : s \in [0, 1])S(t, s) + \gamma I(t, s),$$

where $S \in \hat{S}$, $I \in \hat{I}$, $R \in \hat{R}$, $S(t, s) = S_L(t) + s(S_U(t) - S_L(t))$, $I(t, s) = I_L(t) + s(I_U(t) - I_L(t))$, $R(t, s) = R_L(t) + s(R_U(t) - R_L(t))$ under the above-defined imprecise parameters, the system reduces to the following form:

$$\frac{dS(t, s)}{dt} = \pi - (1 - p)\beta(s)S(t, s)I(t, s) - qu_1(s)S(t, s) - \delta_1 S(t, s)$$

$$\frac{dI(t, s)}{dt} = (1 - p)\beta(s)S(t, s)I(t, s) - ru_2(s)I(t, s) - (\delta_1 + \gamma + \delta_2)I(t, s)$$

$\frac{dR(t, s)}{dt} = ru_2(s)I(t, s) - \delta_1 R(t, s) + qu_1(s)S(t, s) + \gamma I(t, s)$ which is the model (3). Thus, we have obtained the system (3) from the system (2). Similarly, the system (2) can be obtained from the system (3). Therefore, the systems (2) and (3) are equivalent. $\square$

4. Positivity and boundedness of model solutions

In this part, we shall focus on the positivity and boundedness of model solutions of the linear parametric model (3). The system will be biologically significant when all of the variables remain non-negative for $t \geq 0$. Alternatively, the solution with positive initial states will remain positive for all time, which we shall establish in the following Lemma.

Lemma 1 The closed region $\Delta = \{(S(t, s), I(t, s), R(t, s)) \in \mathbb{R}_+^3 : S(t, s) + I(t, s) + R(t, s) \leq \frac{\pi}{\delta_1}\}$ is a positively invariant set for the model (3) with non-negative initial condition in $\mathbb{R}_+^3$.

Proof. Let, $N(t, s) = S(t, s) + I(t, s) + R(t, s)$.

So, the rate of change of total population $N(t, s)$ is,

$$\begin{aligned} N'(t, s) &= \pi - \delta_1 S(t, s) - (\delta_1 + \gamma + \delta_2)I(t, s) - \delta_1 R(t, s) + \gamma I(t, s) \\ &= \pi - \delta_1 N(t, s) - \delta_2 I(t, s) \\ &\leq \pi - \delta_1 N(t, s), \end{aligned}$$

integrating this and taking lim sup as $t \rightarrow \infty$ we get,

$$\limsup_{t \rightarrow \infty} N(t, s) \leq \frac{\pi}{\delta_1}.$$

Thus, the closed region Δ is positively invariant.

Hence, the lemma is proved. $\square$

5. Steady state analysis

In this section, we shall analyze the model (3) qualitatively to understand the condition of existence and stability of its steady states.

5.1. Disease-free equilibrium and basic reproduction number

The basic reproduction number plays a significant role in controlling or spreading the disease. It is prescribed as the size of secondary infections i.e., the size of new infections produced by a single infected person. Analytically, it can be studied easily only when the disease-free state of the system exists. Hethcote [46], Diekmann et al. [47] and Van den Driessche and Watmough [2] introduced a generalized method to determine the basic reproduction number which is acquainted with the next-generation matrix approach. For the system (1) the disease-free equilibrium point is $E_0(\frac{\pi}{qu_1 + \delta_1}, 0, \frac{\pi qu_1}{\delta_1(qu_1 + \delta_1)})$ and hence, basic reproduction number of the mentioned model exists.

Here, we split the right-hand side of the system (1) corresponding to the infected compartments I as $\mathcal{F} - \mathcal{V}$, where $\mathcal{F} = \begin{pmatrix} \beta(1 - p)SI \end{pmatrix}$ and $\mathcal{V} = \begin{pmatrix} ru_2 + \delta_1 + \gamma + \delta_2 I \end{pmatrix}$. Let us define,

$$F = \frac{\partial \mathcal{F}}{\partial x_j}(E_0) = \begin{pmatrix} \frac{\pi(1-p)\beta}{qu_1 + \delta_1} \end{pmatrix},$$

and

$$V = \frac{\partial \mathcal{V}}{\partial x_j}(E_0) = \begin{pmatrix} ru_2 + \delta_1 + \gamma + \delta_2 \end{pmatrix},$$

where $x_j = I$.

The basic reproduction number is the spectral radius of the next-generation matrix FV^{-1} which for the system (1) of the precise model is $R_0 = \frac{\pi(1-p)\beta}{(qu_1(s)+\delta_1)(ru_2(s)+\delta_1+\gamma+\delta_2)}$.

For imprecise parameter, the basic reproduction number $R_0(s)$ is given by

$$R_0(s) = \frac{\pi(1-p)\beta(s)}{(qu_1(s) + \delta_1)(ru_2(s) + \delta_1 + \gamma + \delta_2)}, \quad (4)$$

where $s \in [0, 1]$.

5.2. Stability study of the disease-free equilibrium

Here, we shall perform the stability analysis of the DFE $E_0(\frac{\pi}{qu_1(s)+\delta_1}, 0, \frac{\pi qu_1(s)}{\delta_1(qu_1(s)+\delta_1)})$ of the imprecise model (3) employing the following theorems.

Theorem 5.1. *If $R_0(s) > 1$ then the DFE E_0 of the system (3) is unstable. It is locally asymptotically stable if $R_0(s) < 1$.*

Proof. : The variational matrix of the system (3) at DFE $E_0(\frac{\pi}{qu_1(s)+\delta_1}, 0, \frac{\pi qu_1(s)}{\delta_1(qu_1(s)+\delta_1)})$ is

$$J(E_0) = \begin{pmatrix} -(qu_1(s) + \delta_1) & -a_0 & 0 \\ 0 & a_0 - (b_0 + \delta_1 + \delta_2) & 0 \\ qu_1(s) & b_0 & -\delta_1 \end{pmatrix},$$

where $a_0 = (1-p)\beta(s)S^0$, $b_0 = ru_2(s) + \gamma$ and $S^0 = \frac{\pi}{qu_1(s)+\delta_1}$.

The eigenvalues of this variational matrix $J(E_0)$ are $-\delta_1$, $-(qu_1(s) + \delta_1)$, and $(ru_2(s) + \delta_1 + \gamma + \delta_2)(R_0(s) - 1)$, where $s \in [0, 1]$.

Therefore, E_0 is asymptotically stable for $R_0(s) < 1$ and unstable for $R_0(s) > 1$.

Hence, the theorem is proved. $\square$

Theorem 5.2. *If $R_0(s) < 1$ then the DFE E_0 is globally stable in the closed region*

$$\Delta = \{(S(t, s), I(t, s), R(t, s)) \in \mathbb{R}_+^3 : S(t, s) + I(t, s) + R(t, s) \leq \frac{\pi}{\delta_1}\}.$$

Proof. : Let, $L = I$. Therefore, $\frac{dL}{dt} = \frac{dI}{dt} = (1-p)\beta(s)S(t, s)I(t, s) - ru_2(s)I(t, s) - (\delta_1 + \gamma + \delta_2)I(t, s)$

$$\begin{aligned} &= [(1-p)\beta(s)S(t, s) - (ru_2(s) + \delta_1 + \gamma + \delta_2)]I(t, s) \\ &\leq [\frac{(1-p)\beta(s)\pi}{(\delta_1 + qu_1(s))} - (ru_2(s) + \delta_1 + \gamma + \delta_2)]I(t, s) \\ &= (ru_2(s) + \delta_1 + \gamma + \delta_2)[R_0(s) - 1]I(t, s) \quad [\text{by (4)}] \end{aligned}$$

$$\leq 0, \text{ for } R_0(s) < 1$$

Notice that, $L' = 0$ for $(S(t, s), I(t, s), R(t, s)) \in \Delta$ only if $I(t, s) = 0$,

so that, $(S(t, s), I(t, s), R(t, s)) = E_0$ and that the only positively invariant subset of the plane $I(t, s) = 0$ is the point E_0 .

Thus, it follows from the LaSalle-Liapunov theory [48] that the DFE E_0 is globally stable in Δ for $R_0(s) < 1$.

Hence, the theorem is proved. $\square$

5.3. Existence and stability of endemic equilibrium point

Here, we shall find circumstances for the existence and stability of the EE of the imprecise model (3). Let $E_1(S^*(s), I^*(s), R^*(s))$ be an arbitrary EE of the model (3) which has been achieved from the static states of the model, where the elements of E_1 are given below:

$$\begin{cases} S^*(s) = \frac{\pi}{(qu_1(s)+\delta_1)R_0(s)}, \\ I^*(s) = \frac{(1-p)\beta(s)}{R_0(s)(R_0(s)-1)(ru_2(s)+\gamma)(qu_1(s)+\delta_1)^2+\pi(1-p)qu_1(s)\beta(s)}, \\ R^*(s) = \frac{R_0(s)(R_0(s)-1)(ru_2(s)+\gamma)(qu_1(s)+\delta_1)R_0(s)}{\delta_1(1-p)\beta(s)(qu_1(s)+\delta_1)R_0(s)} \end{cases} \quad (5)$$

whose stability has been explored in the next theorems.

Theorem 5.3. *Thus the EE E_1 to be locally asymptotically stable if $R_0(s) > 1$.*

Proof. : The characteristic equation for E_1 is given by

$$\begin{vmatrix} a_{11} - \lambda & (p-1)\beta(s)S^*(s) & 0 \\ (1-p)\beta(s)I^*(s) & a_{22} - \lambda & 0 \\ qu_1(s) & ru_2(s) + \gamma & -\delta_1 - \lambda \end{vmatrix} = 0,$$

where $a_{11} = (p-1)\beta(s)I^*(s) - qu_1(s) - \delta_1$, and $a_{22} = (1-p)\beta(s)S^*(s) - ru_2(s) - \delta_1 - \gamma - \delta_2$. So, the characteristic equation reduces to-

$$(\lambda + \delta_1)(\lambda^2 - C_1(s)\lambda + C_2(s)) = 0 \quad (6)$$

where, $C_1(s) = (p-1)\beta(s)I^*(s) - qu_1(s) - \delta_1 + (1-p)\beta(s)S^*(s) - ru_2(s) - \delta_1 - \gamma - \delta_2$

$$= -(qu_1(s) + \delta_1)R_0(s)$$

and $C_2(s) = [(p-1)\beta(s)I^*(s) - qu_1(s) - \delta_1][(1-p)\beta(s)S^*(s) - ru_2(s) - \delta_1 - \gamma - \delta_2] - [(p-1)\beta(s)S^*(s)][(1-p)\beta(s)I^*(s)]$

$$= (R_0(s) - 1)(qu_1(s) + \delta_1)(ru_2(s) + \delta_1 + \gamma + \delta_2),$$

$$s \in [0, 1]$$

Then we can say that, $C_1(s) < 0$ and $C_2(s) > 0$ for $R_0(s) > 1$. Hence by the Routh-Hurwitz criterion, we can conclude that, at endemic equilibrium point the system (3) is locally asymptotically stable for $R_0(s) > 1$.

Hence, the theorem is proved. $\square$

Theorem 5.4. *The endemic equilibrium $E_1(S^*(s), I^*(s), R^*(s))$ is globally asymptotically stable in the closed region $\Delta = \{(S(t, s), I(t, s), R(t, s)) \in \mathbb{R}_+^3 : S(t, s) + I(t, s) + R(t, s) \leq \frac{\pi}{\delta_1}\}$ if*

$$\max \left\{ \frac{(ru_2(s) + \gamma + \delta_1 + \delta_2)R_0^2(s)}{(1-p)\beta(s)I^* + qu_1(s) + \delta_1}, \frac{(ru_2(s) + \gamma)^2}{\delta_1(ru_2(s) + \gamma + \delta_1 + \delta_2)} \right\} < 1 - R_0(s)$$

and $(qu_1(s))^2 < ((1-p)\beta(s)I^* + qu_1(s) + \delta_1)\delta_1$.

Proof. : To prove the global stability of the EE $E_1(S^*(s), I^*(s), R^*(s))$, we consider the positive definite function:

$$L(t, s) = \frac{1}{2} \{S(t, s) - S^*(s)\}^2 + \frac{1}{2} \{I(t, s) - I^*(s)\}^2 + \frac{1}{2} \{R(t, s) - R^*(s)\}^2. \quad (7)$$

Now, differentiation of (7) w.r.t. t along the model solution gives the following equation:

$$\begin{cases} \frac{dL(t, s)}{dt} = \{S(t, s) - S^*(s)\} \frac{dS(t, s)}{dt} + \{I(t, s) - I^*(s)\} \frac{dI(t, s)}{dt} \\ + \{R(t, s) - R^*(s)\} \frac{dR(t, s)}{dt}. \end{cases} \quad (8)$$

From the model equation (3) and some simple algebra, $\frac{dL(t, s)}{dt}$ takes the following form:

$$\begin{cases} \frac{dL(t, s)}{dt} = -[(1-p)\beta(s)I^*(s) + qu_1(s) + \delta_1]\{S(t, s) - S^*(s)\}^2 \\ -[ru_2(s) + \gamma + \delta_1 + \delta_2 - (1-p)\beta(s)S(t, s)]\{I(t, s) - I^*(s)\}^2 \\ -\delta_1\{R(t, s) - R^*(s)\}^2 \\ +[(1-p)\beta(s)(I^*(s) - S(t, s))]\{S(t, s) - S^*(s)\}\{I(t, s) - I^*(s)\} \\ +[ru_2(s) + \gamma]\{I(t, s) - I^*(s)\}\{R(t, s) - R^*(s)\} \\ +[qu_1(s)]\{S(t, s) - S^*(s)\}\{R(t, s) - R^*(s)\}. \end{cases}$$

We can rewrite,

$$\begin{cases} \frac{dL(t, s)}{dt} = -a_{11}(s)\{S(t, s) - S^*(s)\}^2 - a_{22}(s)\{I(t, s) - I^*(s)\}^2 \\ -a_{33}(s)\{R(t, s) - R^*(s)\}^2 \\ +a_{12}(s)\{S(t, s) - S^*(s)\}\{I(t, s) - I^*(s)\} \\ +a_{23}(s)\{I(t, s) - I^*(s)\}\{R(t, s) - R^*(s)\} \\ +a_{13}(s)\{S(t, s) - S^*(s)\}\{R(t, s) - R^*(s)\}, \end{cases} \quad (9)$$

where $a_{11}(s) = (1-p)\beta(s)I^*(s) + qu_1(s) + \delta_1$, $a_{22}(s) = ru_2(s) + \gamma + \delta_1 + \delta_2 - (1-p)\beta(s)S(t, s)$, $a_{33} = \delta_1$, $a_{12}(s) = (1-p)\beta(s)(I^*(s) - S(t, s))$, $a_{23}(s) = ru_2(s) + \gamma$, $a_{13}(s) = qu_1(s)$. From the relation (9), it is clear that $\frac{dL(t, s)}{dt}$ will be negative when

$$a_{12}^2(s) < a_{11}(s) \cdot a_{22}(s) \quad (10a)$$

$$a_{23}^2(s) < a_{22}(s) \cdot a_{33} \quad (10b)$$

$$a_{13}^2(s) < a_{11}(s) \cdot a_{33}. \quad (10c)$$

Relations (10a) and (10b) are true when

$$\left\{ \max \left\{ \frac{(ru_2(s) + \gamma + \delta_1 + \delta_2)R_0^2(s)}{(1-p)\beta(s)I^*(s) + qu_1(s) + \delta_1}, \frac{(ru_2(s) + \gamma)^2}{\delta_1(ru_2(s) + \gamma + \delta_1 + \delta_2)} \right\} < 1 - R_0(s) \right\} \quad (11)$$

Relation (10c) is true when

$$(qu_1(s))^2 < ((1-p)\beta(s)I^*(s) + qu_1(s) + \delta_1)\delta_1 \quad (12)$$

Now, $\frac{dL(s, t)}{dt}$ will be negative definite in the closed region Δ if the inequalities (11) and (12) hold. So, $L(t, s)$ is a Liapunov function for the model (3) provided the inequalities (11) and (12) hold. Hence, the EE E_1 is globally asymptotically stable in the closed region Δ under the condition of (11) and (12). $\square$

5.4. Forward bifurcation

In this section, we shall study the bifurcation behavior of the imprecise model (3) at equilibrium points. We see that, the DFE (E_0) passes from stable to unstable state as R_0 increases to the value 1. So, we may conclude that at $R_0(s) = 1$, the system passes through a bifurcation about its disease-free equilibrium E_0 . Again, for $R_0(s) > 1$ the EE E_1 is to be locally asymptotically stable but in

that case, the DFE E_0 is to be unstable. Thus, forward bifurcation occurs in this case-

Theorem 5.5. *Equivalent systems (2) and (3) at E_0 with basic reproduction number $R_0(s) = 1$ will go through transcritical bifurcation for the imprecise parameter threshold $s = s_c$, where $s \in [0, 1]$.*

Proof. : One eigenvalue of the Jacobian matrix $J(E_0(\frac{\pi}{q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1}, 0, \frac{\pi q(u_{1L} + s_c(u_{1U} - u_{1L}))}{\delta_1(q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1)}))$ is zero and other two eigen values of the matrix are negative. Let, eigenvectors of $J(E_0)$ and $J(E_0)^T$ corresponding to zero eigenvalue are $V = (V_1, V_2, 1)^T$ and $W = (0, 1, 0)^T$, where

$$V_1 = \frac{\delta_1(r(u_{2L} + s_c(u_{2U} - u_{2L})) + \delta_1 + \gamma + \delta_2)}{[q(u_{1L} + s_c(u_{1U} - u_{1L}))](r(u_{2L} + s_c(u_{2U} - u_{2L})) + \delta_1 + \gamma + \delta_2) - (q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1)(r(u_{2L} + s_c(u_{2U} - u_{2L})) + \gamma)}$$

and

$$V_2 = \frac{\delta_1(q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1)}{[(q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1)(r(u_{2L} + s_c(u_{2U} - u_{2L})) + \delta_1 + \gamma + \delta_2) - (q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1)(r(u_{2L} + s_c(u_{2U} - u_{2L})) + \gamma)]}$$

Let, $f = [f_1, f_2, f_3]^T$, then we have

(i) $W^T f_s(E_0, s_c) = 0$.

(ii) $W^T Df_s(E_0, s_c)V = ((1-p)\beta_U(\frac{\pi}{q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1}) - ru_{2U})V_2 \neq 0$ if

$(1-p)\beta_U(\frac{\pi}{q(u_{1L} + s_c(u_{1U} - u_{1L})) + \delta_1}) \neq ru_{2U}$

(iii) $W^T D^2 f(E_0, s_c)(V, V) = 2(1-p)(\beta_L + s_c(\beta_U - \beta_L))V_1V_2 \neq 0$

Hence, by the Sotomayor theorem [49], the system (3) undergoes through a Transcritical bifurcation at the imprecise parameter $s = s_c$, when $R_0(s) = 1$.

Justify the above result by considering the following interval form values of the parameters $[u_{1L}, u_{1U}] = [0.81, 0.88]$, $[u_{2L}, u_{2U}] = [0.6, 0.68]$, $[\beta_L, \beta_U] = [0.01, 0.9]$, $\pi = [1, 1]$, $p = [0.01, 0.01]$, $q = [0.8, 0.8]$, $r = [0.9, 0.9]$, $\gamma = [0.07, 0.07]$, $\delta_1 = [0.03, 0.03]$, and $\delta_2 = [0.05, 0.05]$. Therefore, corresponding parametric form of parameters are $u_1(s) = 0.81 + 0.07s$, $u_2(s) = 0.6 + 0.08s$, $\beta(s) = 0.01 + 0.89s$, $\pi = 1$, $p = 0.01$, $q = 0.8$, $r = 0.9$, $\gamma = 0.07$, $\delta_1 = 0.03$, and $\delta_2 = 0.05$. At, $s = s_c = 0.5787$, we have, the disease-free equilibrium point $E_0(1.4076, 0, 31.9257)$ at which the Jacobian matrix

$$J(E_0)|_{s=s_c} = \begin{pmatrix} -0.710407 & -0.731682576 & 0 \\ 0 & 0 & 0 \\ 0.6804072 & 0.6516664 & -0.03 \end{pmatrix} \text{ has a}$$

simple zero eigenvalue. The eigenvectors V and W corresponding to the zero eigenvalue of the matrices $J(E_0)$ and $J(E_0)^T$ are obtained as,

$V = (0.62925361, -0.61097010, 1)^T$ and $W = (0, 1, 0)^T$. This yields $W^T f_s(E_0, s_c) = 0$, $W^T Df_s(E_0, s_c)V = -0.392371 \neq 0$, $W^T D^2 f(E_0, s_c)(V, V) = -0.399674 \neq 0$.

Therefore, by Sotomayor theorem [49], it indicates that system (3) experiences a Transcritical bifurcation at the equilibrium point E_0 when the imprecise parameter s crosses the critical value $s = s_c = 0.5787$ i.e., at $R_0(s) = 1$. Fig. 2, represents the phenomenon of occurrence of Transcritical bifurcation. It is numerically shown that for $R_0(s) < 1$, there is only one equilibrium which is disease-free and stable, whereas, for $R_0(s) > 1$, there are two equilibria, one is disease free and the other is an endemic type of which the EE is asymptotically stable

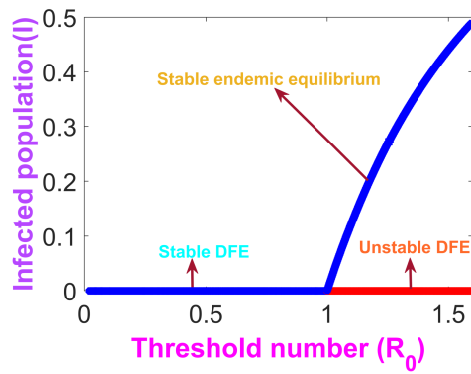


Figure 2. Forward bifurcation diagram.

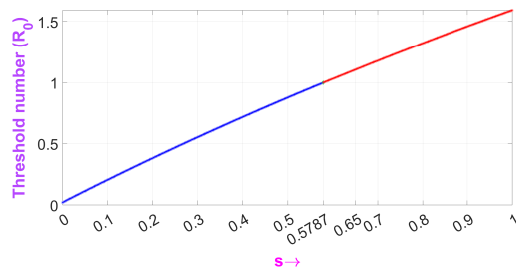


Figure 3. Plot of basic reproduction number (R_0) w.r.t imprecise parameter s .

and the DFE is unstable. The implication of taking into account imprecise values of the parameters is shown in Fig. 3, where we have plotted the basic reproduction number changing s from 0 to 1. It is observed that for $s \leq 0.5787$, the system is disease free (as $R_0(s) < 1$) whereas for $s > 0.5787$, the system becomes endemic (as $R_0(s) > 1$). This means that, the Transcritical or Forward bifurcation occurs or in other words, the disease starts to spread when s crosses the value 0.5787. Finally, from Figs. 2 and 3 we can say that, the disease-free equilibrium point changes stability with the endemic equilibrium point through Transcritical bifurcation. Hence, the result follows. $\square$

6. Sensitivity analysis

Disease control is the main objective of the study of mathematical epidemiology. To fulfill this purpose, it is need to find which are the sensitive parameters and how sensitive they are. To find the most influential model parameters, we enquire about the sensitivity index of the basic reproduction number R_0 , by employing normalized forward approach of sensitivity index. This index of R_0 w.r.t a parameter is defined as the ratio of the relative variation in the threshold number R_0 to the relative variation in that parameter. Thus, the normalized forward sensitivity index of R_0 w.r.t. an arbitrary parameter g is denoted by $Y_g^{R_0}$ and defined as $Y_g^{R_0} = \frac{g}{R_0} \frac{\partial R_0}{\partial g}$ [50].

The significance of this index is that its higher values mean R_0 is more sensitive concerning the parameter g . Again, the sign positive (or negative) implies that the basic reproduction number R_0 increases (or decreases) with increase (or decreases) of g . The explicit expressions of such indices concerning different parameters are obtained from [50]:

$$Y_\pi^{R_0} = 1, Y_p^{R_0} = \frac{p}{p-1}, Y_{\beta(s)}^{R_0} = 1, Y_q^{R_0} = \frac{-qu_1(s)}{(qu_1(s)+\delta_1)},$$

$$Y_{u_1(s)}^{R_0} = \frac{-qu_1(s)}{(qu_1(s)+\delta_1)}, Y_{\delta_1}^{R_0} = \frac{-\delta_1(qu_1(s)+ru_2(s)+\gamma+2\delta_1+\delta_2)}{(qu_1(s)+\delta_1)(ru_2(s)+\delta_1+\gamma+\delta_2)}, Y_{u_2(s)}^{R_0} = \frac{-ru_2(s)}{(ru_2(s)+\delta_1+\gamma+\delta_2)}, Y_r^{R_0} = \frac{-ru_2(s)}{(ru_2(s)+\delta_1+\gamma+\delta_2)}, Y_\gamma^{R_0} = \frac{-\gamma}{(ru_2(s)+\delta_1+\gamma+\delta_2)}, \text{ and } Y_{\delta_2}^{R_0} = \frac{-\delta_2}{(ru_2(s)+\delta_1+\gamma+\delta_2)}.$$

The used values of parameters and the estimated sensitivity indices are presented in the second and last column of Table 3 respectively. The bardigram presentation of sensitivity indices is given in Fig. 4. Our analysis shows that, the number R_0 is most positively sensitive concerning disease transmission rate ($\beta(s)$) and recruitment rate of the population (π) (see Table 3) and most negatively sensitive with rate of vaccination of susceptible people ($u_1(s)$) and rate of treatment of infected people ($u_2(s)$). The sensitivity index of disease transmission rate ($\beta(s)$) is positive. So due to increase (decrease) of parameters ($\beta(s)$) by 5% the number $R_0(s)$ will be increase (decrease) by 5%. This fact is observed in Fig. 5. The controllable negative sensitive model parameters are rate of vaccination of susceptible people ($u_1(s)$), rate of treatment of infected people ($u_2(s)$), the natural death rate of the populations (δ_1), the normal disease recovery proportion of infected individuals (γ) and death rate due to disease (δ_2) with sensitivity indices $[-0.95913, -0.95575]$, $[-0.80315, -0.78261]$, $[-0.08773, -0.08024]$, $[-0.10145, -0.09186]$, and $[-0.07246, -0.06562]$ respectively. Thus, with the increase (decrease) of rate of vaccination of susceptible people ($u_1(s)$) by 3%, the basic reproduction number $R_0(s)$ will be decrease (increase) by 2.867% to 2.877%. This phenomenon is observed in Fig. 6. Due to increase (decrease) of rate of treatment of infected people ($u_2(s)$) by 5%, the basic reproduction number $R_0(s)$ will be decrease (increase) by 3.913% to 4.016%. This phenomenon is observed in Fig. 7. Due to increase (decrease) of the natural death rate of the populations (δ_1) by 5%, the threshold number $R_0(s)$ will be decrease (increase) by 0.401% to 0.439%. Similarly, to increase (decrease) of the normal recovery rate of the infected individuals (γ) by 5%, the number $R_0(s)$ will be decrease (increase) by 0.459% to 0.507% and due to increase (decrease) of disease caused death rate (δ_2) by 5%, the number $R_0(s)$ will be decrease (increase) by 0.328% to 0.362%. Such investigation shows, vaccination and treatment are two most useful parameters to protect the disease. The investigation also shows that, vaccination is more influential than treatment.

Table 3. Used values of the parameter and their sensitivity indices.

State Variables Parameters	Used parameter values	Sensitivity indices
p :	0.01	-0.01
$u_1(s)$:	[0.81, 0.88]	[-0.95913,-0.95575]
q :	0.8	[-0.95913,-0.95575]
$u_2(s)$:	[0.6, 0.68]	[-0.80315,-0.78261]
r :	0.9	[-0.80315,-0.78261]
$\beta(s)$:	[0.01, 0.9]	1
δ_1 :	0.03	[-0.08773,-0.08024]
γ :	0.07	[-0.10145,-0.09186]
π :	1	1
δ_2 :	0.05	[-0.07246,-0.06562]

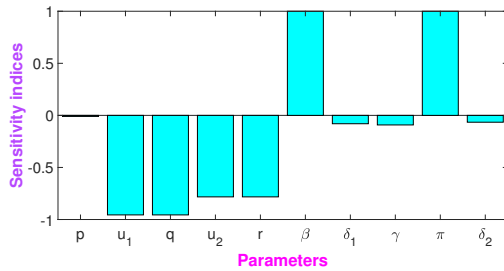


Figure 4. Bar diagram of the sensitivity indices of the model parameters.

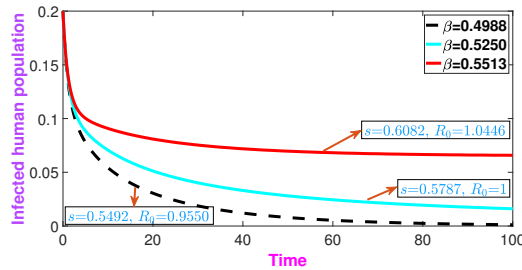


Figure 5. Time series of infected population for the imprecise parameter values $s = 0.5492$, $s = 0.5787$, $s = 0.6082$ respectively for different values of disease transmission rate ($\beta = 0.4988$, $\beta = 0.5250$, $\beta = 0.5513$).

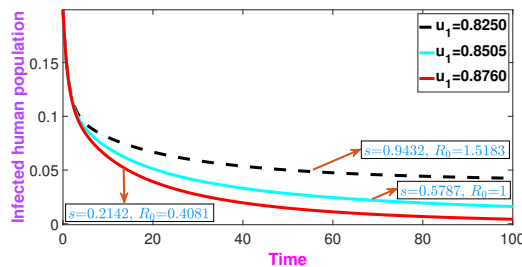


Figure 6. Time series of infected population for the imprecise parameter values $s = 0.2142$, $s = 0.5787$, $s = 0.9432$ respectively for different values of vaccination control ($u_1 = 0.8760$, $u_1 = 0.8505$, $u_1 = 0.8250$).

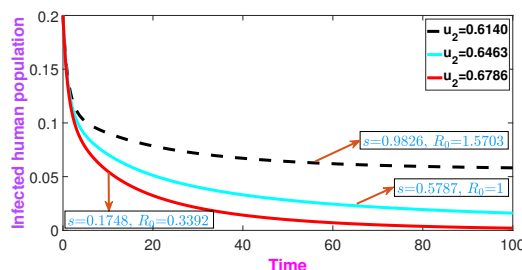


Figure 7. Time series of infected population for the imprecise parameter values $s = 0.1748$, $s = 0.5787$, $s = 0.9826$ respectively for different values of treatment control ($u_2 = 0.6786$, $u_2 = 0.6463$, $u_2 = 0.6140$).

7. Optimal control

In this part, we shall simulate the results of our imprecise model (3) for two cases (i) for lower value of $s (< 0.5787)$ and (ii) for upper value of $s (> 0.5787)$. When the value of s is small, disease transmission is low and the system is almost disease free. So, in this case, disease-free equilibrium will be stable. As s increases, disease transmission rate increases, and when s crosses the values 0.5787, then endemic equilibrium will be stable.

7.1. Simulation of results for lower value of $s (< 0.5787)$

In this section, the theoretical results obtained so far are verified through some graphical illustrations. First of all, to study the stability of disease-free equilibrium, we consider simulated data for the parameters values $\pi = 1$; $p = 0.01$; $q = 0.8$; $u_{1L} = 0.81$; $u_{1U} = 0.88$; $r = 0.9$; $u_{2L} = 0.6$; $u_{2U} = 0.68$; $\gamma = 0.07$; $\delta_1 = 0.03$; $\delta_2 = 0.05$; $\beta_L = 0.01$; $\beta_U = 0.9$; $s = 0.4$. Using these values we compute the numerical value of the basic reproduction number $R_0(s) = 0.7197 < 1$ and the proposed model has a disease-free equilibrium $E_0(\frac{\pi}{qu_1(s)+\delta_1}, 0, \frac{\pi qu_1(s)}{\delta_1(qu_1(s)+\delta_1)}) = (1.4278, 0, 31.9056)$. This shows in Fig. 8 that susceptible individuals and recovered individuals approach steady-state values while infected individuals approach zero as time goes to infinity. Thus, the disease disappears and dies out. This pictorial representation also shows that disease-free equilibrium E_0 is globally asymptotically stable. This is because the disease transmission rate is low in this case for a lower value of s . So, A high rate of vaccination or treatment is not required then.

7.2. Simulation results for upper value of $s (> 0.5787)$

For upper values of s , if disease transmission is very high, then the endemic equilibrium (EE) will be stable. To describe the stability of the endemic equilibrium, we consider a simulated data set for parameters as $\pi = 1$; $p = 0.01$; $q = 0.8$; $u_{1L} = 0.81$; $u_{1U} = 0.88$; $r = 0.9$; $u_{2L} = 0.6$; $u_{2U} = 0.68$; $\gamma = 0.07$; $\delta_1 = 0.03$; $\delta_2 = 0.05$; $\beta_L = 0.01$; $\beta_U = 0.9$; $s = 0.8$. Then, we compute the numerical value of number $R_0(s) = 1.3228$, which is greater than 1. Also, we find the endemic equilibrium $E_1(S^*(s), I^*(s), R^*(s)) = (1.0459, 0.3264, 31.4170)$ of the imprecise model (3). It is seen from Fig. 9 that susceptible, infected, and recovered individuals all approach steady nonzero values as time goes to infinity. Thus, the disease has appeared in the system. This fact is observed in Fig. 9, which indicate that E_1 is globally asymptotically stable. The numerical simulations concerning above mentioned hypothetical data are played by using MATLAB software. Here, we have taken into account two controls, one control u_1 is conducted for vaccinating the susceptible population, and the other control u_2 is conducted for treatment efforts of infected populations. It shows that as s increases the disease transmission rate, rate of vaccination, and rate of treatment control all increase along with the basic reproduction number $R_0(s)$. This means increasing disease transmission rate increases the epidemic and hence, higher vaccination and as well as higher treatment are required. From our data set the critical situation starts when the imprecise parameter crosses the value $s = 0.58$. It is clear from our data set that since the vaccination rate is very high using in $[0.81, 0.88]$ and moderate treatment $[0.6, 0.68]$ despite very high disease transmission rate $\beta(s) = 0.722$ at $s = 0.8$, infected individual number $I^*(s) = 0.3264$ is small and the basic reproduction number $R_0(s) = 1.3228$ is not very large. This shows the effectiveness of vaccination.

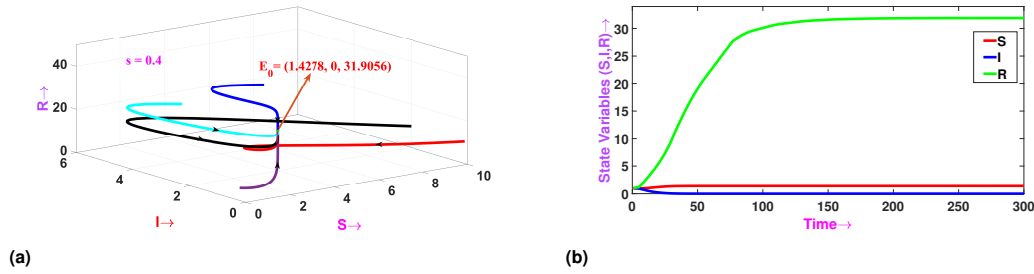


Figure 8. (a) Phase Portrait of global stability of DFE for the imprecise parameter value $s = 0.4$, (b) Time series of state variables (S, I, R).

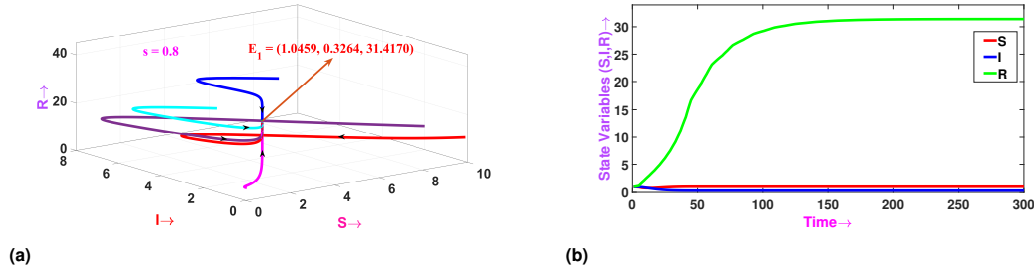


Figure 9. (a) Phase Portrait of global stability of EE for the imprecise parameter values $s = 0.8$, (b) Time series of state variables (S, I, R).

7.3. Analysis of the control problem

In this part, we shall find the optimal control, namely the controls that optimize our objective function. In this study, we shall trace a control set that minimizes number infected population along with the vaccination and treatment controls. So, we can say that, the trick of optimal control is to minimize the susceptible and infected individuals as well as the cost for the two controls vaccination and treatment. For this purpose, we define the objective function:

$$J(u_1, u_2) = \int_0^T (G_1 S + G_2 I + \frac{1}{2} H_1 u_1^2 + \frac{1}{2} H_2 u_2^2) dt$$

subject to the state system of (1).

Therefore, in imprecise environment with imprecise parameter s , for the system (3) the objective function becomes,

$$J(u_1(s), u_2(s)) = \int_0^T (G_1 S(t, s) + G_2 I(t, s) + \frac{1}{2} H_1 u_1^2(t, s) + \frac{1}{2} H_2 u_2^2(t, s)) dt \quad (13)$$

Here, the constants G_1 and G_2 are respectively the per capita loss due to exists of susceptible and infected populations at any instant t . H_1 and H_2 represent the costs connected with vaccination of susceptible and treatment of infected individuals respectively. We also take that the time interval is $[0, T]$.

Let, $u_1^*(t, s)$ and $u_2^*(t, s)$ be the optimal controls, we shall find a set of control functions such that

$$J(u_1^*(t, s), u_2^*(t, s)) = \min\{J(u_1(t, s), u_2(t, s))\}, \quad (14)$$

where $(u_1(t, s), u_2(t, s)) \in U$ and $s \in [0, 1]$ subject to the state system (3), where the control set U is

$$U = (u_1(t, s), u_2(t, s)) | u_i(t, s) \text{ is Lebesgue measurable on } [0, 1], \quad (15)$$

$$0 \leq u_i(t, s) \leq 1, i = 1, 2 \text{ and } t \in [0, T]$$

7.4. Existence and uniqueness of optimal control

The following result is thinking with the presence of optimal control double for the minimization of the cost function (13). In order to establish this result we have used [51–53]

Theorem 7.1. *There exists an optimal control double, u_1^*, u_2^* , that minimizes the objective functional $J(u_1, u_2)$ subject to the system (3), if the following conditions are hold.*

- (i) *The set of control is closed and convex.*
- (ii) *Both the state and control variables of the system (3) is bounded by a linear function.*
- (iii) *The integrand of the objective functional (13) is convex w.r.t the control.*
- (iv) *There are constants $a_1, a_2 > 0$ and $a_3 > 1$ such that the integrand of (13) is bounded below by the number of the form:*

$$a_1 \left(\sum_{i=1}^2 |u_i|^2 \right)^{\frac{a_3}{2}} - a_2$$

Proof. (i) Let, the control set $U = [0, 1]^2$ is closed by definition (15). Also, assume $v, w \in U$, where $v = (v_1, v_2)$ and $w = (w_1, w_2)$, be any two arbitrary points. Then, from the definition of a convex set [51], for all $r \in [0, 1]$, we have $rv + (1 - r)w \in [0, 1]^2$. Then we can say,

$rv + (1 - r)w \in U$, implying the convexity of U .

(ii) Assume that, the right-hand side of (3) be presented by $f_0(t, s, x, u)$. Thus, we can express as

$f_0(t, s, x, u) = f_1(t, s, x) + f_2(t, s, x)u$, where $f_1(t, s, x)$ is an (3×1) Jacobian matrix presenting the right-hand side of the system (3) absence of controls and $f_2(t, s, x)$ is an (3×2) Jacobian matrix presenting the right-hand side of the system (3) present of controls u_i , ($i = 1, 2$). So, we can say,

$$||f_0(t, s, x, u)|| \leq ||f_1(t, s, x)|| + ||f_2(t, s, x)|| ||u||$$

$$\leq b_1 + b_2 ||u||,$$

where

$$f_1(t, s, x) = \begin{pmatrix} \pi - (1-p)\beta(s)S(t, s)I(t, s) - \delta_1 S(t, s) \\ (1-p)\beta(s)S(t, s)I(t, s) - (\delta_1 + \gamma + \delta_2)I(t, s) \\ -\delta_1 R(t, s) + \gamma I(t, s) \end{pmatrix},$$

$$f_2(t, s, x) = \begin{pmatrix} -qS(t, s) & 0 \\ 0 & -rI(t, s) \\ qS(t, s) & rI(t, s) \end{pmatrix}, u(t, s) = \begin{pmatrix} u_1(t, s) \\ u_2(t, s) \end{pmatrix},$$

$$b_1 = \frac{\pi}{\delta_1} \sqrt{2\beta^2(1-p)^2 + 3\delta_1^2 + (\delta_1 + \gamma + \delta_2)^2 + \gamma^2},$$

and

$$b_2 = \frac{\pi}{\delta_1} \sqrt{2(q^2 + r^2)}$$

(iii) The Lagrangian of integrand of the objective function (13) related to developed disease control system (3) is denoted by $L(t, s, x, u)$ and is defined by

$$L(t, s, x, u) = g_0(t, s, x) + h_0(t, s, x), \quad (16)$$

where $g_0(t, s, x) = G_1 S(t, s) + G_2 I(t, s)$ and $h_0(t, s, x) = \frac{1}{2}(H_1 u_1^2(t, s) + H_2 u_2^2(t, s))$. Now, we shall to prove $h_0(t, s, x)$, which is a linear combination of the control functions $q_i(u) = \frac{1}{2}u_i^2$, ($i=1,2$), is convex. To prove this, now we define $q : U \rightarrow \mathbb{R}$ by $q(u) = \frac{1}{2}u^2$. For all $v, w \in U$ and $r \in [0, 1]$, then we have the following inequality:

$$rq(v) + (1-r)q(w) \geq q(rv + (1-r)w).$$

This gratifies the convexity of the Lagrangian with respect to the control.

(iv) We can easily say, from the Lagrangian (14) that $L(t, s, x, u) \geq h_0(t, s, x)$. It fulfill that,

$$L(t, s, x, u) \geq \frac{1}{2}(H_1 u_1^2(t, s) + H_2 u_2^2(t, s))$$

$$\geq a_1 \left(\sum_{i=1}^2 |u_i|^2 \right)^{\frac{a_3}{2}} - a_2,$$

where $a_1 = \frac{1}{2} \min\{H_1, H_2\}$, $a_2 > 0$ and $a_3 = 2$.

Hence, the proof is completed. $\square$

7.5. Characterization of control problem

We shall employ here Pontryagin's maximum principle [54] to search for the optimal controls that gratify the necessary conditions [55]. First of all, the Lagrangian for the optimal control problem is prescribed by

$$L = G_1 S(t, s) + G_2 I(t, s) + \frac{1}{2}H_1 u_1^2(t, s) + \frac{1}{2}H_2 u_2^2(t, s)$$

Now, we form the Hamiltonian H for the problem given by, $H = L + \lambda_1\{\pi - (1-p)\beta(s)S(t, s)I(t, s) - qu_1(t, s)S(t, s) - \delta_1 S(t, s)\} + \lambda_2\{(1-p)\beta(s)S(t, s)I(t, s) - ru_2(t, s)I(t, s) - (\delta_1 + \gamma + \delta_2)I(t, s)\} + \lambda_3\{ru_2(t, s)I(t, s) - \delta_1 R(t, s) + qu_1(t, s)S(t, s) + \gamma I(t, s)\}$,

where λ_1 , λ_2 and λ_3 are the adjoint variables satisfying the following adjoint system:

$$\begin{cases} \lambda_1' = -\frac{\partial H}{\partial S(t, s)} = -G_1 + (\lambda_1 - \lambda_2)(1-p)\beta(s)I(t, s) + \delta_1 \lambda_1 \\ \quad + qu_1(t, s)(\lambda_1 - \lambda_3) \\ \lambda_2' = -\frac{\partial H}{\partial I(t, s)} = -G_2 + (\lambda_1 - \lambda_2)(1-p)\beta(s)S(t, s) \\ \quad + ru_2(t, s)(\lambda_2 - \lambda_3) + (\delta_1 + \delta_2)\lambda_2 + \gamma(\lambda_2 - \lambda_3) \\ \lambda_3' = -\frac{\partial H}{\partial R(t, s)} = \delta_1 \lambda_3. \end{cases} \quad (17)$$

The transversality or boundary conditions are

$$\begin{cases} \lambda_1(T) = 0, \\ \lambda_2(T) = 0, \\ \lambda_3(T) = 0 \end{cases} \quad (18)$$

By the condition of optimality, we get

$$\frac{\partial H}{\partial u_1(t, s)} = 0 \text{ and } \frac{\partial H}{\partial u_2(t, s)} = 0$$

This implies, $H_1 u_1(t, s) - qS(t, s)(\lambda_1 - \lambda_3) = 0$ and $H_2 u_2(t, s) - rI(t, s)(\lambda_2 - \lambda_3) = 0$

Thus,

$$u_1(t, s) = \frac{(\lambda_1 - \lambda_3)qS(t, s)}{H_1} \text{ and } u_2(t, s) = \frac{(\lambda_2 - \lambda_3)rI(t, s)}{H_2}$$

Clearly, $\frac{\partial^2 H}{\partial u_1^2(t, s)} = H_1 > 0$, $\frac{\partial^2 H}{\partial u_2^2(t, s)} = H_2 > 0$ and $\frac{\partial^2 H}{\partial u_1^2(t, s)} \frac{\partial^2 H}{\partial u_2^2(t, s)} - \left(\frac{\partial^2 H}{\partial u_1(t, s) \partial u_2(t, s)} \right)^2 = H_1 H_2 > 0$.

Finally, we get, $u_1^*(t, s) = \max\{0, \min\{\frac{(\lambda_1^* - \lambda_3^*)qS^*(t, s)}{H_1}, 1\}\}$, and $u_2^*(t, s) = \max\{0, \min\{\frac{(\lambda_2^* - \lambda_3^*)rI^*(t, s)}{H_2}, 1\}\}$

Here, $S^*(t, s), I^*(t, s), R^*(t, s)$ are the optimum values of $S(t, s), I(t, s), R(t, s)$ respectively and $(\lambda_1^*, \lambda_2^*, \lambda_3^*)$ is the solution of the system (17) satisfying the conditions (18). Thus, the control functions $u_i^*(t, s)$ ($i = 1, 2$) minimize the objective functional J . In the next section, we shall simulate the results of this control problem.

7.6. Simulation of control problem

Numerical simulations of the mentioned model (3) are carried out to explore the appropriateness of the reported protective measurements. A famous iterative scheme RK4 is employed to count the numerical solution of the propounded model. During this process we have used simulated values of parameters $\pi = 1; p = 0.01; q = 0.8; u_{1L} = 0.81; u_{1U} = 0.88; r = 0.9; u_{2L} = 0.6; u_{2U} = 0.68; \gamma = 0.07; \delta_1 = 0.03; \delta_2 = 0.05; \beta_L = 0.01; \beta_U = 0.9; G_1 = 0.04, G_2 = 0.01, H_1 = 0.92, H_2 = 0.4$. The time period for controls to be applied is $t = 100$ days along with initial values of state variables $S(t), I(t), R(t)$ as $S(0) = 5000, I(0) = 5$, and $R(0) = 0$ respectively. According to our model, the number of control scenarios maybe three. The impact of each scenario on control disease has been discussed below with Figs. (10 - 15) and found best control strategies:

7.6.1. Scenario 1: strategies with vaccination and treatment as both control variables

In this scenario, we have analyzed the combined impact of vaccination control u_1 and treatment control u_2 on the dynamics of the infectious disease model. Using optimal control process in (3), we get some graphical results that are shown in Figs. (10, 13). In Figs. 10(a-c) describe the time series of infected class for

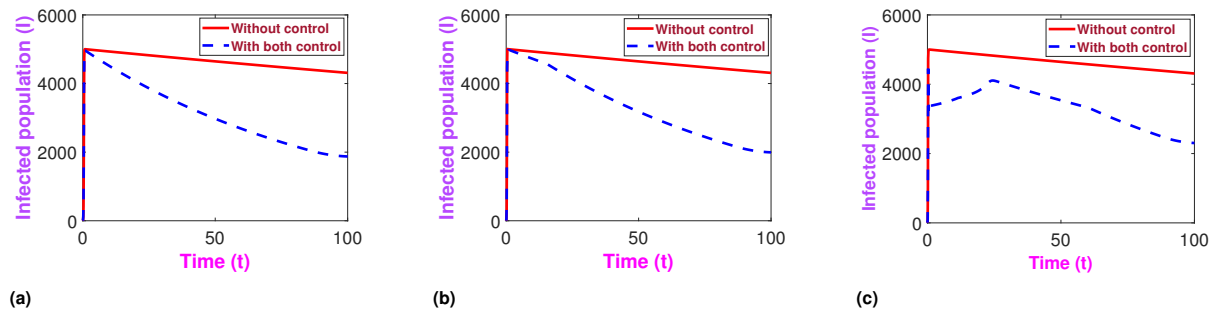


Figure 10. Time series of infected classes with vaccination control u_1 and treatment control u_2 and without both control for imprecise parametric values (a) $s = 0.4$, (b) $s = 0.58$, (c) $s = 0.8$.

imprecise parametric values $s = 0.4, 0.58, 0.8$ respectively with control and without control. Observing these graphical figures we can say that, the infected population is low in the presence of both control variables. Here the imprecise parameter values (s) play a vital role. Fig. 10(a) and Fig. 10(b) show that, for $s = 0.4$ and $s = 0.58$, the number of infected populations reduces drastically as both vaccination control and treatment controls are employed, but for $s = 0.8$, the number of infected populations increases first and then reduces when both controls are used. This happens due to a high value of s close to 1, and disease transmission rate $\beta(s)$ is very high which increases the infected population first, even when both vaccination and treatment control are started. After starting vaccination for the susceptible population and treatment of the infected population, it will take some time to reduce the total infected population, i.e., to control the disease. For this reason, Fig. 10(c) shows that for $s = 0.8$ infected population increase first and then decrease after $t = 24$ days.

Through these graphical figures, we can say that, for imprecise parameter value $s = 0.4$ (when the system is disease-free), neither vaccination control (u_1) nor treatment control (u_2) is necessary as shown in Fig. 13(a). For imprecise parameter value $s = 0.58$ (when the disease is just coming into the system), vaccination control (u_1) and treatment control (u_2) both are important, and use these two controls simultaneously according to the need of the system i.e., in time zone (day 1 to day 15), in which vaccination and treatment both necessary, after that (day 16 to day 100) vaccination is almost stopped that shown in Fig. 13(b) but, using full treatment for day 16 to day 88, and finally, for day 89 to day 100 it will decrease that shown in Fig. 13(d). For imprecise parameter value $s = 0.8$ (when disease transmission is very high), then vaccination control (u_1) and treatment control (u_2) are very important to control the epidemic i.e., there is relaxation to change of the vaccination and treatment strategies. There is a suitable time zone in which use of vaccination and treatment is crucial. In the time zone (day 1 to day 10) in which full vaccination and treatment should apply, then a relaxation zone (day 11 to day 60), and finally for day 61 to day 100, vaccination will decrease, and will little amount that is showing in Fig. 13(c) but using full treatment for day 61 to day 89, and finally, for day 90 to day 100, it will decrease and little amount that shown in Fig. 13(e).

7.6.2. Scenario 2: strategies with only vaccination as a control variable

In this scenario, we have analyzed only impact of vaccination control u_1 on the dynamics of an imprecise infectious disease model. Through, the optimal control process in (3) we get some graphical results that are shown in Fig. 11 and Fig. 14. In Figs. 11(a-c) describe the time series of infected class with only vaccination

control, and compare that with the same without any control for imprecise parametric values $s = 0.4, 0.58, 0.8$ respectively. Observing these graphical figures we can say, imprecise parameter values (s) play a vital role in disease dynamics. For $s = 0.4$ and $s = 0.58$, the number of infected populations does not change. Although, for $s = 0.8$, the number of infected population reduces a small amount, after some time the number of infected people increased, and was almost equal to the number of infected population due to without control variables shown in Fig. 11(c). The Figs. 14(a-c) describes the control profile of vaccination control u_1 for imprecise parametric values $s = 0.4, 0.58, 0.8$ respectively. Through these graphical figures, we can say, for imprecise parameter value $s = 0.4$ (when the system is disease-free), vaccination control (u_1) is not necessarily shown in Fig. 14(a), for imprecise parameter value $s = 0.58$ (when the disease is just coming in system), vaccination control (u_1) using increasing-decreasing way i.e., there is a suitable time zone in which use of vaccination is crucial. In the time zone (day 1 to day 5) in which full vaccination should apply, then a relaxation zone for (day 6 to day 97), and finally for day 98 to day 100, it will decrease and will little amount that is showing in Fig. 14(b). For imprecise parameter value $s = 0.8$ (when a huge amount of disease exists in the system), vaccination control (u_1) according to increasing- decreasing way i.e., there is relaxation to change of the vaccination strategies. There is a suitable time zone in which use of vaccination is crucial. In the time zone (day 1 to day 59) in which full vaccination should be applied, then a relaxation zone (day 60 to day 99), and finally, for day 100, it will decrease and will little amount that shown in Fig. 14(c).

7.6.3. Scenario 3: strategies with only treatment as a control variable

In this part, we have analyzed only impact of treatment control u_2 on the disease dynamics of imprecise infectious disease model. Through, the optimal control process in (3) we get some graphical results that are shown in Fig. 12 and Fig. 15. Fig. 12(a) and Fig. 12(b) describe the time series of infected class with only treatment control and without any control, for imprecise parametric values $s = 0.58, 0.8$ respectively. Observing these graphical figures, we can say that, the number of infected people is less due to treatment control variables. Here, the imprecise parameter values (s) play a vital role. For $s = 0.58$, the size of infected individuals reduces a large amount, but for $s = 0.8$, increases first and then decreases as in the case of only vaccination control. Fig. 15(a) and Fig. 15(b) describe the control profile of treatment control u_2 for imprecise parametric values $s = 0.58, 0.8$ respectively. For imprecise parameter value $s = 0.58$ (when disease is entering in system), necessity of treatment control (u_2)

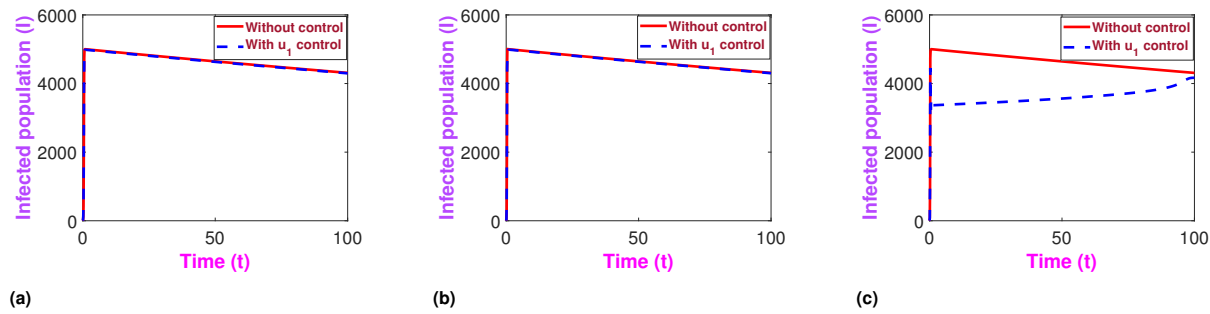


Figure 11. Time series of infected classes with only u_1 control and without both control for imprecise parametric values (a) $s = 0.4$, (b) $s = 0.58$, (c) $s = 0.8$.

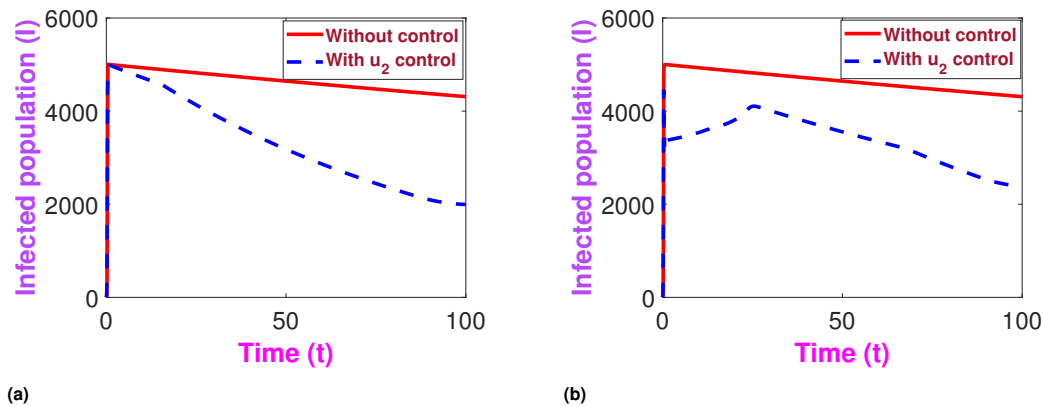


Figure 12. Time series of infected classes with only u_2 control and without both control for imprecise parametric values (a) $s = 0.58$, (b) $s = 0.8$.

is started to control the spread of the disease. Once the treatment is started, infected population is reduced and then, treatment necessity is also reduced which is shown in Fig. 15(a). For imprecise parameter value, $s = 0.8$ (when a huge amount of disease exists in the system), first necessity of treatment control (u_2) is very high to reduce the infected population. Once the infected population is reduced necessity of treatment control is also reduced which is shown in Fig. 15(b). If we compare Fig. 15(a) and Fig. 15(b), necessity of treatment control is very high in the latter case, and needs more time to reduce the infection from the system than in the former one.

8. Conclusions

In this paper, we have formulated and studied an imprecise epidemic model with vaccination and treatment controls. Basic properties including positivity and boundedness of the model solution have been studied. We have found the disease-free equilibrium point, and derived the expression of the basic reproduction number. The disease-free equilibrium point is locally as well as globally stable for $s < 0.5787$ when $R_0(s) < 1$. A unique endemic equilibrium point exists, which is locally asymptotically stable for $s > 0.5787$ when $R_0(s) > 1$. The endemic equilibrium point is globally stable under some conditions. We have seen that, a Transcritical bifurcation occurs at $s = 0.5787$ when the basic reproduction number $R_0(s) = 1$. So, at $s = 0.5787$ the stability exchange from disease-free equilibrium point to endemic equilibrium point. Thus, disease persists in the population when $s > 0.5787$ and is eradicated from the population when $s < 0.5787$. The sensitivity analysis has been carried out.

According to sensitivity analysis, vaccination and treatment are the two most important parameters to control the disease. It also shows that, vaccination is more effective than treatment in this type of system. Our investigation shows that, for a low value of $s (< 0.5787)$ disease transmission rate $\beta(s)$ is very low and hence, the system is almost disease free. Then neither vaccination nor treatment control is necessary. Both controls become important for $s (> 0.5787)$, when the disease transmission rate increases to a certain level. It is also seen from our study that only vaccination or only treatment is not sufficient to control the epidemic, as it is seen that the infected population starts to fall at $t = 24$ days in Fig. 10(c) and it falls at $t = 25$ days in Fig. 12(b). Using optimal control theory we find the proper control policy of the disease. We have studied the optimal control model including existence and uniqueness of optimal control, characterization of control problem, and simulation of control and we have found the proper control policy for the disease.

The contributions of this work are as follows:

- The model undergoes through a Transcritical bifurcation at $s = 0.5787$ when the basic reproduction number $R_0(s) = 1$. So, at $s = 0.5787$ the stability exchange from disease-free equilibrium point to endemic equilibrium point.
- According to sensitivity analysis vaccination and treatment are two most important parameters to control the disease. It also shows that, vaccination is more effective than treatment in this type of system.
- Our investigation shows that for a low value of an imprecise parameter, $s (< 0.5787)$ disease transmission rate $\beta(s)$ is very low, and hence the system is almost disease free. In

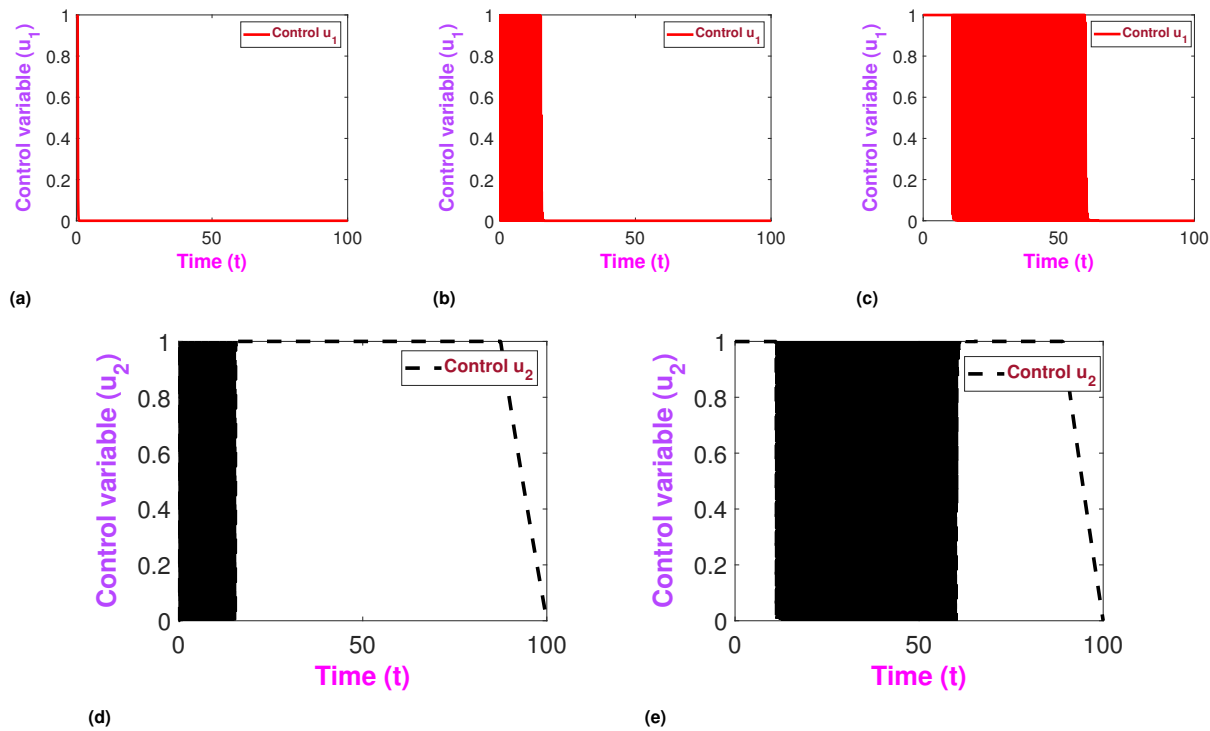


Figure 13. Control profile of vaccination control u_1 and treatment control u_2 for imprecise parametric values (a) $s = 0.4$, (b),(d) $s = 0.58$, (c),(e) $s = 0.8$.

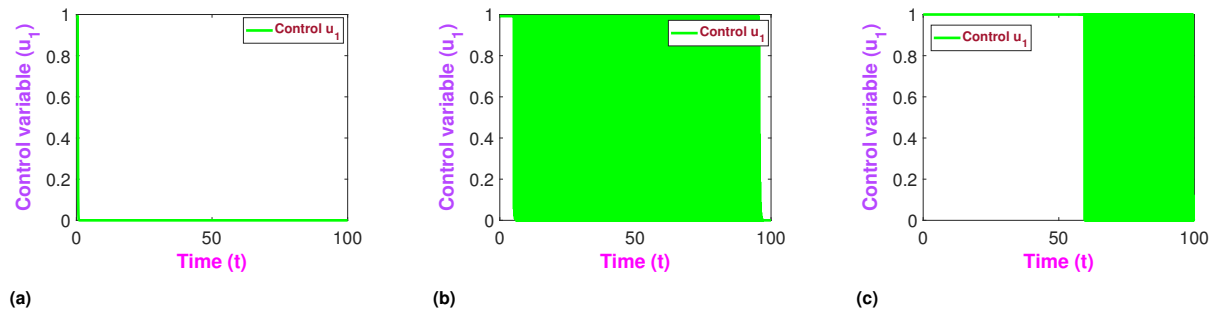


Figure 14. Control profile of vaccination control u_1 for imprecise parametric values (a) $s = 0.4$, (b) $s = 0.58$, (c) $s = 0.8$.

this situation neither vaccination nor treatment control is necessary.

- d. Both of vaccination and treatment controls become important for $s(> 0.5787)$ when disease transmission rate increases a certain level.
- e. It is also seen from our study that, only vaccination or only treatment is not sufficient to control the epidemic, as it is seen that the infected population starts to fall at $t = 24$ days in Fig. 10(c) and it falls at $t = 25$ days in Fig. 12(b).
- f. We have studied optimal control model including existence and uniqueness of optimal control, characterization of control problem, and simulation of control and we have found the proper control policy for the disease in an uncertain environment.
- g. The basic reproduction number has been defined in terms of imprecise parameters.
- h. The disease-free equilibrium point of the imprecise model is unstable if $R_0(s) > 1$ and it is locally asymptotically stable if $R_0(s) < 1$, where $R_0(s)$ is the basic reproduction number, a function of imprecise parameter s .

- i. If $R_0(s) < 1$, then DFE is globally stable in the closed region Δ .
- j. The EE is locally stable, if $R_0(s) > 1$.
- k. The EE is globally stable in the closed region Δ under some conditions.
- l. Finally from this work, we have concluded that, when the disease transmission rate $\beta(s)$ is over 50% although the system is disease free.

Declarations

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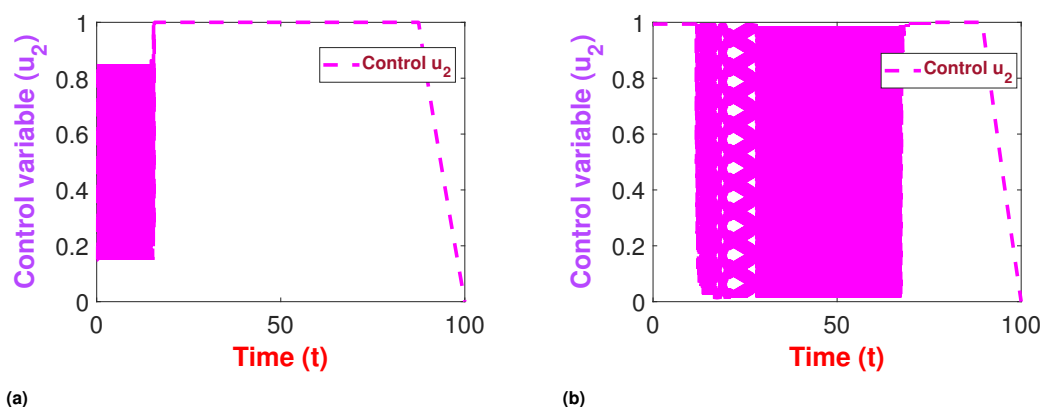


Figure 15. Control profile of treatment control u_2 for imprecise parametric values (a) $s = 0.58$, (b) $s = 0.8$.

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