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STABILITY ANALYSIS AND APPROXIMATE SOLUTION OF SIR EPIDEMIC MODEL WITH CROWLEY-MARTIN TYPE FUNCTIONAL RESPONSE AND HOLLING TYPE-II TREATMENT RATE BY USING HOMOTOPY ANALYSIS METHOD

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Abstract In this paper, SIR epidemic model with Crowley-Martin type functional response and Holling type-II treatment rate is investigated. The analysis of the model shows that it has two equilibria, namely disease-free and endemic. We investigate the existence and stability results of equilibria by using LaSalle's invariant principle and Lyapunov function. $\mathfrak{R}_0$ has been found to ensure the extinction or persistence of the infection. Furthermore, homotopy analysis method is employed to obtain the series solution of the proposed model. By using the homotopy solutions, firstly, several $\hbar$-curves are plotted to demonstrate the regions of convergence, then the residual and square residual errors are obtained for different values of these regions. Secondly, the numerical solutions are presented for various iterations and the absolute error functions are applied to show the accuracy of the applied homotopy analysis method.

Keywords SIR epidemic model, homotopy analysis method, stability analysis, reproduction number $\mathfrak{R}_0$, Crowley-Martin type incidence rate, Holling type-II treatment rate, auxiliary parameter $\hbar$.

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1. Introduction

Epidemiology has its roots in the study of infectious diseases. While modern epidemiology deals with a host of different health-related topics, infectious diseases are still an important component. The classical study approach of epidemiology, based on randomized controlled trials, cohort studies, case-control studies, and related designs can be successfully applied to infectious diseases [7]. Modern infectious disease epidemiology makes heavy use of computational model-based approaches and a dynamical systems perspective. The importance of analyzing infectious diseases in such a way keeps increasing. Human Immunodeficiency Virus (HIV) is a virus

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that causes worldwide devastation. Its host cells are CD4+ T-cells, which are part of the immune system component that signals to the rest of the immune system that infection is present [7, 24]. HIV is a retrovirus that targets the CD4+ T lymphocytes, which are the most abundant white blood cells of the immune system. Although HIV infects other cells also, it wreaks the most havoc on the CD4+ T-cells by causing their decline and destruction, thus decreasing the resistance of the immune system [5,23,25]. In the field of epidemiology, a crucial issue in the study of the spread of an infectious disease is how it is transmitted. The transmission of an infectious disease is determined by the incidence rate, which is defined as the rate at which susceptible becomes infectious. The incidence rate therefore plays a key role in study of the qualitative description of the transmission dynamics of infectious diseases. The form of the incidence rate that is used in the classical Kermack-Mckendrick model [2, 24] is the simple mass action βSI, where S and I denote the number of susceptible and infectious, respectively, β is called the infection coefficient. The standard incidence is $\frac{\beta SI}{N}$, where N is the total population size and β is called the daily contact rate. Another kind of incidence is the saturation incidence $\frac{\beta SI}{d+S}$, where d is a constant. When the number of susceptible S is large compared to d that incidence is approximately βI . This kind of incidence was proposed by Anderson and May [3, 37]. Many researchers [1, 14, 21, 26] have proposed a general incidence rate as $\frac{\beta I^p S}{1+\chi I^q}$.

On the other hand, interventions such as treatment, quarantine, vaccination etc. play an important role in controlling the disease spread. It is well known that a proper and timely treatment methodology can substantially reduce the effect of disease on society. In classical epidemic models, the treatment rate of infected individuals is assumed to be either constant or proportional to the number of infected individuals. There are many diseases for which treatment is available like flu, tuberculosis, measles [12, 15, 38] treatment is a useful tool to eradicate them. Several researchers [22, 30, 31, 36, 46], have studied the effect of treatment using different type of treatment functions. In classical models treatment rate is considered to be proportional to the number of infectives. This treatment rate is not suitable in case of large number of infectives due to availability of limited resources in a community. To study this effect of limited resources, Wang and Ruan [48] developed the constant removal rate (i.e. recovery per unit time), which is given by

$$g(I) = \begin{cases} r, & I > 0, \\ 0, & I = 1, \end{cases}$$

where r is a positive constant and I is the number of infected individuals. They studied stability analysis and showed that this model exhibits various bifurcations. Wang [49] in his study further improved the removal rate given by Wang and Ruan [48] by taking the following removal rate as a function [49]

$$g(I) = \begin{cases} rI, & 0 \le I \le I_0, \\ rI_0, & I > I_0, \end{cases}$$

where r and I_0 are positive constants. This removal rate shows that when the capacity of treatment is not reached then the removal rate is proportional to the number of infectives otherwise it takes the maximum capacity. Further, Zhou and

Fan [52] modified the treatment rate to Holling type-II

$$g(I) = \frac{\beta I}{1+\gamma I}, \qquad 0 \le \beta, \qquad 0 \le I, \qquad 0 \le \gamma.$$

In their study, they have shown that, with varying amount of medical resources and their supply efficiency, the target model admits both backward bifurcation and Hopf bifurcation. Dubey et al. [8] have also used Holling type-III treatment rate given by

$$g(I) = \frac{\beta I^2}{1+\gamma I^2}, \qquad 0 \le \beta, \qquad 0 \le I, \qquad 0 \le \gamma$$

and Holling type-IV treatment rate given by

$$g(I) = \frac{\beta I}{I^2/a + I + b}, \qquad 0 \le \beta, \qquad 0 \le a, \qquad 0 < b.$$

In recent years the use of approximate analytical methods has become very popular for solving a wide class of science and engineering problems involving algebraic, integro-differential, delay differential, integral equation, system of linear and nonlinear ordinary and partial differential equations such as strongly coupled reaction-difusion system [16], age structure population model [17], flow between two coaxial rotating disks [11], EIAV infection [18], q-difference equations [43], HIV infection CD4+ T-cell [20], fractional partial differential equations [35], inner-resonance of tangent nonlinear cushioning packaging system with critical components [19] and several others. The approximate analytical methods provide the solution in a rapidly convergent series with components that can be elegantly computed. Therefore, these methods are useful for obtaining both a closed form and the explicit solution and numerical approximation of linear and nonlinear differential equations.

The homotopy analysis method was first developed by Liao [27,28] who utilized the idea of homotopy in topology. It has been reported that homotopy analysis method, as an analytical method, has an advantage over perturbation methods in that it is not dependent on small or large parameters. Perturbation methods are based on the existence of small or large parameters and they cannot be applied to all nonlinear equations. Non-perturbative methods, such as δ-expansion and adomian decomposition method, are independent of small parameters. Both perturbation techniques and non-perturbative methods [17] cannot provide a simple procedure to adjust or control the convergence region and rate of convergence of given approximate series. Homotopy analysis method allows for fine tuning of convergence region and rate of convergence by allowing an auxiliary parameter to vary [19]. The proper choice of the initial condition, the auxiliary linear operator, and auxiliary parameter will guarantee the convergence of the homotopy analysis method solution series. Compared to the homotopy perturbation method [39] the homotopy analysis method solution series will be convergent by considering two factors: the auxiliary linear operator and initial guess.

The aim of this paper is to introduce and apply an effective method so-called homotopy analysis method to obtain a series solution of SIR epidemic model along Crowley-Martin incidence rate and Holling type-II treatment rate. Semi analytical schemes such as variational iteration method, adomian decomposition method, homotopy perturbation method, and homotopy analysis method have been widely

employed to solve various linear and nonlinear ordinary and partial differential equations. One of the advantages of the semi approximate analytical methods is that these methods generate an infinite series solution and, unlike finite difference methods, semi approximate analytical methods do not have the problem of rounding error. Therefore, in contrast to implicit finite difference methods that require the solution of systems of equation, the semi analytical schemes require only the solution of recursive process.

In the deterministic epidemic model, individuals in the population are assigned to different subgroups or compartments, these compartments are defined with respect to disease status of the epidemic. To the best knowledge of the authors, an SIR epidemic model with Crowley-Martin type functional response [6, 40] and Holling type-II treatment rate [8, 52] has not been considered. Taking these important facts into account, in this paper, we have analysed the SIR epidemic model by incorporating Crowley-Martin type functional response and Holling type-II treatment rate for better understanding of disease dynamics. Further, we evaluate the basic reproduction number $\mathfrak{R}_0$, analysed the dynamical behaviour of the model and also discussed the stability of the model. The stability analysis of the model has been done by Laselle's principle, Lyapunov function and Routh- Hurwitz criterion. Finally, homotopy analysis method in employed to obtained the series solution of the modified SIR or xyz model.

The present paper has been organized as follows: after the abstract and introduction, Section 2 discusses the formulation of the mathematical model and well-posedness of the model. In Section 3, we discuss the mathematical analysis of the proposed model along with equilibrium points and the stability of equilibrium points. Further, in Section 4, application of the homotopy analysis method is performed on the proposed model and the numerical simulations are done to validate the analytical studies. In Section 5, numerical results are given to illustrate the capability of HAM. In Section 6, we discuss on the solution obtained by using HAM and the convergence of the series solution along with the $\hbar$-curves is obtained. In this Section we also improve the solution obtained by applying the least squares method. Finally, Section 7 concludes all the major findings of the present research study.

2. Model Formulation

In the following section, we develop a mathematical model for the dynamics of susceptible-infected-recovered population by introducing the Crowley-Martin type functional response and Holling type-II treatment rate in the existing SIR or xyz epidemic model. In the compartmental epidemiological models, the total population is often divided into several disjoint classes. Here, it is assumed that the entire population is divided into three classes: susceptible individuals (x), infected individuals (y), and removed or recovered individuals (z). Susceptible individuals are those who are healthy and can contract disease under appropriate conditions. Infected individuals are the one who have contracted the disease and are now infected with it. These individuals are capable of transferring the disease to susceptible individuals via contacts. As time progresses, infected individuals lose infectivity and move to the removed or recovered compartment (by auto recovery due to immune response of the body or by treatment). These recovered individuals are immune to infectious microbes and thus do not acquire the disease again. The proposed model

is represented by the following system of non-linear ordinary differential equations

$$\begin{aligned} x^{'} &= \Lambda - \alpha_0 x - \frac{axy}{(1+bx)(1+cy)}, \\ y^{'} &= \frac{axy}{(1+bx)(1+cy)} - \alpha_0 y - \alpha_1 y - \alpha_2 y - \frac{\beta y}{1+\delta y}, \\ z^{'} &= \alpha_2 y - \alpha_0 z + \frac{\beta y}{1+\delta y}. \end{aligned} \tag{2.1}$$

The initial conditions for the above model are

$$x(0) = x_0 \geq 0, \qquad y(0) = y_0 \geq 0, \qquad z(0) = z_0 \geq 0. \tag{2.2}$$

In the above model (2.1), $x^{'} = \frac{dx}{dt}$, $y^{'} = \frac{dy}{dt}$ and $z^{'} = \frac{dz}{dt}$. Let the susceptibles be recruited at a constant rate Λ and α_0 be the natural death rate of the population in each class. Let α_1 be the death rate of infected individuals due to infection and α_2 be the natural recovery rate at which infected individuals get recovered by immunity to join the recovered class. We take the incidence rate as the Crowley-Martin type in the model (2.1) as

$$\kappa(x, y) = \frac{axy}{(1+bx)(1+cy)},$$

where a is a transmission rate and b is a measure of inhibition effect, such as preventive measure taken by susceptible individuals, and c is a measure of inhibition effect such as treatment with respect to infectives. The term

$$h(y) = \frac{\beta y}{1+\delta y}, \qquad \beta \geq 0, \qquad y \geq 0, \qquad \delta \geq 0,$$

represents Holling type-II treatment rate, where β and δ are non-negative constants and can be understood as treatment given to the infected individuals and limitation to the treatment availability, respectively. Unlike the Holling type-III treatment rate which grows first very fast and later on increases slowly with increase in number of infection and gets saturated to its maximum level $\frac{\beta}{\delta}$ (treatment capacity of community) due to limited availability of resources in the community.

From the above system of equations (2.1), we can infer that x and y are free from the effect of z. Thus it is enough to consider the following reduced system for the study

$$\begin{aligned} x^{'} &= \Lambda - \alpha_0 x - \frac{axy}{(1+bx)(1+cy)}, \\ y^{'} &= \frac{axy}{(1+bx)(1+cy)} - \lambda y - \frac{\beta y}{1+\delta y}, \end{aligned} \tag{2.3}$$

where $\lambda = \alpha_0 + \alpha_1 + \alpha_2$ and $x(0) = x_0 \geq 0,\ y(0) = y_0 \geq 0$.

3. Mathematical analysis of the model

3.1. Boundness and positivity of the solution

Theorem 3.1. *For all time $t \geq 0$, the solutions of the equations of system (2.3) are eventually confined in a positively invariant compact set*

$$\phi(x, y) = \{(x, y) \in \Re^2_+ : 0 \leq x + y \leq \frac{\Lambda}{\alpha_0},\ \ x, y \geq 0\}$$

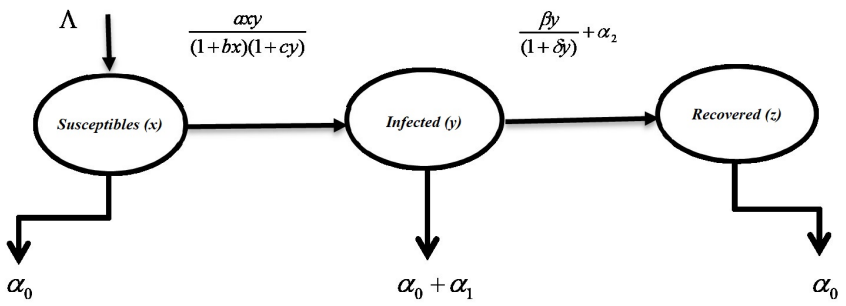

Figure 1. Schematic diagram of the modified SIR epidemic model.

Table 1. Parameters with their values for the proposed model [7, 8, 12, 33, 34, 37, 48, 49, 52]

Parameter symbol	Meaning	Values
Λ	Recruitment rate	2 person day^{-1}
α_0	Natural death rate of each sub-population	0.05 day^{-1}
α_1	Disease induced death rate of infected	0.001 day^{-1}
α_2	Recovery rate of infected due to auto immunity	0.002 person^{-1}
β	Treatment rate	0.02 person^{-1}
δ	Limitation rate in treatment availability	0.0004 person^{-1}
a	Transmission rate	0.004 person^{-1}
b	Inhibition rate due to susceptible	0.004 person^{-1}
c	Inhibition rate due to infected	0.002 person^{-1} day^{-1}
x_0	Initially susceptibles	20
y_0	Initially infected	15
z_0	Initial recovered	10

Proof. To prove this, let $\phi(x,y) = \{(x,y) \in \Re^2_+ : 0 \leq x+y \leq \frac{\Lambda}{\alpha_0}, \ \ x,y \geq 0\}$ be any solution of the system of equations (2.3) with non-zero initial conditions. Also let N be the total population available, then $N = x + y$ and

$$\frac{dN}{dt} = \frac{dx}{dt} + \frac{dy}{dt},$$

$$\frac{dN}{dt} = \Lambda - \alpha_0 N - (\alpha_1 + \alpha_2)y - \frac{\beta y}{1+\delta y}.$$

Then

$$N(t) = N(0)e^{-\alpha_0 t} + \frac{\Lambda}{\alpha_0}(1 - e^{-\alpha_0 t}). \tag{3.1}$$

Therefore, as $t \to \infty$ in equation (3.15), the total population $N \to \frac{\Lambda}{\alpha_0}$. Clearly, it has been proved that all the solutions of system (2.3) which initiate in $\Re^2_+$ confined in the region ϕ , i.e., the solutions are bounded in the interval $[0, \infty)$, i.e. $\lim\limits_{t\to\infty} Sup(x,y) \leq \frac{\Lambda}{\alpha_0}$. Furthermore, $N^{'} < 0$ if $N > \frac{\Lambda}{\alpha_0}$, where $N^{'} = \frac{dN}{dt}$. This shows that solutions of system (2.3) point towards the region ϕ. Hence ϕ is positively invariant and

solutions of system (2.3) are bounded. The Theorem 3.1 shows that all solutions of the model (2.3) are non-negative and bounded. Thus the model is biologically well behaved. In the next section, we show the existence of equilibrium points of system (2.3). □

3.2. Equilibria and their feasibility criteria

In this section, we discuss the local as well as the global stability of disease-free and endemic equilibrium of the modified SIR model by analyzing the corresponding characteristic equations respectively. By defining reasonable Lyapunov functions, we resolve the global dynamics of equilibria without requiring any extra conditions. We see that system (2.3) has only two equilibria:

Disease-free equilibrium E^0 : The equilibrium state with the absence of infection is known as disease-free equilibrium or zero equilibrium. The disease-free equilibrium has been always feasible, as in this equilibrium the infection dies out from the population. The disease-free equilibrium is given by $E^0 = (x_0, y_0) = (\frac{\Lambda}{\alpha_0}, 0)$ i.e., the state when infection $(y = 0)$ dies out from the population.

Endemic equilibrium E^*:The positive endemic equilibrium is that state of the system, where the infection spreads throughout the population and causes the disease persistence. For the system (2.3), the endemic equilibrium is considered as $E^* = (x^*, y^*)$. i.e., the state in which infection spreads in the susceptible population. We can infer from system (2.3) that the disease-free equilibrium E^0 is the equilibrium point which exists trivially. To study the local stability of the disease-free equilibrium, we compute the basic reproduction number by using next generation matrix method [9, 10, 29, 41].

Basic reproduction number: The basic reproduction number $\Re_0$ is arguably the most important quantity in infectious disease epidemiology. It is among the quantities most urgently estimated for emerging infectious diseases in outbreak situations, and its value provides insight when designing control interventions for established infections. From a theoretical point of view $\Re_0$ plays a vital role in the analysis of, and consequent insight from, infectious disease models. It is defined as the number of secondary cases produced by a typical infected individual during its entire period of infectiousness in a completely susceptible population. The basic reproduction number $\Re_0$ is the threshold quantity in the disease transmission that determines whether an infection will spread in a susceptible population or not. It shows that if $\Re_0 < 1$, then the disease/infection does not spread and the infection dies. On the other hand if $\Re_0 > 1$, then the disease persists in the whole population. We find $\Re_0$ using the next generation matrix [9, 10, 29, 41]. This next generation matrix approach is now illustrated by returning to the model compartments. we define $\theta^* = F(\theta) - V(\theta)$, where $\theta = [y, x]^T$, $F(\theta)$ is the matrix of new infection terms, and $V(\theta)$ is the matrix of transfer terms into compartment and out of compartment. At the disease-free equilibrium (DFE) matrices F and V are obtained as

$$F = \left[\frac{\partial f_i}{\partial \theta_j}\right]_{E^0}, \qquad V = \left[\frac{\partial v_i}{\partial \theta_j}\right]_{E^0}, \qquad i, j = 1, 2$$
$$f = \begin{bmatrix} \frac{axy}{(1+bx)(1+cy)} \\ 0 \end{bmatrix} \tag{3.2}$$

and

$$v = \begin{bmatrix} \lambda y + \frac{\beta y}{1+\delta y} \\ \alpha_0 x + \frac{axy}{(1+bx)(1+cy)} \end{bmatrix}. \tag{3.3}$$

Then

$$F = \begin{bmatrix} \frac{\alpha\Lambda}{\alpha_0+b\Lambda} & 0 \\ 0 & 0 \end{bmatrix} \tag{3.4}$$

and

$$V = \begin{bmatrix} \lambda+\beta & 0 \\ \frac{\alpha\Lambda}{\alpha_0+b\Lambda} & 0 \end{bmatrix}. \tag{3.5}$$

This implies

$$F = \begin{bmatrix} \frac{\alpha\Lambda}{\alpha_0+b\Lambda} & 0 \\ 0 & 0 \end{bmatrix} \tag{3.6}$$

and

$$V^{-1} = \begin{bmatrix} \frac{1}{\lambda+\beta} & 0 \\ \frac{\alpha\Lambda}{\alpha_0(\alpha_0+b\Lambda)(\Lambda+\beta)} & \frac{1}{\alpha_0} \end{bmatrix} \tag{3.7}$$

then

$$FV^{-1}(E^0) = \begin{bmatrix} \frac{\alpha\Lambda}{(\alpha_0+b\Lambda)(\Lambda+\beta)} & 0 \\ 0 & 0 \end{bmatrix}. \tag{3.8}$$

So FV^{-1} has eigenvalues 0 and $\Re_0$, where $\Re_0$ is the spectral radius (largest eigenvalue) given by

$$\Re_0 = \rho(FV^{-1}) = \frac{\alpha\Lambda}{(\beta+\lambda)(\alpha_0+b\Lambda)}.$$

When $\Re_0 < 1$, one infected person on average produces less than one newly infected person, and the infection population fails to spread and goes extinct, thus the system converges to the disease-free equilibrium E^0 . However, when $\Re_0 > 1$, one infected person on average gives rise to more than one newly infected persons, and the infection can spread, the system will converge to the positive endemic equilibrium E^*.

3.3. Local and Global Stability of the Equilibria

In this section, we discuss the local as well as global stability analysis of equilibrium points. For this, we state the results in the form of theorems and prove them.

3.4. Disease-free equilibrium E^0 and its stability

The global stability of the disease-free equilibrium E^0 is proved by using common quadratic and linear Lyapunov functions and LaSalle's invariance principle.

Theorem 3.2. *The disease-free equilibrium E^0 is locally asymptotically stable if $\Re_0 < 1$ and occurs as a saddle point with stable manifold locally in the x-direction and unstable manifold locally in the y-direction for $\Re_0 > 1$.*

Proof. In epidemiological sense, the above result depicts that small inflow of infected individuals will not be able to spread infection if $\Re_0 < 1$. In this case the spread of infection is dependent on initial sizes of sub-population. To prove the above Theorem 3.2, the general variatonal matrix and the matrices corresponding to each equilibrium point will be obtained. Therefore, the variational matrix is given by

$$\Gamma = \begin{bmatrix} -\alpha_0 - \frac{ay}{(1+cy)}\left(\frac{(1+bx)-bx}{(1+bx)^2}\right) & \frac{ax}{(1+by)}\left(\frac{(1+cy)-cy}{(1+cy)^2}\right) \\ \frac{ay}{(1+cy)}\left(\frac{(1+bx)-bx}{(1+bx)^2}\right) & \frac{ax}{(1+by)}\left(\frac{(1+cy)-cy}{(1+cy)^2}\right) - \lambda - \beta\frac{(1+\delta y)-\delta y}{(1+\delta y)^2} \end{bmatrix}. \tag{3.9}$$

Now at the disease-free equilibrium E^0,

$$\Gamma(E^0) = \begin{bmatrix} -\alpha_0 & -\frac{\alpha\Lambda}{\alpha_0+b\Lambda} \\ 0 & \frac{\alpha\Lambda}{\alpha_0+b\Lambda} - \lambda - \beta \end{bmatrix}, \tag{3.10}$$

$$\Gamma(E^0) = \begin{bmatrix} -\alpha_0 & -\frac{\alpha\Lambda}{\alpha_0+b\Lambda} \\ 0 & \left(\frac{\alpha\Lambda}{(\alpha_0+b\Lambda)(\lambda+\beta)} - 1\right)(\beta+\lambda) \end{bmatrix}, \tag{3.11}$$

$$\Gamma(E^0) = \begin{bmatrix} -\alpha_0 & -\frac{\alpha\Lambda}{\alpha_0+b\Lambda} \\ 0 & (\beta+\lambda)[\Re_0 - 1] \end{bmatrix}. \tag{3.12}$$

The eigenvalues of the variational matrix which is upper triangular at disease-free equilibrium E^0 are $\lambda_1 = -\alpha_0$ and $\lambda_2 = (\beta+\lambda)[\Re_0 - 1]$. Therefore, we have seen the eigenvalue $\lambda_1 = -\alpha_0$ is negative at disease-free equilibrium and the eigenvalue $\lambda_2 = (\beta+\lambda)[\Re_0 - 1]$ is negative if $\Re_0 < 1$. Again $\lambda_2 = (\beta+\lambda)[\Re_0 - 1] > 0$ if $\Re_0 > 1$. This proves the Theorem 3.2. □

Theorem 3.3. *The disease-free equilibrium E^0 is globally asymptotically stable in $\Re_2^+$ whenever $\Re_0 \leq 1$ and unstable otherwise.*

Proof. To prove this, let us define the positive definite function $\phi(x, y) : \Re_2^+ \to \Re_2^+$ by

$$\phi(x, y) = \frac{1}{(1+bx_0)}\left(-x_0 \ln\frac{x}{x_0} + x - x_0\right) + y \tag{3.13}$$

where $x_0 = \frac{\Lambda}{\alpha_0}$. Differentiating the above function with respect to the time t along the solutions of model (2.3), we get from equation (3.13)

$$\begin{aligned} \phi'(x, y) &= \frac{1}{(1+bx_0)}\left(x' - x_0\frac{1}{\frac{x}{x_0}}\frac{x'}{x_0}\right) + y', \\ \phi'(x, y) &= \frac{1}{(1+bx_0)}\left(\frac{x-x_0}{x}\right)x' + y', \\ \phi'(x, y) &= \frac{1}{(1+bx_0)}\left(\frac{x-x_0}{x}\right)\left(\Lambda - \alpha_0 x - \frac{axy}{(1+bx)(1+cy)}\right) \end{aligned}$$

$$+\left(\frac{axy}{(1+bx)(1+cy)}-\lambda y-\frac{\beta y}{(1+\delta y)}\right).$$

After simplification with some manipulations, the above equation leads to

$$\phi^{'}(x,y)=-\left[\frac{\alpha_0(x-x_0)^2}{x(1+bx_0)}+\left(\frac{\beta}{1+\delta y}+\frac{(\beta+\lambda)c}{1+cy}\right)y\right]+\frac{(\beta+\lambda)y}{1+cy}[\Re_0-1].$$

This shows $\phi^{'}(x,y)<0$ if $\Re_0\leq 1$ and $\forall x,y>0$ therefore $\phi^{'}(x,y)=0$ only when $x=x_0=\frac{\Lambda}{\alpha_0}$ and $y=y_0=0$, this implies that the maximum invariant set in $\{(x,y)\in\phi:\phi^{'}=0\}$ is the singleton set $[E^0=(x_0,y_0)]$. Hence by LaSalle's invariant principle [9,29,41], the disease-free equilibrium E^0 is globally asymptotically stable in $\Re_2^+$ whenever $\Re_0\leq 1$. It is apparent from the above result that the infection can be cleared from the population if the basic reproduction number is less than one which is independent of the initial concentrations of sub populations. □

3.5. Analysis at $\Re_0=1$

In this section, we analyze the behavior of system (2.3) when the basic reproduction number $\Re_0=1$. We notice that the Jacobian matrix of system (2.3) evaluated at $\Re_0=1$ and $a=a^*=\frac{(\beta+\lambda)(\alpha_0+b\Lambda)}{\Lambda}$ has a simple zero eigenvalue and another eigenvalue with negative real part. Stability behavior of equilibrium points at $\Re_0=1$ cannot be determined using linearization, so we use Center manifold theory [4,8,12,42]. In order to proceed, we state and prove the following Theorem 3.4 which provides the behavior of disease-free equilibrium E^0 at $\Re_0=1$.

Theorem 3.4. *The disease-free equilibrium E^0 changes its stability from stable to unstable at $\Re_0=1$ in $\Re_2^+$ and system (2.3) exhibits transcritical bifurcation with bifurcation parameter $a=a^*=\frac{(\beta+\lambda)(\alpha_0+b\Lambda)}{\Lambda}$.*

Proof. To prove this, the Jacobian matrix of the system (2.3) at disease-free equilibrium E^0 and bifurcation constant $a=a^*=\frac{(\beta+\lambda)(\alpha_0+b\Lambda)}{\Lambda}$ is given by

$$\Gamma=\begin{bmatrix}-\alpha_0 & -\frac{\alpha^*\Lambda}{\alpha_0+b\Lambda}\\ 0 & \frac{\alpha^*\Lambda}{\alpha_0+b\Lambda}-\lambda-\beta\end{bmatrix}. \tag{3.14}$$

Let $\xi=[\xi_1,\xi_2]$ and $\eta=[\eta_1,\eta_2]^T$ be the left eigenvector and right eigenvector of the Jacobian matrix corresponding to the zero eigenvalue respectively. Therefore, $\xi=[0,1]$ and $\eta=\left[-\frac{\alpha^*\Lambda}{\alpha_0(\alpha_0+b\Lambda)},1\right]^T$, then we have

$$\xi_1=0,\qquad \xi_2=1,\qquad \eta_1=\frac{\alpha^*\Lambda}{\alpha_0(\alpha_0+b\Lambda)},\qquad \eta_2=1.$$

Then from first Theorem of Castillo-Chavez and Song [4], the bifurcation constants μ_1 and μ_2 are given by

$$\mu_1=\sum_{l,m,n}^{2}\xi_l\eta_m\eta_n\left(\frac{\partial^2 f_l}{\partial x_m\partial y_n}\right)_{E^0,a^*},\qquad \mu_2=\sum_{l,m}^{2}\xi_l\eta_m\left(\frac{\partial^2 f_l}{\partial x_m\partial a^*}\right)_{E^0,a^*}.$$

The nonzero partial derivatives associated with the functions of the system (2.3) evaluated at $\Re_0 = 1$ and $a = a^*$ are

$$\frac{\partial f_2}{\partial y} = \frac{ax}{1+bx}\left[\frac{1}{(1+cy)^2}\right] - \lambda - \frac{\beta}{(1+\delta y)^2},$$
$$\frac{\partial^2 f_2}{\partial x \partial y} = \frac{a}{(1+cy)^2}\left[\frac{1}{(1+bx)}\right] = \frac{a^*\alpha_0^2}{(\alpha_0+b\Lambda)^2}.$$

This implies

$$\left(\frac{\partial^2 f_2}{\partial x \partial y}\right)_{E^0,a^*} = \frac{a^*\alpha_0^2}{(\alpha_0+b\Lambda)^2}.$$

Again

$$\frac{\partial^2 f_2}{\partial y^2} = \frac{-2acx}{(1+bx)(1+cy)^3} - \frac{2\beta\delta}{(1+\delta y)^3}.$$

This implies

$$\left(\frac{\partial^2 f_2}{\partial x \partial y}\right)_{E^0,a^*} = -2\left(\frac{a^*c\Lambda}{(\alpha_0+b\Lambda)} + \beta\delta\right).$$

Similarly,

$$\frac{\partial^2 f_2}{\partial y \partial a^*} = \frac{x}{1+bx}\left[\frac{1}{(1+cy)^2}\right].$$

This implies

$$\left(\frac{\partial^2 f_2}{\partial x \partial a^*}\right)_{E^0,a^*} = \frac{\Lambda}{(\alpha_0+b\Lambda)}.$$

Now

$$\mu_1 = \sum_{l,m,n}^{2} \xi_l \eta_m \eta_n \left(\frac{\partial^2 f_l}{\partial x_m \partial y_n}\right)_{E^0,a^*},$$
$$\mu_1 = \sum_{l,m,n}^{2} \xi_l \eta_m \eta_n \left(\frac{\partial^2 f_l}{\partial x_m \partial y_n}\right)_{E^0,a^*}$$
$$= \xi_2\left[\mu_1\mu_2\frac{a^*\alpha_0^2}{(\alpha_0+b\Lambda)^2} + \mu_2^2\left(-2\left(\frac{a^*c\Lambda}{\alpha_0+b\Lambda}+\beta\delta\right)\right)\right]$$
$$= \left[\frac{a^*\alpha_0^2}{(\alpha_0+b\Lambda)^2}\frac{a^*\Lambda}{\alpha_0(\alpha_0+b\Lambda)} + \left(-2\left(\frac{a^*c\Lambda}{\alpha_0+b\Lambda}+\beta\delta\right)\right)\right]$$
$$= \left[\frac{a^*\alpha_0}{(\alpha_0+b\Lambda)^2}\frac{a^*\Lambda}{(\alpha_0+b\Lambda)} + \left(-2\left(\frac{a^*c\Lambda}{\alpha_0+b\Lambda}+\beta\delta\right)\right)\right].$$

This implies

$$\mu_1 = \left[-\frac{a^*\Lambda}{(\alpha_0+b\Lambda)}\left(\frac{a^*\alpha_0}{(\alpha_0+b\Lambda)^2} + 2\left(c+\beta\delta\frac{\alpha_0+b\Lambda}{a^*\Lambda}\right)\right)\right] < 0$$

and

$$\mu_2 = \sum_{l,m}^{2} \xi_l \eta_m \left(\frac{\partial^2 f_l}{\partial x_m \partial a^*}\right)_{E^0,a^*}$$

$$= \xi_2 \left(\eta_2 \frac{\partial^2 f_l}{\partial y \partial a^*} \right)_{E^0, a^*}$$
$$= \frac{\Lambda}{(\alpha_0 + b\Lambda)} > 0.$$

This shows that, at $\Re_0 = 1$ and $a = a^* = \frac{(\beta+\lambda)(\alpha_0+b\Lambda)}{\Lambda}$, $\mu_1 < 0$ and $\mu_2 > 0$. This implies from first Theorem of Castillo-Chavez and Song [4], the disease-free equilibrium E^0 changes its stability from stable to unstable when $\Re_0$ crosses the threshold value i.e., 'one' and exhibits transcritical bifurcation. This proves the above Theorem 3.4. □

3.6. Endemic equilibrium $E^* = (x^*, y^*)$ and its stability

In this section, we will now show the existence of endemic equilibrium $E^* = (x^*, y^*)$ by using isocline method under certain threshold value or conditions. Let us assume that

$$\Phi(x, y) = \Lambda - \alpha_0 x - \frac{axy}{(1 + bx)(1 + cy)}, \tag{3.15}$$

$$\Psi(x, y) = \frac{axy}{(1 + bx)(1 + cy)} - \lambda - \frac{\beta y}{1 + \delta y}. \tag{3.16}$$

From the first isocline (3.15), we observe that if $y = 0$ then $x = \frac{\Lambda}{\alpha_0} = x_0$ and

$$\frac{dx}{dy} = -\frac{\frac{\partial \Phi}{\partial y}}{\frac{\partial \Phi}{\partial x}}$$

where

$$\frac{\partial \Phi}{\partial x} = -\alpha_0 - \frac{ay}{(1 + bx)^2(1 + cy)}, \qquad \frac{\partial \Phi}{\partial y} = -\frac{ax}{(1 + bx)(1 + cy)^2}.$$

This implies that

$$\frac{dx}{dy} = -\frac{\frac{ax}{(1+bx)(1+cy)^2}}{\alpha_0 + \frac{ay}{(1+bx)^2(1+cy)}} < 0.$$

Hence, it is seen that the first isocline (3.15) is decreasing function of y. Now, from the second isocline (3.16), we have the following observations $y = 0$ then $x = \frac{\beta+\lambda}{a-b(\beta+\lambda)} = \hat{x}$ that implies

$$\hat{x} > 0 \qquad \text{if} \qquad a > b(\beta + \lambda), \tag{3.17}$$

and

$$\frac{dx}{dy} = -\frac{\frac{\partial \Psi}{\partial y}}{\frac{\partial \Psi}{\partial x}}$$

where

$$\frac{\partial \Psi}{\partial x} = -\frac{a}{(1 + bx)^2(1 + cy)}, \qquad \frac{\partial \Psi}{\partial y} = -\frac{acx}{(1 + bx)(1 + cy)^2} + \frac{\beta\delta}{(1 + \delta y)^2}.$$

This implies that

$$\frac{dx}{dy} = \frac{\frac{acx}{(1+bx)(1+cy)^2} - \frac{\beta\delta}{(1+\delta y)^2}}{\frac{a}{(1+bx)^2(1+cy)}}.$$

It can be seen from the above expression that the denominator is always positive and the numerator is positive if

$$\frac{acx}{(1+bx)(1+cy)^2} > \frac{\beta\delta}{(1+\delta y)^2}.$$

After substituting the maximum values of x and y $\left(i.e. x = \frac{\Lambda}{\alpha_0}, y = 0\right)$, we get the inequality $\frac{ac\Lambda}{\alpha_0+b\Lambda} > \beta\delta$. Thus $\frac{dx}{dy}$ is positive if $\frac{ac\Lambda}{\alpha_0+b\Lambda} > \beta\delta$ and $\Psi(x,y)$ is increasing function of y. This implies that the two isoclines (3.15) and (3.16) intersects at a unique point $E^* = (x^*, y^*)$ i.e., if $\Re_0 = \frac{a\Lambda}{(\beta+\lambda)(\alpha_0+b\Lambda)} > 1$. Thus we can state the existence and uniqueness of the endemic equilibrium in the Theorem 3.5.

Theorem 3.5. *The endemic equilibrium $E^* = (x^*, y^*)$ exists if the following inequalities hold true*

$$\frac{ac\Lambda}{\alpha_0 + b\Lambda} > \beta\delta, \tag{3.18}$$

$$\Re_0 = \frac{a\Lambda}{(\beta+\lambda)(\alpha_0+b\Lambda)} > 1. \tag{3.19}$$

Proof. It may be noted that if condition (3.19) hold, then condition (3.17) is satisfied by default. Also, if the condition (3.18) fails, then $\frac{dx}{dy}$ for isocline (3.16) may be positive or negative depending upon the values of parameters. In such a case there may exist more than one endemic equilibrium. □

3.7. Uniform persistence of the system

The uniform persistence of system (2.3) in biological sense means that the subpopulations exists always and will never extinct if they are present initially. Now in the next Theorem 3.6, we obtain the conditions for the uniform persistence of system (2.3) as

Theorem 3.6. *From the Theorem 3.1 which holds, lets us assume that the following inequality is satisfied:*

$$\max\left\{\frac{a\Lambda}{(\alpha_0+b\Lambda)(\alpha_0+c\Lambda)}, \frac{\beta}{(\alpha_0+\delta\Lambda)}\right\} < 1.$$

Then the system (2.3) is uniformly persistent.

Proof. To prove this, we have $x(0) > 0$ and $y(0) > 0$. Then the system (2.3) is said to be uniformly persistent [45,47] if there exists positive constants K_1 and K_2 such that

$$K_1 \le \lim_{t\to\infty} \inf x(t) \le \lim_{t\to\infty} \sup x(t) \le K_2,$$
$$K_1 \le \lim_{t\to\infty} \inf y(t) \le \lim_{t\to\infty} \sup y(t) \le K_2.$$

Now from Theorem 3.1, we have

$$\lim_{t\to\infty}\sup x(t) \leq \frac{\Lambda}{\alpha_0},$$
$$\lim_{t\to\infty}\sup y(t) \leq \frac{\Lambda}{\alpha_0}.$$

This implies, for any $\epsilon > 0$ there exists a $T > 0$ such that

$$x(t) \leq \frac{\Lambda}{\alpha_0} + \epsilon = \hat{x_1},$$
$$y(t) \leq \frac{\Lambda}{\alpha_0} + \epsilon = \hat{x_1}.$$

From the equation first of model (2.3), we have

$$\frac{dx}{dt} \geq \Lambda - \alpha_0 x - \frac{a\hat{x_1}^2}{(1+b\hat{x_1})(1+c\hat{x_1})}.$$

This implies that

$$\lim_{t\to\infty}\inf x(t) \geq \frac{1}{\alpha_0}\left(\Lambda - \frac{a\hat{x_1}^2}{(1+b\hat{x_1})(1+c\hat{x_1})}\right)$$

which is true for every sufficiently small $\epsilon > 0$. Hence for large t , it follows that

$$\lim_{t\to\infty}\inf x(t) \geq \frac{\Lambda}{\alpha_0}\left(1 - \frac{a\Lambda}{(1+b\Lambda)(1+c\Lambda)}\right) = \hat{x_2}$$

and $\hat{x_2} > 0$ if $\frac{a\Lambda}{(1+b\Lambda)(1+c\Lambda)} < 1$ or $\Re_0 < \frac{\alpha_0+c\Lambda}{\beta+\lambda}$.
Again from the system (2.3), we have

$$\frac{d}{dt}(x+y) \geq \Lambda - \lambda_m(x+y) - \frac{\beta y}{1+\delta y}$$

where $\lambda_m = \max\{\alpha_0, \lambda\}$.
This implies

$$\lim_{t\to\infty}\inf(x(t)+y(t)) \geq \frac{\Lambda}{\lambda_m}\left(1 - \frac{\beta}{(\alpha_0+\delta\Lambda)}\right) = \hat{x_3}.$$

This shows that $\bar{x_3} > 0$ if $\frac{\beta}{(\alpha_0+\delta\Lambda)} < 1$. This proves the above Theorem 3.6. □

Now we will discuss the local and global stability of the endemic equilibrium $E^* = (x^*, y^*)$ for that we state and prove the following Theorem 3.7.

Theorem 3.7. *The endemic equilibrium E^* is locally asymptotically stable iff the following inequalities holds true*

$$\frac{ax^*}{(1+bx^*)(1+cy^*)^2} < Q_1, \tag{3.20}$$
$$\frac{a\alpha_0 x^*}{(1+bx^*)(1+cy^*)^2} < Q_2 \tag{3.21}$$

where

$$Q_1 = \left[\alpha_0 + \lambda + \frac{\beta}{(1+\delta y^*)^2} + \frac{ay^*}{(1+bx^*)^2(1+cy^*)}\right],$$
$$Q_2 = \left[\left(\alpha_0 + \frac{ay^*}{(1+bx^*)^2(1+cy^*)}\right)\left(\lambda + \frac{\beta}{(1+\delta y^*)^2}\right)\right].$$

Proof. To prove the local stability of the endemic equilibrium $E^* = (x^*, y^*)$, the Jacobian matrix associated with the system (2.3) at the endemic equilibrium E^* is given by

$$\Gamma^*(E^*) = \begin{bmatrix} -\alpha_0 - \frac{ay^*}{(1+bx^*)^2(1+cy^*)} & -\frac{ax^*}{(1+bx^*)(1+cy^*)^2} \\ \frac{ay^*}{(1+bx^*)^2(1+cy^*)} & \frac{ax^*}{(1+bx^*)(1+cy^*)^2} - \lambda - \frac{\beta}{(1+\delta y^*)^2} \end{bmatrix}.$$

After simplification, we get the characteristic polynomial associated with the Jacobian matrix at the endemic equilibrium E^* as

$$\Delta^2 + \xi_1\Delta + \xi_2 = 0, \tag{3.22}$$

where

$$\xi_1 = \left[\alpha_0 + \frac{ay^*}{(1+bx^*)^2(1+cy^*)} + \frac{ax^*}{(1+bx^*)(1+cy^*)^2} + \lambda + \frac{\beta}{(1+\delta y^*)^2}\right],$$
$$\xi_2 = \left[\left(\alpha_0 + \frac{ay^*}{(1+bx^*)^2(1+cy^*)}\right)\left(\lambda + \frac{\beta}{(1+\delta y^*)^2}\right) - \frac{a\alpha_0 x^*}{(1+bx^*)(1+cy^*)^2}\right].$$

According to the Routh-Hurwitz criteria, It can be seen that the eigenvalues of the Jacobian matrix at the endemic equilibrium have negative real parts if and only if $\xi_1 > 0$ and $\xi_2 > 0$. This implies that the endemic equilibrium $E^* = (x^*, y^*)$ is locally asymptotically stable if and only if inequalities (3.20) and (3.21) hold true. It can be seen that conditions (3.20) and (3.21) holds if

$$\frac{ax^*}{(1+bx^*)(1+cy^*)^2} < \lambda + \frac{\beta}{(1+\delta y^*)^2}.$$

□

In the next Theorem 3.8, we show the endemic equilibrium E^* is globally asymptotically stable. The globally asymptotic stability of the endemic equilibrium E^* is proved by constructing a global Lyapunov function. We obtain the Lyapunov function of a suitable combination of Volterra type functions

Theorem 3.8. *If $\Re_0 > 1$ then the unique endemic equilibrium $E^* = (x^*, y^*)$ is globally asymptotically stable in the interior of ϕ.*

Proof. To prove the global stability of endemic equilibrium, let us define a Lyapunov function $\hat{\phi} : \{(x, y) \in \phi : x, y > 0\} \to \Re$ by

$$\hat{\phi}(x, y) = \left(x - x^* - x^* \ln \frac{x}{x^*}\right) + \left(y - y^* - y^* \ln \frac{y}{y^*}\right). \tag{3.23}$$

This function is defined, continuous and positive definite for all $x, y > 0$. It can be verified that the function $\hat{\phi}(x, y)$ takes the value $\hat{\phi}(x, y) = 0$ at the steady state E^*,

and thus, the global minimum of $\hat{\phi}(x,y)$ occurs at the endemic steady state E^* . Since (x^*,y^*) is an endemic steady state point of (2.3), we have

$$\Lambda = \alpha_0 x^* + \frac{ax^*y^*}{(1+bx^*)(1+cy^*)}, \tag{3.24}$$

$$\frac{ax^*y^*}{(1+bx^*)(1+cy^*)} = \lambda y^* + \frac{\beta y^*}{(1+\delta y^*)}. \tag{3.25}$$

Computing the derivative of $\hat{\phi}(x,y)$ along the solutions of system (2.3), we obtain

$$\begin{aligned}
\hat{\phi}'(x,y) &= \left(\frac{x-x^*}{x}\right)x' + \left(\frac{y-y^*}{y}\right)y',\\
\hat{\phi}'(x,y) &= \left(\frac{x-x^*}{x}\right)\left[\Lambda - \alpha_0 x + \frac{axy}{(1+bx)(1+cy)}\right]\\
&\quad + \left(\frac{y-y^*}{y}\right)\left[\frac{axy}{(1+bx)(1+cy)} - \lambda y - \frac{\beta y}{(1+\delta y)}\right],\\
\hat{\phi}'(x,y) &= \left(\frac{x-x^*}{x}\right)\left[\alpha_0 x^* + \frac{ax^*y^*}{(1+bx^*)(1+cy^*)} - \alpha_0 x - \frac{axy}{(1+bx)(1+cy)}\right]\\
&\quad + \left(\frac{y-y^*}{y}\right)\left[\lambda y^* + \frac{\beta y^*}{(1+\delta y^*)} - \lambda y - \frac{\beta y}{(1+\delta y)}\right].
\end{aligned}$$

This implies

$$\hat{\phi}'(x,y) \le -\frac{(x-x^*)^2}{x}\left[\alpha_0 x + \frac{ay}{(1+bx)(1+cy)}\right] - \frac{(y-y^*)^2}{y}\left[\lambda + \frac{\beta}{(1+\delta y)}\right]. \tag{3.26}$$

Therefore, $\hat{\phi}'(x,y) \le 0$ for all $x,y>0$, where the equality $\hat{\phi}'(x,y)=0$ holds only when $x=x^*$ and $y=y^*$. It is easy to see that the endemic equilibrium E^* is the only largest invariant set in $\{(x,y)\in\phi : \hat{\phi}'(x,y)=0\}$. Therefore, by LaSalles invariance principle [9,29,41], the endemic equilibrium E^* is globally asymptotically stable in the interior of ϕ. This proves that the endemic equilibrium E^* is globally asymptotically stable and hence the proof of the Theorem 3.8. □

Theorem 3.9. *The model (2.3) does not have any periodic solution in the interior of the positive quadrant of the xy-plane if the following inequalities hold* $\beta c > \beta\delta$.

Proof. To prove the above Theorem, let us take the following real valued function in the interior of positive quadrant of the xy-plane as

$$\Pi(x,y) = \frac{(1+bx)(1+cy)}{xy} > 0. \tag{3.27}$$

Consider

$$\begin{aligned}
\Pi_1(x,y) &= \Lambda - \alpha_0 x - \frac{axy}{(1+bx)(1+cy)},\\
\Pi_2(x,y) &= \frac{axy}{(1+bx)(1+cy)} - \lambda y - \frac{\beta y}{(1+\delta y)},
\end{aligned}$$

$$
\begin{aligned}
div(\Pi\Pi_1, \Pi\Pi_2) &= \frac{\partial}{\partial x}(\Pi\Pi_1) + \frac{\partial}{\partial y}(\Pi\Pi_2) \\
&= \frac{\partial}{\partial x}\left(\frac{\Lambda(1+bx)(1+cy)}{xy} - \alpha_0\frac{(1+bx)(1+cy)}{xy} - a\right) \\
&\quad + \frac{\partial}{\partial y}\left(a - \frac{\Lambda(1+bx)(1+cy)}{xy} - \frac{\beta y}{(1+\delta y)}\frac{1+cy}{y}\right), \\
div(\Pi\Pi_1, \Pi\Pi_2) &= -\Lambda\frac{1+cy}{x^2y} - \alpha_0 b\frac{1+cy}{y} - \lambda c\frac{1+bx}{x} - \frac{1+bx}{x(1+\delta y)^2}(\beta c - \beta\delta).
\end{aligned} \tag{3.28}
$$

It can be seen that the above expression is not equal to zero and this will not change sign in the positive quadrant of the xy-plane if the inequality $\beta c > \beta\delta$ holds. Then from Dulac's criterion [42], we can say that model (2.3) does not have any periodic solution in the interior of the positive quadrant of the xy-plane. The meaning of the above Theorem 3.9 in epidemiological sense is that if the inequality $\beta c > \beta\delta$ holds good then the disease will not reoccur in the population. □

4. Homotopy analysis solution

4.1. Basic idea

As in the proposed SIR epidemic model explored in this study, many practical situations in engineering, biology and science can be modeled with different types of systems of ordinary differential equations of the form

$$
\theta_i = f_i(t, \theta_1, \theta_2, \ldots, \theta_n), \qquad \theta_i(t_0) = \theta_{i,0}, \quad i = 1, 2, \ldots, n. \tag{4.1}
$$

As per the homotopy analysis method proposed by Liao [27], each equation of the system (4.1) is written in the form

$$
N_i[(u_1(\theta_1, t), u_2(\theta_2, t), \ldots, u_n(\theta_n, t)] \qquad i = 1, 2, \ldots, n \tag{4.2}
$$

where $N_1, N_2, \ldots, N_n$ are nonlinear operators, θ and t denote the independent variables and $u_1, u_2, \ldots, u_n$ are unknown functions. For simplicity, we ignore all boundary or initial conditions, which can be treated in a similar way. By means of the homotopy analysis method, we first construct the so-called zeroth-order deformation equation

$$
(1-q)L[\psi_i(t;q) - u_{i,0}(t)] = q\hbar H_i(t) N_i[\psi_1(t;q), \psi_2(t;q), \ldots, \psi_n(t;q)], i = 1, 2, \ldots, n \tag{4.3}
$$

where $q \in [0, 1]$ is the embedding parameter, $\hbar \neq 0$ is an auxiliary artificial parameter, L is an auxiliary linear operator, $\psi_i(t;q)$ are unknown functions, , $u_i(\theta_0, t) = u_{i,0}(t)$ is an initial guess of $\psi_i(t;q)$ and H_i denotes a nonzero auxiliary function. It is important to emphasize that, in the frame of homotopy analysis method, there is a great freedom to choose auxiliary entities such as $\hbar$, $H_i(t)$ and L base functions for the representation of the solution $u_i(t)$. Specifically, we can use in the construction of the solution $u_i(t)$ base functions such as polynomials, exponentials, rational functions, etc. It is obvious that when the embedding parameter $q = 0$ and $q = 1$ equation (4.3) becomes

$$
q(t;0) = u_{i,0}(t) \qquad \text{and} \qquad q_i(t;1) = u_i(t) \quad i = 1, 2, \ldots, n
$$

respectively. Thus as q increases from 0 to 1 , the solution varies from the initial guess $u_{i,0}(t)$ to the solution $u_i(t)$. Expanding $\psi_i(t;q)$ in Taylor series with respect to q, one obtain

$$\psi_i(t;q) = u_{i,0}(t) + \sum_{m=1}^{\infty} u_{i,m}(t)q^m \qquad i = 1, 2, \ldots, n \tag{4.4}$$

where

$$u_{i,m}(t) = \frac{1}{m!} \left. \frac{\partial^m \psi_i(t;q)}{\partial q^m} \right|_{q=0}.$$

The convergence of the series (4.4) depends upon the auxiliary artificial parameter, the auxiliary linear operators and the base functions. If these are properly chosen then at $q = 1$, the series is convergent and one has

$$u_i(t) = \psi_i(t;1) = u_{i,0}(t) + \sum_{m=1}^{\infty} u_{i,m}(t) \qquad i = 1, 2, \ldots, n \tag{4.5}$$

which must be one of the solutions of the original nonlinear equations, as proven by Liao [27]. Define the vectors

$$\vec{u_n} = \{u_1(t), u_2(t), \ldots, u_n(t)\}.$$

Differentiate the zeroth-order deformation equation (4.3) m-times with respect to q and then dividing them by $m!$ and finally setting $q = 0$, we get the following m^{th}-order deformation equation

$$L[u_{i,m}(t) - \chi_m u_{i,m-1}(t)] = \hbar H_i(t) \Re_{i,m}[u_{1,m-1}(t), u_{2,m-1}(t), \ldots, u_{i,m-1}(t)], i = 1, 2, \ldots, n \tag{4.6}$$

where

$$\begin{aligned} &\Re_{i,m}[u_{1,m-1}(t), u_{2,m-1}(t), \ldots, u_{i,m-1}(t)] \\ &= \frac{1}{(m-1)!} \left. \frac{\partial^{m-1} N_i[(u_1(\theta_1,t), u_2(\theta_2,t), \ldots, u_n(\theta_n,t)]}{\partial q^{m-1}} \right|_{q=0} \end{aligned}$$

and

$$\chi_m := \begin{cases} 0, & m \leq 1, \\ 1, & m > 1. \end{cases}$$

A one-parameter family of explicit series solutions is obtained by solving the linear equation (4.6). It should be emphasized that $u_{i,m}(t)$ for $m \geq 1$ is governed by the linear equation (4.6) with linear boundary conditions that comes from the original problem, which can be solved by the symbolic computation software such as MATLAB, Mathematica or Maple. In this way the homotopy analysis method converts a complicated nonlinear problem into simpler linear sub-problems. If equation (4.1) admits unique solution, then this method will produce the unique solution. If equation (4.1) does not possess a unique solution, the homotopy analysis method will give a solution among many other possible solutions.

4.2. Application of HAM for SIR or xyz modified model

Mostly researchers focus only on discussing the stability analysis, bifurcation analysis and the limit cycles of the system and thus giving the qualitative analysis of the system. The direct and simple numerical simulation is lack. So finding explicit analytic solution of the system is extremely important in epidemiology. In this section, we employ the homotopy analysis method [27,28,32] to obtain an approximate solution of the proposed SIR epidemic model. To construction the homotopy analytical solution for the proposed modified SIR model, we nondimensionalize our system (2.1) by using the following rescaling [13,45,50]

$$x = x_1, \qquad y = x_2, \qquad z = x_3.$$

subject to the initial conditions

$$x_1(0) = IC_1, \qquad x_2(0) = IC_2, \qquad x_3(0) = IC_3.$$

Following the homotopy analysis method, it is straightforward to choose

$$x_{1,0}(0) = IC_1, \qquad x_{2,0}(0) = IC_2, \qquad x_{3,0}(0) = IC_3.$$

as our initial approximations of $x_1(t)$, $x_2(t)$ and $x_3(t)$, respectively. In this work we will use

$$IC_1 = 20, \qquad IC_2 = 15, \qquad IC_3 = 10.$$

The dimensionless equations for system (2.1) are written as [13,45,50]

$$\begin{aligned}
&\frac{dx_1}{dt} + \frac{dx_1}{dt}[a_{1,1}x_1 + a_{1,2}x_2 + a_{1,3}x_1x_2] + x_1[a_{1,4} + a_{1,6}x_1] \\
&+ x_2[a_{1,5} + a_{1,8}x_1 + a_{1,7}x_1^2] + a_{1,9} = 0, \\
&\frac{dx_2}{dt} + \frac{dx_2}{dt}[a_{2,1}x_2 + a_{2,2}x_1 + a_{2,3}x_1x_2 + a_{2,4}x_2^2 + a_{2,5}x_1x_2^2] \\
&+ x_2[a_{2,6} + a_{2,7}x_2 + a_{2,10}x_2^2] + x_1x_2[a_{2,8} + a_{2,9}x_2 + a_{2,11}x_2^2] = 0, \\
&\frac{dx_3}{dt} + \frac{dx_3}{dt}[a_{3,1}x_1 + a_{3,2}x_2 + a_{3,3}x_1x_2 + a_{3,4}x_2^2 + a_{3,5}x_1x_2^2] \\
&+ x_2[a_{3,6} + a_{3,7}x_2 + a_{3,8}x_2^2] + x_1x_2[a_{3,9} + a_{3,10}x_2 + a_{3,11}x_2^2] + a_{3,12}x_1x_2x_3 \\
&+ x_3[a_{3,14} + a_{3,15}x_1 + a_{3,13}x_2] - x_2^2x_3[a_{3,16} + a_{3,17}x_1] = 0,
\end{aligned} \tag{4.7}$$

Due to governing equations, we choose the auxiliary linear operators

$$L[\psi_i(t;q)] = \frac{\partial \psi_i(t;q)}{\partial t}$$

with the property $L[C_i] = 0$, where C_i are integral constants (hereafter $i = 1, 2, 3$). We define the homotopy maps as [27,28,32]

$$\begin{aligned}
\Psi_1(\psi_1(t;q), \psi_2(t;q), \psi_3(t;q)) =& (1-q)L[\psi_1(t;q) - x_{1,0}(t)] \\
&- q\hbar H_1(t)N_1[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q)], \\
\Psi_2(\psi_1(t;q), \psi_2(t;q), \psi_3(t;q)) =& (1-q)L[\psi_2(t;q) - x_{2,0}(t)] \\
&- q\hbar H_2(t)N_2[\psi_1(t;q), \psi_2(t;q), \psi_3(t;q)],
\end{aligned}$$

$$\Psi_3(\psi_1(t;q),\psi_2(t;q),\psi_3(t;q)) = (1-q)L[\psi_3(t;q) - x_{3,0}(t)] - q\hbar H_3(t)N_3[\psi_1(t;q),\psi_2(t;q),\psi_3(t;q)],$$

where $\hbar \neq 0$ and $H_i(t) \neq 0$ denote the so-called auxiliary parameter and auxiliary function, respectively and due to system (4.7), the nonlinear operators N_1, N_2 and N_3 are defined as

$$\begin{aligned}
N_1[\psi_1(t;q),\psi_2(t;q),\psi_3(t;q)] =& \frac{d\psi_1(t;q)}{dt} + \frac{d\psi_1(t;q)}{dt}[a_{1,1}\psi_1(t;q) + a_{1,2}\psi_2(t;q) \\
&+ a_{1,3}\psi_1(t;q)\psi_2(t;q)] + \psi_1(t;q)[a_{1,4} + a_{1,6}\psi_1(t;q)] \\
&+ \psi_2(t;q)[a_{1,5} + a_{1,8}\psi_1(t;q) + a_{1,7}\psi_1^2(t;q)] \\
&+ a_{1,9}, \\
N_2[\psi_1(t;q),\psi_2(t;q),\psi_3(t;q)] =& \frac{d\psi_2(t;q)}{dt} + \frac{d\psi_2(t;q)}{dt}[a_{2,1}\psi_2(t;q) + a_{2,2}\psi_1(t;q) \\
&+ a_{2,3}\psi_1(t;q)\psi_2(t;q) + a_{2,4}\psi_2^2(t;q) \\
&+ a_{2,5}\psi_1(t;q)\psi_2^2(t;q)] + \psi_2(t;q)[a_{2,6} + a_{2,7}\psi_2(t;q) \\
&+ a_{2,10}\psi_2^2(t;q)] + \psi_1(t;q)\psi_2(t;q)[a_{2,8} \\
&+ a_{2,9}\psi_2(t;q) + a_{2,11}\psi_2^2(t;q)] = 0,
\end{aligned}$$

$$\begin{aligned}
N_3[\psi_1(t;q),\psi_2(t;q),\psi_3(t;q)] =& \frac{d\psi_3(t;q)}{dt} + \frac{d\psi_3(t;q)}{dt}[\\
&a_{3,1}\psi_1(t;q) + a_{3,2}\psi_2(t;q) + a_{3,3}\psi_1(t;q)\psi_2(t;q) \\
&+ a_{3,4}\psi_2^2(t;q) + a_{3,5}\psi_1(t;q)\psi_2^2(t;q)] \\
&+ \psi_2(t;q)[a_{3,6} + a_{3,7}\psi_2(t;q) + a_{3,8}\psi_2^2(t;q)] \\
&+ \psi_1(t;q)\psi_2(t;q)[a_{3,9} + a_{3,10}\psi_2(t;q) + a_{3,11}\psi_2^2(t;q)] \\
&+ a_{3,12}\psi_1(t;q)\psi_2(t;q)\psi_3(t;q) + \psi_3(t;q)[a_{3,14} \\
&+ a_{3,15}\psi_1(t;q) + a_{3,13}\psi_2(t;q)] \\
&- \psi_2^2(t;q)\psi_3(t;q)[a_{3,16} + a_{3,17}\psi_1(t;q)] = 0.
\end{aligned}$$

Clearly, when $q = 0$, we have the homotopy maps are

$$\begin{aligned}
\Psi_1(\psi_1(t;0),\psi_2(t;0),\psi_3(t;0)) &= L[\psi_1(t;q) - x_{1,0}(t)], \\
\Psi_2(\psi_1(t;0),\psi_2(t;0),\psi_3(t;0)) &= L[\psi_2(t;q) - x_{2,0}(t)], \\
\Psi_3(\psi_1(t;0),\psi_2(t;0),\psi_3(t;0)) &= L[\psi_3(t;q) - x_{3,0}(t)]
\end{aligned}$$

and when $q = 1$ will be

$$\begin{aligned}
\Psi_1(\psi_1(t;1),\psi_2(t;1),\psi_3(t;1)) &= -\hbar H_1(t)N_1[\psi_1(t;1),\psi_2(t;1),\psi_3(t;1)], \\
\Psi_2(\psi_1(t;1),\psi_2(t;1),\psi_3(t;1)) &= -\hbar H_2(t)N_2[\psi_1(t;1),\psi_2(t;1),\psi_3(t;1)], \\
\Psi_3(\psi_1(t;1),\psi_2(t;1),\psi_3(t;1)) &= -\hbar H_3(t)N_3[\psi_1(t;1),\psi_2(t;1),\psi_3(t;1)].
\end{aligned}$$

Thus, by using the embedding parameter $q \in [0,1]$ and $\hbar$ the non-zero auxiliary parameter, we construct a family of equations the zeroth-order deformation equations in the following form

$$(1-q)L[\psi_i(t;q) - u_{i,0}(t)] = q\hbar H_i(t)N_i[\psi_1(t;q),\psi_2(t;q),\psi_3(t;q)], \quad i = 1,2,3 \quad (4.8)$$

subject to the initial conditions

$$\psi_1(0;q) = 20, \qquad \psi_2(0;q) = 15, \qquad \psi_3(0;q) = 10.$$

For $q = 0$ and $q = 1$, the above zeroth-order Eq. (4.8) have the solutions

$$\psi_1(t;0) = x_{1,0}(t), \qquad \psi_2(t;0) = x_{2,0}(t), \qquad \psi_3(t;0) = x_{3,0}(t) \tag{4.9}$$

and

$$\psi_1(t;1) = x_1(t), \qquad \psi_2(t;1) = x_2(t), \qquad \psi_3(t;1) = x_3(t). \tag{4.10}$$

Therefore, as the embedding parameter q increases from 0 to 1, the functions $\psi_1(t;q)$, $\psi_2(t;q)$ and $\psi_3(t;q)$ vary from the initial values $x_{1,0}(t)$, $x_{2,0}(t)$ and $x_{3,0}(t)$ to the exact solution $x_1(t)$, $x_2(t)$ and $x_3(t)$, respectively. This is the basic idea of the homotopy and this kind of variation is called deformations in topology. Expanding $\psi_1(t;q)$, $\psi_2(t;q)$ and $\psi_3(t;q)$ in Taylor series with respect to q, we have the homotopy-Maclaurin series

$$\psi_i(t;q) = x_{i,0}(t) + \sum_{m=1}^{\infty} x_{i,m}(t) q^m, \quad i = 1,2,3 \tag{4.11}$$

where

$$x_{i,m}(t) = \frac{1}{m!} \left. \frac{\partial^m \psi_i(t;q)}{\partial q^m} \right|_{q=0} \tag{4.12}$$

where $\hbar$ is chosen in such a way that these series are convergent at $q = 1$. Thus, through Eqs. (4.9)-(4.12), we have the homotopy series solutions

$$x_i(t) = x_{i,0}(t) + \sum_{m=1}^{\infty} x_{i,m}(t), \quad i = 1,2,3. \tag{4.13}$$

Taking the m^{th}-order homotopy derivative of zeroth-order Eq. (4.8), and using the properties

$$D_m(\psi_i) = x_{i,m} D_m(q^k \psi_i) = D_{m-k}(\psi_i) = \begin{cases} x_{i,m-k}, & 0 \le k \le m, \\ 0, & k > m, \end{cases}$$

$$D_m(\psi_i^2) = \sum_{k=0}^{m} x_{i,m-k} x_{i,k}$$

and

$$D_m(\psi_i \phi_i) = \sum_{k=0}^{m} D_k(\psi_i) D_{m-k}(\phi_i) = \sum_{k=0}^{m} x_{i,k} y_{i,m-k}$$

where D_m means the m^{th}-order derivative with respect to q, we obtain the m^{th}-order deformation equations

$$L[x_{i,m}(t) - \chi_m x_{i,m-1}(t)] = \hbar H_i(t) \Re_{i,m}[[x_{1,m-1}(t), x_{2,m-1}(t), x_{3,m-1}(t)] \tag{4.14}$$

where

$$\chi_m := \begin{cases} 0, & m \le 1, \\ 1, & m > 1, \end{cases}$$

and the following initial conditions

$$x_{1,m}(0) = 0, \qquad x_{2,m}(0) = 0, \qquad x_{3,m}(0) = 0. \tag{4.15}$$

Defining the vector

$$\vec{x}_{m-1}(t) = \{x_{1,m-1}(t), x_{2,m-1}(t), x_{3,m-1}(t)\}$$

we drive

$$\begin{aligned}
\Re_{1,m}[\vec{x}_{m-1}(t)] =& \dot{x}_{1,m-1}(t) + a_{1,1}\left[\sum_{k=0}^{m-1} x_{1,k}(t)\dot{x}_{1,m-1-k}(t)\right] \\
&+ a_{1,2}\left[\sum_{k=0}^{m-1} x_{2,k}(t)\dot{x}_{1,m-1-k}(t)\right] \\
&+ a_{1,3}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{1,k-j}(t)x_{2,j}(t)\right)\dot{x}_{1,m-1-k}(t)\right] \\
&+ a_{1,4}\left[x_{1,m-1}(t)\right] + a_{1,5}[x_{2,m-1}(t)] \\
&+ a_{1,6}\left[\sum_{k=0}^{m-1} x_{1,k}(t)x_{1,m-1-k}(t)\right] \\
&+ a_{1,7}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{1,k-j}(t)x_{1,j}(t)\right)x_{2,m-1-k}(t)\right] \\
&+ a_{1,8}\left[\sum_{k=0}^{m-1} x_{1,k}(t)x_{2,m-1-k}(t)\right] + a_{1,9},
\end{aligned} \tag{4.16}$$

$$\begin{aligned}
\Re_{2,m}[\vec{x}_{m-1}(t)] =& \dot{x}_{2,m-1}(t) + a_{2,1}\left[\sum_{k=0}^{m-1} x_{2,k}(t)\dot{x}_{2,m-1-k}(t)\right] \\
&+ a_{2,2}\left[\sum_{k=0}^{m-1} x_{1,k}(t)\dot{x}_{2,m-1-k}(t)\right] \\
&+ a_{2,3}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{1,k-j}(t)x_{2,j}(t)\right)\dot{x}_{2,m-1-k}(t)\right] \\
&+ a_{2,4}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{2,k-j}(t)x_{2,j}(t)\right)\dot{x}_{2,m-1-k}(t)\right] \\
&+ a_{2,5}\left[\sum_{k=0}^{m-1}\left[\sum_{j=0}^{k} x_{1,k-j}(t)\dot{x}_{2,j}(t)\sum_{i=0}^{k} x_{1,k-i}(t)x_{1,i}(t)\right]\right] \\
&+ a_{2,6}x_{2,m-1}(t) + a_{2,7}\sum_{k=0}^{m-1} x_{2,k}(t)x_{2,m-1-k}(t) \\
&+ a_{2,8}\left[\sum_{k=0}^{m-1} x_{1,k}(t)x_{2,m-1-k}(t)\right]
\end{aligned} \tag{4.17}$$

$$
\begin{aligned}
&+ a_{2,9}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{1,k-j}(t)x_{2,j}(t)\right)x_{2,m-1-k}(t)\right],\\
&+ a_{2,10}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{2,k-j}(t)x_{2,j}(t)\right)x_{2,m-1-k}(t)\right]\\
&+ a_{2,11}\left[\sum_{k=0}^{m-1}\left[\sum_{j=0}^{k} x_{1,k-j}(t)x_{2,j}(t)\sum_{i=0}^{k} x_{2,k-i}(t)x_{2,i}(t)\right]\right],
\end{aligned}
$$

$$
\begin{aligned}
\Re_{3,m}[\vec{x}_{m-1}(t)] =&\dot{x}_{3,m-1}(t) + a_{3,1}\left[\sum_{k=0}^{m-1} x_{1,k}(t)\dot{x}_{3,m-1-k}(t)\right]\\
&+ a_{3,2}\left[\sum_{k=0}^{m-1} x_{2,k}(t)\dot{x}_{3,m-1-k}(t)\right]\\
&+ a_{3,3}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{1,k-j}(t)x_{2,j}(t)\right)\dot{x}_{3,m-1-k}(t)\right]\\
&+ a_{3,4}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{2,k-j}(t)x_{2,j}(t)\right)\dot{x}_{3,m-1-k}(t)\right]\\
&+ a_{3,5}\left[\sum_{k=0}^{m-1}\left[\sum_{j=0}^{k} x_{1,k-j}(t)\dot{x}_{3,j}(t)\sum_{i=0}^{k} x_{2,k-i}(t)x_{2,i}(t)\right]\right]\\
&+ a_{3,6}x_{2,m-1}(t) + a_{3,7}\left[\sum_{k=0}^{m-1} x_{2,k}(t)x_{2,m-1-k}(t)\right]\\
&+ a_{3,8}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{2,k-j}(t)x_{2,j}(t)\right)x_{2,m-1-k}(t)\right]\\
&+ a_{3,9}\left[\sum_{k=0}^{m-1} x_{1,k}(t)x_{2,m-1-k}(t)\right]\\
&+ a_{3,10}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{2,k-j}(t)x_{2,j}(t)\right)x_{1,m-1-k}(t)\right]\\
&+ a_{3,11}\left[\sum_{k=0}^{m-1}\left[\sum_{j=0}^{k} x_{1,k-j}(t)x_{2,j}(t)\sum_{i=0}^{k} x_{2,k-i}(t)x_{2,i}(t)\right]\right]\\
&+ a_{3,12}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{1,k-j}(t)x_{2,j}(t)\right)x_{3,m-1-k}(t)\right]\\
&+ a_{3,13}\left[\sum_{k=0}^{m-1} x_{2,k}(t)x_{3,m-1-k}(t)\right] + a_{3,14}[x_{3,m-1}(t)]
\end{aligned}
\tag{4.18}
$$

$$+ a_{3,15}\left[\sum_{k=0}^{m-1} x_{1,k}(t)x_{3,m-1-k}(t)\right]$$
$$- a_{3,16}\left[\sum_{k=0}^{m-1}\left(\sum_{j=0}^{k} x_{2,k-j}(t)x_{2,j}(t)\right)x_{3,m-1-k}(t)\right]$$
$$+ a_{3,17}\left[\sum_{k=0}^{m-1}\left[\sum_{j=0}^{k} x_{2,k-j}(t)x_{2,j}(t)\sum_{i=0}^{k} x_{1,k-i}(t)x_{3,i}(t)\right]\right]$$

and

$$\chi_m := \begin{cases} 0, & m \leq 1, \\ 1, & m > 1. \end{cases}$$

According to the notations and definitions provided above and by putting $H_i(t) = 1, i = 1, 2, 3$, the solution of the linear non-homogeneous Eq. (4.14) at initial conditions (4.15) for all $m \geq 1$, becomes

$$x_{i,m}(t) = \chi_m x_{i,m-1}(t) + \hbar \int_0^t \Re_{i,m}[\vec{x}_{m-1}]d\tau. \tag{4.19}$$

Finally, the m^{th}-order approximate solution of non-linear system (2.1), can be obtained as

$$x_i(t) = x_{i,0} + \sum_{n=1}^{m} x_{i,n}(t), \qquad i = 1, 2, 3. \tag{4.20}$$

The exact solutions are given by the limits

$$x_i(t) = \lim_{m\to+\infty} x_{i,m}(t), \qquad i = 1, 2, 3.$$

5. Numerical results

In this section, we present numerical results for the model (2.1) with the help of Mathematica software for homotopy analysis method. To illustrate the capability of the homotopy analysis method, we have chosen the following set of parameter values described in table 1 and in the appendix. By using Mathematica software, 8^{th}-order approximations for susceptibles $x(t)$, infected $y(t)$ and recovered $z(t)$ were calculated and are presented below

$$\begin{aligned}\psi_{x,8}(t;q) = \sum_{m=0}^{7} x_m(t) =& 20 + 0.4473\hbar t + 1.492737\hbar^2 t + 2.76752\hbar^3 t + 3.07859\hbar^4 t \\ &+ 2.05477\hbar^5 t + 0.76191\hbar^6 t \\ &+ 0.12108\hbar^7 t + 0.0943628\hbar^2 t^2 + 0.350478\hbar^3 t^2 + 0.58578\hbar^4 t^2 \\ &+ 0.52217\hbar^5 t^2 + 0.24243\hbar^6 t^2 \\ &+ 0.046307\hbar^7 t^2 + 0.0043493\hbar^3 t^3 + 0.014575\hbar^4 t^3 \\ &+ 0.019536\hbar^5 t^3 + 0.012123\hbar^6 t^3 \\ &+ 0.0029018\hbar^7 t^3 + 0.00006297\hbar^4 t^4 + 0.0001701\hbar^5 t^4\end{aligned}$$

$$
\begin{aligned}
&+ 0.0001595\hbar^6t^4 + 0.00005129\hbar^7t^4\\
&+ 1.11301\times10^{-7}\hbar^5t^5 + 1.11301\times10^{-7}\hbar^5t^5\\
&+ 1.1975\times10^{-7}\hbar^7t^5 - 3.41717\times10^{-9}\hbar^6t^6\\
&- 3.19538\times10^{-9}\hbar^7t^6\\
&- 1.5972\times10^{-11}\hbar^7t^7 + 2.28516\times10^{-7}\hbar^7t^6, \qquad (5.1)
\end{aligned}
$$

$$
\begin{aligned}
\psi_{y,8}(t;q) = \sum_{m=0}^{7} y_m(t) =& 15 + 0.17831\hbar t + 0.59862\hbar^2t + 1.11648\hbar^3t + 1.2494\hbar^4t\\
&+ 0.83889\hbar^5t + 0.31293\hbar^6t\\
&+ 0.050026\hbar^7t - 0.035782\hbar^2t^2 - 0.13292\hbar^3t^2\\
&- 0.22218\hbar^4t^2 - 0.19807\hbar^5t^2 - 0.091968\hbar^6t^2\\
&- 0.017569\hbar^7t^2 - 0.003048\hbar^3t^3 - 0.010215\hbar^4t^3\\
&- 0.013691\hbar^5t^3 - 0.008495\hbar^6t^3\\
&- 0.002033\hbar^7t^3 - 0.000063743\hbar^4t^4 - 0.00017165\hbar^5t^4\\
&- 0.00016048\hbar^6t^4\\
&- 0.000051442\hbar^7t^4 - 2.98764\times10^{-7}\hbar^5t^5\\
&- 5.71816\times10^{-7}\hbar^6t^5 - 2.81208\times10^{-7}\hbar^7t^5\\
&- 2.49227\times10^{-9}\hbar^6t^6 - 2.31991\times10^{-9}\hbar^7t^6\\
&- 1.6326\times10^{-11}\hbar^7t^7, \qquad (5.2)
\end{aligned}
$$

$$
\begin{aligned}
\psi_{z,8}(t;q) = \sum_{m=0}^{7} z_m(t) =& 10 - 6.24762\hbar t - 20.9744\hbar^2t - 39.1193\hbar^3t - 43.7768\hbar^4t\\
&- 29.3933\hbar^5t - 10.9643\hbar^6t\\
&- 1.75282\hbar^7t - 0.54129\hbar^2t^2 - 2.02408\hbar^3t^2 - 3.40591\hbar^4t^2\\
&- 3.05654\hbar^5t^2 - 1.42863\hbar^6t^2\\
&- 0.27472\hbar^7t^2 - 0.0148338\hbar^3t^3 - 0.0502404\hbar^4t^3\\
&- 0.068057\hbar^5t^3 - 0.0426775\hbar^6t^3\\
&- 0.0103217\hbar^7t^3 - 0.00013785\hbar^4t^4 - 0.000381295\hbar^5t^4\\
&- 0.00036590\hbar^6t^4 - 0.000012029\hbar^7t^4\\
&- 2.81857\times10^{-7}\hbar^5t^5 - 6.18926\times10^{-7}\hbar^6t^5\\
&- 3.41791\times10^{-7}\hbar^7t^5 - 5.61582\times10^{-12}\hbar^6t^6\\
&- 2.07272\times10^{-10}\hbar^7t^6\\
&- 6.68787\times10^{-12}\hbar^7t^7, \qquad (5.3)
\end{aligned}
$$

6. Discussion

The homotopy terms depend on both the physical variable t and the convergence control parameter $\hbar$. The artificial parameter $\hbar$ can be freely chosen to adjust and

control the interval of convergence, and even more, to increase the convergence at a reasonable rate, fortunately at the quickest rate. This concept plays a key role in the HAM and is generally used to gain sufficiently accurate approximations with the smallest number of homotopy terms in the homotopy series (4.20). In fact, the use of such an auxiliary parameter clearly distinguishes the HAM from other perturbation-like analytical techniques. According to convergence theorem [39,53], it is to be noted that the homotopy series solution contain the auxiliary parameter which provides a simple way to adjust and control the convergence of the series (5.1)-(5.3). In fact, it is very important to ensure that the series Eqs. (4.20)-(4.22) are convergent. To this end, the $\hbar$-curves of $x(t)$, $y(t)$ and $z(t)$ under 8^{th}-order approximation of the homotopy analysis method are plotted in Fig. 2.

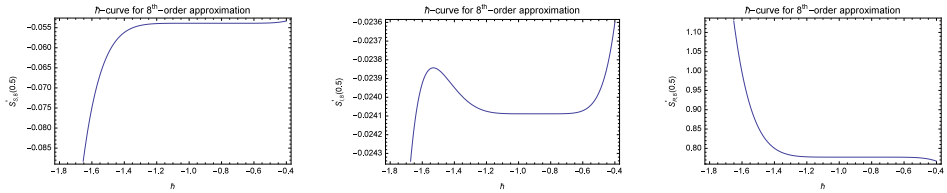

Figure 2. Samples of $\hbar$-curves for $x(t)$, $y(t)$ and $z(t)$ under 8^{th}-order approximation for $t = 0.5$.

According to these $\hbar$-curves, it is easy to gain the valid region the interval of convergence and optimum value for parameter $\hbar$ which corresponds to the line segment nearly parallel to the horizontal axis. For better presentation, these valid regions have been listed in table 2. We exhibit the interval of convergence of $\hbar$ and the respective optimum value $\hbar^*$ corresponding to the dynamical regime presented in Fig. 2. It is to be noted that these valid regions ensure the convergence of the obtained series.

Table 2. The admissible values of $\hbar$ derived from Fig. 2.

$\hbar_1^*$	$\hbar_2^*$	$\hbar_3^*$
$[-1.3, -0.5]$	$[-1.15, -0.65]$	$[-1.25, -0.55]$

When $\hbar = -1$ the solution obtained by the HAM is the same as the series solution obtained by using homotopy perturbation method (HPM) [28,39]. A procedure to check the convergence of a homotopy-series solution is to substitute this series into the original governing equations and initial conditions, and then to evaluate the corresponding squared residual errors-the more quickly the residual error decays to zero, the faster the homotopy-series converges. In this context, an error analysis is performed in the following lines [13]. We substitute Eqs. (5.1)-(5.3) into model (2.1) and obtain the residual functions $ER_i(x, y, z; \hbar_i), i = 1, 2, 3$ as follows

$$\begin{aligned} ER_{1,m}(x, y, z, \hbar_1) =& \frac{d\psi_x(t;\hbar_1)}{dt} + \frac{d\psi_x(t;\hbar_1)}{dt}[a_{1,1}\psi_x(t;\hbar_1) + a_{1,2}\psi_y(t;\hbar_1) \\ &+ a_{1,3}\psi_x(t;\hbar_1)\psi_y(t;\hbar_1)] + \psi_x(t;\hbar_1)[a_{1,4} + a_{1,6}\psi_x(t;\hbar_1)] \\ &+ \psi_y(t;\hbar_1)[a_{1,5} + a_{1,8}\psi_x(t;\hbar_1) + a_{1,7}\psi_x^2(t;\hbar_1)] + a_{1,9}, \quad (6.1) \end{aligned}$$

$$\begin{aligned}ER_{2,m}(x,y,z,\hbar_2) =& \frac{d\psi_y(t;\hbar_2)}{dt} + \frac{d\psi_y(t;\hbar_2)}{dt}[a_{2,1}\psi_y(t;\hbar_2) + a_{2,2}\psi_x(t;\hbar_2)\\ &+ a_{2,3}\psi_x(t;\hbar_2)\psi_y(t;\hbar_2) + a_{2,4}\psi_y^2(t;\hbar_2)\\ &+ a_{2,5}\psi_x(t;\hbar_2)\psi_y^2(t;\hbar_2)] + \psi_y(t;\hbar_2)[a_{2,6}\\ &+ a_{2,7}\psi_y(t;\hbar_2) + a_{2,10}\psi_y^2(t;\hbar_2)]\\ &+ \psi_x(t;\hbar_2)\psi_y(t;\hbar_2)[a_{2,8} + a_{2,9}\psi_y(t;\hbar_2) + a_{2,11}\psi_y^2(t;\hbar_2)] = 0,\end{aligned} \tag{6.2}$$

$$\begin{aligned}ER_{3,m}(x,y,z,\hbar_3) =& \frac{d\psi_z(t;\hbar_3)}{dt} + \frac{d\psi_z(t;\hbar_3)}{dt}[a_{3,1}\psi_x(t;\hbar_3)\\ &+ a_{3,2}\psi_y(t;\hbar_3) + a_{3,3}\psi_x(t;\hbar_3)\psi_y(t;\hbar_3) + a_{3,4}\psi_y^2(t;\hbar_3)\\ &+ a_{3,5}\psi_x(t;\hbar_3)\psi_y^2(t;\hbar_3)] + \psi_y(t;\hbar_3)[a_{3,6} + a_{3,7}\psi_y(t;\hbar_3)\\ &+ a_{3,8}\psi_y^2(t;\hbar_3)]\\ &+ \psi_x(t;\hbar_3)\psi_y(t;\hbar_3)[a_{3,9} + a_{3,10}\psi_y(t;\hbar_3) + a_{3,11}\psi_y^2(t;\hbar_3)]\\ &+ a_{3,12}\psi_x(t;\hbar_3)\psi_y(t;\hbar_3)\psi_z(t;\hbar_3) + \psi_z(t;\hbar_3)[a_{3,14}\\ &+ a_{3,15}\psi_x(t;\hbar_3)\\ &+ a_{3,13}\psi_y(t;\hbar_3)] - \psi_y^2(t;\hbar_3)\psi_z(t;\hbar_3)[a_{3,16} + a_{3,17}\psi_x(t;\hbar_3)] = 0.\end{aligned} \tag{6.3}$$

Yabushita et al. [51] in 2007 suggested an optimization method for convergence control parameters. Their work is based on the squared residual error. Inspired by their approach, and following the studies carried out in [20,27], we define the square residual error for the m^{th}-order approximation to be

$$\Delta x_m(\hbar_1) = \int_0^1 (ER_{1,m}(x,y,z,\hbar_1))^2 dt \tag{6.4}$$

$$\Delta y_m(\hbar_2) = \int_0^1 (ER_{2,m}(x,y,z,\hbar_2))^2 dt, \tag{6.5}$$

$$\Delta z_m(\hbar_3) = \int_0^1 (ER_{3,m}(x,y,z,\hbar_3))^2 dt. \tag{6.6}$$

Values of $\hbar_1$, $\hbar_2$ and $\hbar_3$ can be obtained for which $\Delta x_m(\hbar_1)$, $\Delta y_m(\hbar_2)$ and $\Delta z_m(\hbar_3)$ are minimum. The optimal values $\hbar_1^*$, $\hbar_2^*$ and $\hbar_3^*$ are determined by solving the system of equation as

$$\frac{d\Delta x_m(\hbar_1^*)}{dt} = 0, \qquad \frac{d\Delta y_m(\hbar_2^*)}{dt} = 0, \qquad \frac{d\Delta z_m(\hbar_3^*)}{dt} = 0,$$

respectively. The optimal values for all of these considered cases are $\hbar_1^*$, $\hbar_2^*$ and $\hbar_3^*$. The curves of square residual errors for $\Delta x_m(\hbar_1)$, $\Delta y_m(\hbar_2)$ and $\Delta z_m(\hbar_3)$ regarding 8^{th}-order approximation are shown in Fig. 3. For the central information regarding the order of approximation, in table 3, the minimum values of $\Delta x_m(\hbar_1)$, $\Delta y_m(\hbar_2)$ and $\Delta z_m(\hbar_3)$ have been given with the optimal values of $\hbar_1^*$, $\hbar_2^*$ and $\hbar_3^*$ for 8^{th}-order approximation.

Table 3. The minimum values of $\Delta x_m(\hbar_1)$, $\Delta y_m(\hbar_2)$ and $\Delta z_m(\hbar_3)$.

$\hbar_1^*$	$\Delta x_7(\hbar_1^*)$	$\hbar_2^*$	$\Delta y_7(\hbar_2^*)$	$\hbar_3^*$	$\Delta y_7(\hbar_2^*)$
-0.89681	9.26098×10^{-16}	-0.89724	6.14983×10^{-16}	-0.88833	8.49430×10^{-13}

In table 4, the absolute errors $ER_{1,m}(x,y,z,\hbar_1^*)$, $ER_{2,m}(x,y,z,\hbar_2^*)$ and $ER_{3,m}(x,y,z,\hbar_3^*)$ have been calculated for various $t \in (0,1)$ under 8^{th}-order approximation of homotopy analysis method. From the table, it can be clearly seen that the HAM provides us the accurate approximate solution for the nonlinear modified SIR epidemic model (2.1).

Table 4. The absolute errors ER_1, ER_2 and ER_3 for various $t \in (0,1)$.

t	$ER_{1,7}(x,y,z;\hbar_1^*)$	$ER_{2,7}(x,y,z;\hbar_2^*)$	$ER_{3,7}(x,y,z;\hbar_3^*)$
0.0	-4.44089×10^{-16}	0.0	-3.55271×10^{-15}
0.1	-4.72888×10^{-16}	9.76336×10^{-17}	-2.36161×10^{-15}
0.2	-1.49074×10^{-15}	2.73812×10^{-15}	8.27016×10^{-15}
0.3	-1.00877×10^{-14}	1.86703×10^{-14}	7.37585×10^{-16}
0.4	-4.59364×10^{-14}	7.45827×10^{-14}	-2.84079×10^{-14}
0.5	-1.49684×10^{-13}	2.20912×10^{-13}	-3.43425×10^{-14}
0.6	-3.90665×10^{-13}	5.39232×10^{-13}	1.04278×10^{-13}
0.7	-8.73963×10^{-13}	1.14863×10^{-12}	6.18178×10^{-13}
0.8	-1.74634×10^{-12}	2.21051×10^{-12}	1.88543×10^{-12}
0.9	-3.20055×10^{-12}	3.9318×10^{-12}	4.47497×10^{-12}
1	-5.47761×10^{-12}	6.56173×10^{-12}	9.19622×10^{-12}

In Fig. 3, the residual error for $t = 0.5$ and versus auxiliary parameter $\hbar$ are demonstrated. We have also shown the square residual error based of HAM in Fig. 4. Fi-

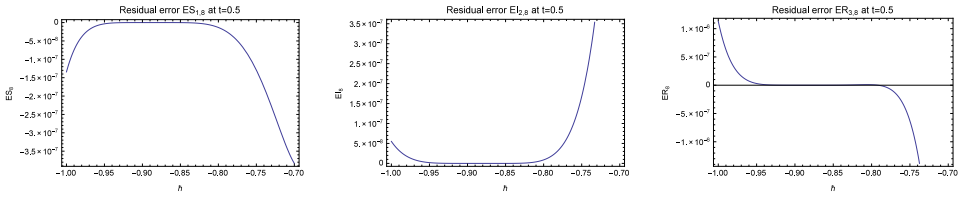

Figure 3. The residual error function for $x(t)$, $y(t)$ and $z(t)$ at $t = 0.5$ under 8^{th}-order approximation versus auxiliary parameter $\hbar$.

nally, the absolute errors $ER_{1,m}(x,y,z,\hbar_1^*)$, $ER_{2,m}(x,y,z,\hbar_2^*)$ and $ER_{3,m}(x,y,z,\hbar_3^*)$ have been plotted in Fig.5 for $t \in (0,1)$ under 8^{th}-order approximation. By considering these figures, it is to be noted that the solution obtained using HAM gives an analytical solution with high order of accuracy.

7. Conclusion

In this paper, the SIR epidemic model with Crowley-Martin type incidence rate and Holling type-II treatment rate was illustrated and we examined the local as well as the global dynamics of the system by analysing the basic reproduction

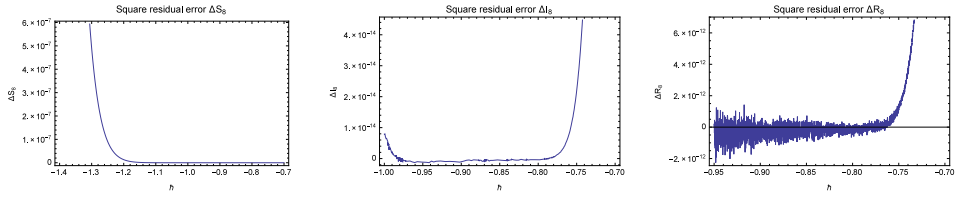

Figure 4. The square residual error function for $x(t)$, $y(t)$ and $z(t)$ at $t = 0.5$ under 8^{th}-order approximation versus auxiliary parameter $\hbar$.

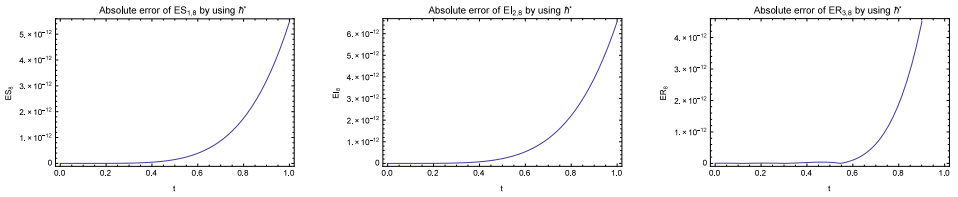

Figure 5. The absolute error function for $x(t)$, $y(t)$ and $z(t)$ at $t = 0.5$ under 8^{th}-order approximation.

number $\Re_0$. We found that the system (2.1) exhibits two equilibria namely disease-free equilibrium E^0 and endemic equilibrium E^*. It is observed, that if $\Re_0 > 1$, then the infection persists and if $\Re_0 < 1$ the infection is cleared. The stability analysis i.e., local and global stability of disease-free equilibrium E^0 and endemic equilibrium E^* were studied and found that persistence or eradication of infection is independent of initial status of the subpopulation. From Theorem 3.6, we found the system is uniformly persistent under the given condition in the said theorem. The disease-free equilibrium E^0 has been shown to be stable for $\Re_0 < 1$, i.e., disease dies out for $\Re_0 < 1$ and for $\Re_0 > 1$, it becomes unstable and the endemic equilibrium exists. We also discussed the stability of disease-free equilibrium at $\Re_0 = 1$ using center manifold theorem. We observed that at $\Re_0 = 1$, the disease-free equilibrium changes its stability from stable to unstable and undergoes transcritical bifurcation. The endemic equilibrium is locally asymptotically stable for $\Re_0 > 1$ as shown in the Theorem 3.7. Also, the global asymptotic stability of endemic equilibrium is obtained in the Theorem 3.8 for $\Re_0 > 1$. We have found that system (2.1) has periodic solution if inequalities as stated in Theorem 3.9 hold true and there is no periodic solution if $\beta c > \beta\delta$ holds true. The existence of periodic solution shows that the infection may reoccur in the future. Also, HAM was applied to obtain an approximate analytical solution of the presented model. It is important to note that in this method we have some auxiliary parameters and functions. One of these parameters is the convergence control parameter which can be applied to adjust and control the convergence region of obtained solutions. Thus, by plotting several $\hbar$-curves and finding the regions of convergence, we showed the advantages and abilities of the method. The residual and absolute errors were applied to show the efficiency and accuracy of the method. The results obtained shown that the HAM is an accurate and effective technique for obtaining the approximate solution of the modified SIR epidemic model.

Here in the current paper, we have investigated the stability analysis as well

as obtained the corresponding approximate solution of the proposed SIR epidemic model. The stability analysis is performed in order to know the disease status i.e., its persistence or eradication corresponding to Crowley-Martin type incidence rate and the approximate solution is obtained for the better treatment strategies according to the Holling type -II treatment rate because more appropriate solutions leads towards proper treatment and control measures for the disease transmission.

The results presented in this article are likely to inspire applications of the HAM analytical procedure for solving highly nonlinear problems in theoretical biology. This study provides another illustration of how an integrated approach, involving numerical evidences and theoretical reasoning within the theory of dynamical systems, can directly enhance our understanding of biologically motivated models.

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Competing interests. The authors declare that there are no competing interests.

Apendix: Parameter Values.

$a_{1,1} = b = 0.004,\ \ a_{1,2} = c = 0.002,\ \ a_{1,3} = bc = 0.000008,\ \ a_{1,4} = \alpha_0 - b\Lambda = 0.042,$
$a_{1,5} = \alpha_0 c - \Lambda c = -0.0039,\ a_{1,6} = \alpha_0 b = 0.0002,\ \ a_{1,7} = \alpha_0 bc = 0.0000004,$
$a_{1,8} = a = 0.004,\ \ a_{1,9} = -\Lambda = -2,$

$a_{2,1} = c + \delta = 0.0024,\ \ a_{2,2} = b = 0.004,\ \ a_{2,3} = bc + b\delta = 0.0000096,$
$a_{2,4} = \delta c = 0.0000008,$
$a_{2,5} = bc\delta = 3.2 \times 10^{-9},\ \ a_{2,6} = \lambda + \lambda\delta + \beta = 0.073021,\ \ a_{2,7} = \lambda c + \beta c = 0.000146$
$a_{2,8} = \lambda b - a + \beta b = -0.003708,\ \ a_{2,9} = \lambda bc + a\delta + \lambda b\delta + \beta bc = 2.1248 \times 10^{-6},$
$a_{2,10} = \lambda\delta c = 4.24 \times 10^{-8}$
$a_{2,11} = \lambda\delta bc = 1.696 \times 10^{-10},$

$a_{3,1} = b = 0.004,\ \ a_{3,2} = c + \delta = 0.0024,\ \ a_{3,3} = bc + b\delta = 0.0000096,$
$a_{3,4} = \delta c = 0.0000008,\ \ a_{3,5} = bc\delta = 3.2 \times 10^{-9},\ \ a_{3,6} = -\alpha_2 - \beta = -0.022,$
$a_{3,7} = -\alpha_2 - \alpha_2\delta - \beta c = -0.0020408,$
$a_{3,8} = -\alpha_2\delta c = 1.6 \times 10^{-9},\ \ a_{3,9} = -\alpha_2 - \beta b = -0.00208,$
$a_{3,10} = -\alpha_2 bc - \alpha_2 b\delta - \beta bc = 1.792 \times 10^{-9},$
$a_{3,11} = \alpha_2 b\delta c = 6.4 \times 10^{-12},\ \ a_{3,12} = -\alpha_0 bc - \alpha_0 b\delta = -4.8 \times 10^{-7},$
$a_{3,13} = -\alpha_0 c - \alpha_0\delta = -0.00012,$
$a_{3,14} = \alpha_0 = 0.05,\ \ a_{3,15} = \alpha_0 b = 0.0002,\ \ a_{3,16} = \alpha_0 c\delta = 4 \times 10^{-8},$
$a_{3,17} = \alpha_0 bc\delta = 1.6 \times 10^{-10}.$

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