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Improving the hepatitis viral transmission model's dynamics by vaccination and contrasting it with the fractional-order model

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ABSTRACT

We investigate mathematical models of the Hepatitis B and C viruses in the study, considering vaccination effects into account. By utilising fractional and ordinary differential equations, we prove the existence of equilibrium and the well-posedness of the solution. We prove worldwide stability with respect to the fundamental reproduction number. Our numerical techniques highlight the biological relevance and highlight the effect of fractional derivatives on temporal behaviour. We illustrate the relationships among susceptible, immunised, and infected populations in our epidemiological model. Using comprehensive numerical simulations, we analyse the effects of fractional derivatives and highlight solution behaviours. Subsequent investigations will examine the impact of regional heterogeneity, providing significant perspectives for epidemiological research.

1. Introduction

With symptoms that are frequently similar across different strains, hepatitis, a virally-induced liver inflammation, poses a serious danger to world health. The hepatitis B (HBV) and C (HCV) viruses in particular do a great deal of harm; persistent infections can result in cancer and liver cirrhosis, and they are the cause of around a million fatalities each year. Not all treatments work, even after a great deal of research. In order to comprehend and stop the transmission of hepatitis, mathematical modelling has become an essential tool. Different features of the dynamics of infection and treatment approaches have been the focus of several models that have been put forth. This reflects the broad interest in addressing this intricate health issue. Studies on HBV using logistic hepatocyte growth, delay models, optimum control formulations, and fractional calculus-based techniques are among them.

Hepatitis C virus (HCV) was identified in 1989 as the major agent of post-transfusion hepatitis previously referred to as non-A, non-B hepatitis, see Ref. 1. During the replication of the viral genome, errors are frequent and lead to the circulation of a large number of viral molecular species in the human population. The viral variants identified to date are grouped into 7 genotypes which present variable susceptibilities to treatments. Since the identification of these viruses, a huge focus by researchers on obtaining proper treatment. Moreover, numerous mathematical models are considered to study the spread of the hepatitis virus in the host body. In Ref. 2, the authors considered a hepatitis B infection model with logistic hepatocyte growth, where the purpose is to show the asymptotic behaviour of the solution, where the logistic growth will lead to the existence of Hopf bifurcation. Steffen and Hews in Ref. 3, proposed and analysed a delay model of hepatitis B virus infection with logistic hepatocyte growth. In Ref. 4, the authors considered a hepatitis a infection model with co-infection, where the optimal control strategy is considered. Farman et al.[5] studied the effect of the treatment on the dynamical hepatitis B model. Different other approximations are used for predicting the spread of the hepatitis disease in population for example, Din et al. constructed system of equation for Hepatitis B disease in sense of Atanganaa–Baleanu Caputo (ABC) fractional-order derivative[6]. Friedman and Siewe[7], formulated a mathematical model of hepatitis B virus and liver fibrosis. In Ref. 8

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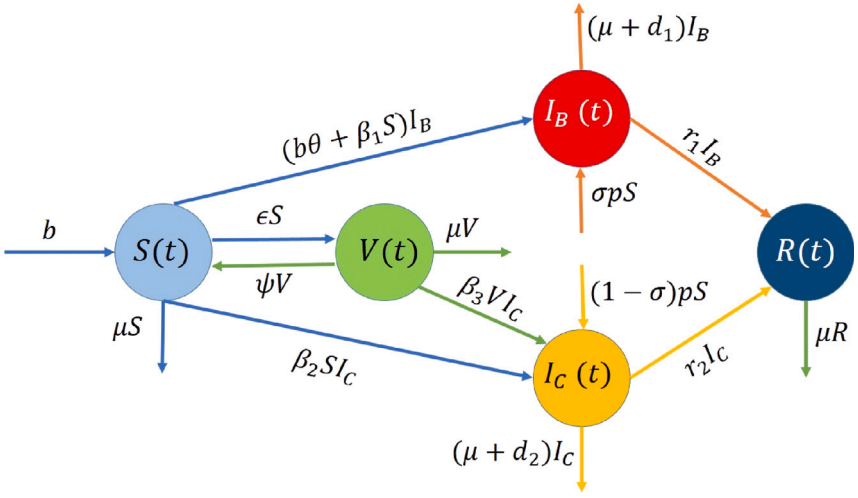

Fig. 1. Diagram of viral hepatitis model.

Liu et al. studied and simulation a fractional model for the transmission dynamics of hepatitis B. Khan et al.[9,10] analysed and discussed the time dynamics of hepatitis B under the effect of various infectious periods. The authors in Ref. 11, suggested a mathematical fractional-order hepatitis C virus HCV. The mathematical model of hepatitis B virus with optimal control formulated by Dominic et al. in Ref. 12. Also, Nadia et al.[13] considering a new mathematical model for hepatitis B virus with asymptomatic carriers. Beside this the fractional calculus played an important role for descriptions and control of such types of diseases see Refs. 14–23. We methodically organise our concern, beginning with the definition and proof of the well-posedness of the mathematical model. After identifying important variables like equilibria and the basic reproduction number, we perform studies of both local and global stability. Furthermore, we present a fractional-order model that offers a numerical framework for simulating nonlinear temporal dynamics.

2. Formulation of the viral hepatitis model

In this part, we propose the epidemiological mathematical model of hepatitis viruses " B and C " type infection disease. To write this model by nonlinear differential equations. We suppose that $N(t)$ represents the total human population that divided into five classes are: the susceptible individuals denoted by $S(t)$ at time t; individuals who have been given the hepatitis B vaccine denoted by $V(t)$ at time t; individuals infected with viral hepatitis B denoted by $I_B(t)$at time t; individuals infected with viral hepatitis C denoted by $I_C(t)$ at time t and final class is removal individuals denoted by $R(t)$ at time t respectively. Our assumptions on the transmission of viral hepatitis disease in this work are illustrated in Fig. 1 and corresponding model nonlinear differential equations are presented below:

$$\begin{aligned}
\frac{dS}{dt} &= b(1-\theta I_B) - (\mu + \epsilon + p + \beta_1 I_B + \beta_2 I_C)S + \psi V,\\
\frac{dV}{dt} &= \epsilon S - (\mu + \psi + \beta_3 I_C)V,\\
\frac{dI_B}{dt} &= (b\theta + \beta_1 S - \mu - d_1 - r_1)I_B + \sigma pS,\\
\frac{dI_C}{dt} &= (\beta_2 S + \beta_3 V - \mu - d_2 - r_2)I_C + (1-\sigma)pS,\\
\frac{dR}{dt} &= r_1 I_B + r_2 I_C - \mu R,
\end{aligned} \tag{1}$$

with the non negative initial population size for (1) verifies

$$S(0) > 0, V(0) > 0, I_B(0) \geq 0, I_C(0) \geq 0, R(0) \geq 0. \tag{2}$$

The description of the all parameters of model (1) is given as; the b is natural birth. The death rate represented by μ. The $\beta_i, i = 1,2,3$ are the transmission rates between susceptible individuals with infected individuals as well between vaccinated individuals with infected individuals by viral hepatitis B respectively. In addition to transmission by direct contact, viral hepatitis B is also transmitted vertically (that is, there are newborns infected) that represented by $(0 \leq \theta \leq 1)$. The external sources of disease in the environment represented by $p \geq 0$ with fraction $(0 \leq \sigma \leq 1)$. The $\epsilon > 0$ is vaccine rate and $(0 \leq \psi \leq 1)$ is vaccination failure rate. The $d_i, i = 1,2$ are death rates due to disease. The $r_i, i = 1,2$ are recovery rates from I_B and I_C, respectively.

3. Model analysis

In this part, we establish the existence, uniqueness, boundedness and non-negativity of solutions of model (1) in the following theorems.

Theorem 3.1. *For any initial population size satisfying (2), there is a unique solution of (1). Moreover, the solution is always non-negative for $t \geq 0$ and remains in $\Re^5_+$.*

Proof. The expressions on the right of all the equations of our model is continuous and have continuously partial derivative in $\Gamma = (t, S(t), V(t), I_B(t), I_C(t), R(t))$, then they are Lipschitzian. Then, we guarantees the existence of a global solution for (1) in $\Re^5_+$.

3.1. Boundedness

Theorem 3.2. *Let $X(t)$ be the unique solution of (1) for any $t \geq 0$, then the solution $X(t)$ is bounded above, such that $X(t) \in \Omega$ where Ω is the feasible region.*

Proof. By adding the left and right equations of model (1), we obtain that

$$\frac{dN}{dt} = b - \mu N - d_1 I_B - d_2 I_C. \tag{3}$$

Thus,

$$\frac{dN}{dt} \leq b - \mu N.$$

Applying integration on the preceding inequality, we obtain

$$N(t) \leq N(0)e^{-\mu t} + \frac{b}{\mu}(1 - e^{-\mu t}),$$

Hence,

$$N(t) \leq \frac{b}{\mu}. \tag{4}$$

Consequently, Ω is positively invariant. However, if $N(0) > b/\mu$, then either the solution enters Ω in finite time, or $N(t)$ approach to b/μ as $t \to \infty$. Hence, Ω is attracting (i.e., all solutions of model (1) in $\Re^5_+$ eventually approach, enter, or stay in Ω).

3.2. Positivity

Theorem 3.3. *Let the initial points which given in Eq. (2) belonging to* $\mathfrak{R}^5_+$. *Then, the corresponding solution set* $(S(t), V(t), I_B(t), I_C(t), R(t))$ *of model (1) is non-negative for all* $t \geq 0$.

Proof. From model (1), we have

$$\begin{aligned}
&\frac{dS}{dt}|_{S=0} = b(1-\theta I_B)+\psi V > 0, \quad \text{for all} \quad I_B, V \geq 0,\\
&\frac{dV}{dt}|_{V=0} = \epsilon S > 0, \quad \text{for all} \quad S > 0,\\
&\frac{dI_B}{dt}|_{I_B=0} = \sigma p S \geq 0, \quad \text{for all} \quad S > 0,\\
&\frac{dI_C}{dt}|_{I_C=0} = (1-\sigma)pS \geq 0, \quad \text{for all} \quad S > 0,\\
&\frac{dR}{dt}|_{R=0} = r_1 I_B + r_2 I_C \geq 0, \quad \text{for all} \quad I_B, I_C \geq 0.
\end{aligned}$$

Therefore, the solution of the model is positive.

It easy see that, variable R is not show in the first four equations of model (1). So, we can reduction it is to system in below

$$\begin{aligned}
\frac{dS}{dt} &= b(1-\theta I_B) - (\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C)S + \psi V,\\
\frac{dV}{dt} &= \epsilon S - (\mu+\psi+\beta_3 I_C)V,\\
\frac{dI_B}{dt} &= (b\theta+\beta_1 S-\mu-d_1-r_1)I_B + \sigma p S,\\
\frac{dI_C}{dt} &= (\beta_2 S+\beta_3 V-\mu-d_2-r_2)I_C + (1-\sigma)pS.
\end{aligned} \tag{5}$$

4. Model equilibria and effective basic reproduction number of (5)

Clearly, if $I_B = I_C = 0$ with $p = 0$ there is uninfected viral hepatitis equilibrium point $(UVHE)$ of model (5) and indicate it via $e_0 = (S_0, V_0, 0, 0)$, where

$$\begin{cases} S_0 = \frac{b(\mu+\psi)}{\mu(\mu+\epsilon+\psi)}, \\ V_0 = \frac{\epsilon b}{\mu(\mu+\epsilon+\psi)}. \end{cases} \tag{6}$$

Therefore, the basic reproduction number of our model (5), which is denoted by $\mathcal{R}_0$ is obtained by using the next generation matrix (i.e., The maximum eigenvalue of the reproduction number, those computed of each disease). Given by

$$\mathcal{R}_0 = \mathcal{R}_{0B} + \mathcal{R}_{0C}, \tag{7}$$

such that

$$\mathcal{R}_{0B} = \frac{b\theta\mu(\mu+\epsilon)(\mu+\epsilon+\psi) + b\beta_1[\mu(\mu+\epsilon+\psi)+\epsilon\psi]}{\mu(\mu+\epsilon)(\mu+\epsilon+\psi)(\mu+d_1+r_1)},$$

$$\mathcal{R}_{0C} = \frac{b\beta_2[\mu(\mu+\epsilon+\psi)+\epsilon\psi] + \beta_3\epsilon b(\mu+\epsilon)}{\mu(\mu+\epsilon)(\mu+\epsilon+\psi)(\mu+d_2+r_2)},$$

for viral B hepatitis disease and for viral C hepatitis disease respectively.

Clearly, by letting $I_B = 0$ we deduce that (5) has a hepatitis B virus-free equilibrium point $(HBVFE)$, with $\sigma = 0$, which is denoted by $e_1 = (S_1, V_1, 0, I_{C1})$, where.

$$\begin{cases} S_1 = \frac{b(\mu+\psi+\beta_3 I_{C1})}{(\mu+\beta_3 I_{C1})(\mu+\epsilon+p+\beta_2 I_{C1})+\psi(\mu+p+\beta_2 I_{C1})}, \\ V_1 = \frac{\epsilon b}{(\mu+\beta_3 I_{C1})(\mu+\epsilon+p+\beta_2 I_{C1})+\psi(\mu+p+\beta_2 I_{C1})}. \end{cases} \tag{8}$$

Considering that I_{C1} is positive then we obtain the following equation

$$A_1 I_{C1}^3 + A_2 I_{C1}^2 + A_3 I_{C1} + A_4 = 0, \tag{9}$$

here

$$\begin{aligned}
A_1 &= -\beta_2\beta_3(\mu+d_2+r_2) < 0,\\
A_2 &= b\beta_2\beta_3 - (\mu+d_2+r_2)(\mu\beta_2+\beta_3(\mu+\epsilon+p)+\psi\beta_2),\\
A_3 &= b\beta_3(\epsilon+p) - [\mu(\mu+\epsilon+p)+\psi(\epsilon+p)(\mu+d_2+r_2)],\\
A_4 &= bp\beta_3 > 0.
\end{aligned}$$

Hence, (9) has a unique positive root that exists if and only if $A_2 < 0$ or $A_3 > 0$.

Next, by letting $\sigma = 1$ we deduce that (5) has a hepatitis C virus-free equilibrium point $(HCVFE)$, denoted $e_2 = (S_2, V_2, I_{B2}, 0)$, with

$$\begin{cases} S_2 = \frac{b(1-\theta I_{B2})(\mu+\psi)}{\mu(\mu+\epsilon+p+\beta_1 I_{B2})+\psi(\mu+p+\beta_1 I_{B2})}, \\ V_2 = \frac{b(1-\theta I_{B2})}{\mu(\mu+\epsilon+p+\beta_1 I_{B2})+\psi(\mu+p+\beta_1 I_{B2})}. \end{cases} \tag{10}$$

Clearly, S_2 and V_2 are positive if

$$\theta I_{B2} < 1. \tag{11}$$

Next, we calculated I_{B2} which is the positive root of the equation

$$B_1 I_{B2}^2 + B_2 I_{B2} + B_3 = 0, \tag{12}$$

where

$$\begin{aligned}
B_1 &= -\beta_1(\mu+\psi)(\mu+d_1+r_1) < 0,\\
B_2 &= b[\mu\theta(\mu+\epsilon+p)+\psi\theta(\mu+p)+\beta_1(\mu+\psi)] - bp\theta(\mu+\psi)\\
&\quad -(\mu+d_1+r_1)[\psi(\mu+p)+\mu(\mu+\epsilon+p)],\\
B_3 &= bp(\mu+\psi) > 0.
\end{aligned}$$

Then I_{B2} the root of Eq. (12) if and only if $B_2 < 0$ or $B_2 > 0$.

At last, model (5) has the endemic equilibrium (EE) and denoted by $e_3 = (S_3, V_3, I_{B3}, I_{C3})$, with.

$$\begin{cases} S_3 = \frac{b(1-\theta I_{B3})(\mu+\psi+\beta_3 I_{C3})}{(\mu+\psi+p+\beta_3 I_{C3})(\mu+p+\beta_1 I_{B3}+\beta_2 I_{C3})+\epsilon(\mu+\beta_3 I_{C3})}, \\ V_3 = \frac{\epsilon b(1-\theta I_{B3})}{(\mu+\psi+p+\beta_3 I_{C3})(\mu+p+\beta_1 I_{B3}+\beta_2 I_{C3})+\epsilon(\mu+\beta_3 I_{C3})}. \end{cases} \tag{13}$$

While (I_{B3}, I_{C3}) is the positive intersection point of the two isoclines

$$\begin{aligned}
f(I_B, I_C) &= \left\{(b\theta-\mu-d_1-r_1)[(\mu+\psi+\beta_3 I_C)(\mu+p+\beta_1 I_B+\beta_2 I_C)\right.\\
&\quad \left. + \epsilon(\mu+\beta_3 I_C)]\right\} I_B\\
&\quad + b(\beta_1 I_B)(1-\theta I_B)(\mu+\psi+\beta_3 I_C) = 0,
\end{aligned} \tag{14}$$

$$\begin{aligned}
g(I_B, I_C) &= b(1-\theta I_B)(\mu+\psi+\beta_3 I_C)(\beta_2 I_C+(1-\sigma)p) + \epsilon b(1-\theta I_B)\beta_3 I_C\\
&\quad - \left\{(\mu+d_2+r_2)[(\mu+\psi+\beta_3 I_C)(\mu+p+\beta_1 I_B+\beta_2 I_C)\right.\\
&\quad \left. + \epsilon(\mu+\beta_3 I_C)]\right\} I_C = 0.
\end{aligned} \tag{15}$$

Next, if $I_C \to 0$, we obtain

$$f(I_B) = C_1 I_B^2 + C_2 I_B + C_3 = 0, \tag{16}$$

with

$$\begin{aligned}
C_1 &= -\beta_1(\mu+\psi)(\mu+d_1+r_1) < 0,\\
C_2 &= (b\theta-\mu-r_1-d_1)[(\mu+\psi)(\mu+p)+\epsilon\mu] + b(\mu+\psi)(\beta_1-\sigma p\theta),\\
C_3 &= bp\sigma(\mu+\psi) > 0.
\end{aligned}$$

$$g(I_B) = bp(\mu+\psi)(1-\sigma)(1-\theta I_B) = 0. \tag{17}$$

Hence, (16) intersects the I-axis at $\widetilde{I_B}$ which is positive. Also, (17) intersects the I-axis at$\widehat{I_B} = \frac{1}{\theta}$, which also positive. Uniqueness can be deduce if the following condition holds (see Fig. 2)

$$\begin{cases} C_2 < 0, \\ or \\ C_2 > 0, \end{cases} \tag{18}$$

$$\widehat{I_B} < \widetilde{I_B}, \tag{19}$$

$$\begin{cases} \frac{dI_B}{dI_C} = -\frac{\partial f/\partial I_C}{\partial f/\partial I_B} < 0, \\ \frac{dI_B}{dI_C} = -\frac{\partial g/\partial I_C}{\partial g/\partial I_B} > 0. \end{cases} \tag{20}$$

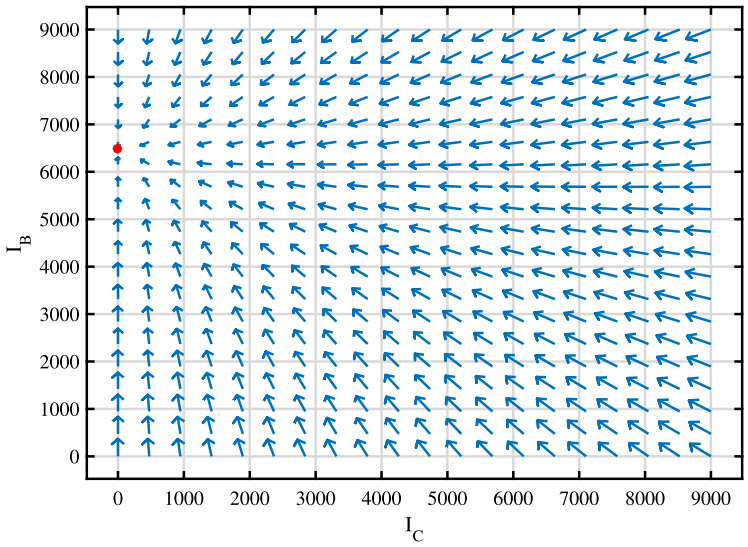

Fig. 2. A unique positive intersection point.

5. Local stability analysis

In this part, the local behaviour of above equilibrium points is explored by calculating the model's (5) Jacobian matrix about an arbitrary point $e^* = (S, V, I_B, I_C)$:

$$J(e^*) = \begin{pmatrix} -(\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C) & \psi & -(b\theta+\beta_1 S) & -\beta_2 S \\ \epsilon & -(\mu+\psi+\beta_3 I_C) & 0 & -\beta_3 V \\ \beta_1 I_B+\sigma p & 0 & b\theta+\beta_1 S-k_1 & 0 \\ \beta_2 I_C+(1-\sigma)p & \beta_3 I_C & 0 & \beta_2 S+\beta_3 V-k_2 \end{pmatrix}, \tag{21}$$

where $k_1 = \mu + d_1 + r_1$ and $k_2 = \mu + d_2 + r_2$. Now, the Jacobian matrix (21) at (UVHE) is

$$J(e_0) = \begin{pmatrix} -(\mu+\epsilon) & \psi & -(b\theta+\beta_1 S_0) & -\beta_2 S_0 \\ \epsilon & -(\mu+\psi) & 0 & -\beta_3 V_0 \\ 0 & 0 & b\theta+\beta_1 S_0-(\mu+d_1+r_1) & 0 \\ 0 & 0 & 0 & \beta_2 S_0+\beta_3 V_0-(\mu+d_2+r_2) \end{pmatrix}. \tag{22}$$

Then, the characteristic equation is

$$[b\theta+\beta_1 S_0-(\mu+d_1+r_1)-\lambda][\beta_2 S_0+\beta_3 V_0-(\mu+d_2+r_2)-\lambda][\lambda^2+T_0\lambda+D_0] = 0, \tag{23}$$

here

$$T_0 = 2\mu+\epsilon+\psi > 0,$$
$$D_0 = \mu(\mu+\psi+\epsilon) > 0.$$

Hence, the eigenvalues in following

$$\begin{cases} \lambda_1 = -(\mu+\epsilon+\psi), \\ \lambda_2 = -\mu, \\ \lambda_3 = b\theta+\beta_1 S_0-(\mu+d_1+r_1), \\ \lambda_4 = \beta_2 S_0+\beta_3 V_0-(\mu+d_2+r_2). \end{cases}$$

It is easy see that, $\lambda_i, i = 1,2$ have always negative real parts, whereas $\lambda_i, i = 3,4$ are negative real parts if $\mathcal{R}_0 < 1$. Then, e_0 is (LAS). But, it is unstable point otherwise.

Also, the Jacobian matrix at (HBVFE) in the following result

$$J(e_1) = \begin{pmatrix} m_{11} & m_{12} & m_{13} & m_{14} \\ m_{21} & m_{22} & 0 & m_{24} \\ 0 & 0 & m_{33} & 0 \\ m_{41} & m_{42} & 0 & m_{44} \end{pmatrix}. \tag{24}$$

Here

$$m_{11} = -(\mu+\epsilon+p+\beta_2 I_{C1}) \;;\; m_{12} = \psi \;;\; m_{13} = -(b\theta+\beta_1 S_1) \;;\; m_{14} = -\beta_2 S_1$$
$$m_{21} = \epsilon \;;\; m_{22} = -(\mu+\psi+\beta_3 I_{C1}) \;;\; m_{24} = -\beta_3 V_1$$
$$m_{33} = b\theta+\beta_1 S_1-(\mu+d_1+r_1) \;;\; m_{41} = \beta_2 I_{C1}+p \;;\; m_{42} = \beta_3 I_{C1}$$
$$m_{44} = \beta_2 S_1+\beta_3 V_1-(\mu+d_2+r_2) \;;\; m_{23} = m_{31} = m_{23} = m_{34} = m_{43} = 0.$$

Direct computations show that this Jacobian matrix (24) has the following characteristic equation:

$$[b\theta+\beta_1 S_1-(\mu+d_1+r_1)-\lambda][\lambda^3+M_1\lambda^2+M_2\lambda+M_3] = 0, \tag{25}$$

where

$$M_1 = -(m_{11}+m_{22}+m_{44}),$$
$$M_2 = (m_{11}m_{22}-m_{12}m_{21}+m_{11}m_{44}-m_{14}m_{41}+m_{22}m_{44}-m_{24}m_{42}),$$
$$M_3 = -(m_{44}(m_{11}m_{22}-m_{12}m_{21})+m_{41}(m_{12}m_{24}-m_{22}m_{14})+m_{42}(m_{21}m_{14}-m_{11}m_{24})).$$

Obviously, one of the eigenvalues is $b\theta+\beta_1 S_1-(\mu+d_1+r_1) < 0$ when $m_{33} < 0$. The other eigenvalues have negative real parts when the $(R-H)$ conditions are satisfied (i.e. $M_i > 0$ for $i = 1,3$ and $M_1 M_2 - M_3 > 0$). So, these conditions are true if and only if $m_{44} < 0$ and $m_{11}m_{22} > m_{12}m_{21}$. Therefore the e_1 of the model (5) is (LAS).

Now, the Jacobian matrix at (HCVFE) in the following result

$$J(e_2) = \begin{pmatrix} w_{11} & w_{12} & w_{13} & w_{14} \\ w_{21} & w_{22} & 0 & w_{24} \\ w_{31} & 0 & w_{33} & 0 \\ 0 & 0 & 0 & w_{44} \end{pmatrix}. \tag{26}$$

Here

$$w_{11} = -(\mu+\epsilon+p+\beta_1 I_{B1}) \;;\; w_{12} = \psi \;;\; w_{13} = -(b\theta+\beta_1 S_2) \;;\; w_{14} = -\beta_2 S_2$$
$$w_{21} = \epsilon \;;\; w_{22} = -(\mu+\psi) \;;\; w_{24} = -\beta_3 V_2$$
$$w_{31} = \beta_1 I_{B1}+p \;;\; w_{33} = b\theta+\beta_1 S_2-(\mu+d_1+r_1) \;;$$
$$w_{44} = \beta_2 S_2+\beta_3 V_2-(\mu+d_2+r_2)$$
$$w_{23} = w_{32} = w_{34} = w_{41} = w_{42} = w_{43} = 0.$$

Direct computations show that this Jacobian matrix (26) has the following characteristic equation:

$$[\beta_2 S_2+\beta_3 V_2-(\mu+d_2+r_2)-\lambda][\lambda^3+W_1\lambda^2+W_2\lambda+W_3] = 0, \tag{27}$$

where

$$W_1 = -(w_{11}+w_{22}+w_{33}),$$
$$W_2 = (w_{11}w_{22}-w_{12}w_{21}+w_{11}w_{33}-w_{13}w_{31}+w_{22}w_{33}),$$
$$W_3 = -(w_{33}(w_{11}w_{22}-w_{12}w_{21})-w_{22}w_{13}w_{31}).$$

Obviously, one of the eigenvalues is $\beta_2 S_2+\beta_3 V_2-(\mu+d_2+r_2) < 0$ when $w_{44} < 0$ The other eigenvalues have negative real parts when

the $(R-H)$ conditions are satisfied (i.e. $W_i > 0$ for $i = 1,3$ and $W_1W_2 - W_3 > 0$). So, these conditions are true if and only if $w_{33} < 0$ and $w_{11}w_{22} > w_{12}w_{21}$. Therefore the e_2 of the model (5) is (LAS).

The last point's (EE) Jacobian matrix is

$$J(e_3) = \begin{pmatrix} q_{11} & q_{12} & q_{13} & q_{14} \\ q_{21} & q_{22} & 0 & q_{24} \\ q_{31} & 0 & q_{33} & 0 \\ q_{41} & q_{42} & 0 & q_{44} \end{pmatrix}. \tag{28}$$

Here

$$q_{11} = -(\mu+\epsilon+p+\beta_1 I_{B3}+\beta_2 I_{C3}) \;;\; q_{12} = \psi \;;\; q_{13} = -(b\theta+\beta_1 S_3)$$
$$q_{14} = -\beta_2 S_3 q_{21} = \epsilon \;;\; q_{22} = -(\mu+\psi+\beta_3 I_{C3}) \;;\; q_{24} = -\beta_3 V_3$$
$$q_{31} = \beta_1 I_{B3}+\sigma p \;;\; q_{33} = b\theta+\beta_1 S_3-(\mu+d_1+r_1)$$
$$q_{41} = \beta_2 I_{C3}+(1-\sigma)p \;;\; q_{42} = \beta_3 I_{C3} \;;\; q_{44} = \beta_2 S_3+\beta_3 V_3-(\mu+d_2+r_2).$$

Applying Gersgorin's theorem if the condition $\mid q_{ii} \mid > \sum_{\substack{i=1, \\ i\neq j}}^{4} \mid q_{ij} \mid$, holds. Then, we obtain the eigenvalues of (28) lie in the union of the following circles in the complex plane:

centre in $q_{11}+0i$, radius $R_1 = min\{q_{21}+q_{31}+q_{41}, q_{12}+q_{13}+q_{14}\}$;
centre in $q_{22}+0i$, radius $R_2 = min\{q_{12}+q_{42}, q_{21}+q_{24}\}$;
centre in $q_{33}+0i$, radius $R_3 = min\{q_{13}, q_{31}\}$;
centre in $q_{44}+0i$, radius $R_4 = min\{q_{14}+q_{24}, q_{41}+q_{42}\}$;

Hence, the Jacobian matrix (28) at (EE), has four eigenvalues with negative real part when $\mathcal{R}_0 > 1$. Therefore the e_3 is (LAS). Otherwise it is unstable point.

6. Global stability

In this part, the global behaviour of above equilibrium points is explored by applying the results of Castillo-Chavez's method[24] on (UVHE). While, the other equilibrium points (HBVFE, HCVFE and EE) respectively can be find the basin of attraction of them by applying the LaSalle method[25] and building a proper Lyapunov function.

Theorem 6.1. *The UVHE point is globally asymptotically stable when $\mathcal{R}_0 < 1$, otherwise is unstable.*

Proof. Letting $Y = (S, V)$ be the susceptible class and $Z = (I_B, I_C)$ represent to the viral hepatitis B and C infected. Hence,

$$\frac{dX}{dt} = K(Y,Z) = \begin{cases} b(1-\theta I_B)-(\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C)S+\psi V \\ \epsilon S-(\mu+\psi+\beta_3 I_C)V. \end{cases}$$

If $S = S_0, V = V_0$ and $K(Y,0) = 0$, then,

$$\frac{dX}{dt} = \begin{cases} b-(\mu+\epsilon)S_0+\psi V_0 \\ \epsilon S_0-(\mu+\psi)V_0. \end{cases}$$

As $t \to \infty$, and $Y \to Y_0$. Therefore, $Y = Y_0 = (S_0, V_0)$ is globally asymptotically stable. Now,

$$BZ - \bar{G}(Y,Z) = \begin{pmatrix} b\theta+\beta_1 S_0-(\mu+d_1+r_1) & 0 \\ 0 & \beta_2 S_0+\beta_3 V_0-(\mu+d_2+r_2) \end{pmatrix} \cdot \begin{pmatrix} I_B \\ I_V \end{pmatrix} - \begin{pmatrix} \beta_1 I_B[S_0-S]+\sigma PS \\ \beta_2 I_C[S_0-S]+\beta_3 I_C[V_0-V]+(1-\sigma)PS \end{pmatrix}.$$

Hence

$$B = \begin{pmatrix} b\theta+\beta_1 S_0-(\mu+d_1+r_1) & 0 \\ 0 & \beta_2 S_0+\beta_3 V_0-(\mu+d_2+r_2) \end{pmatrix}; \quad Z = \begin{pmatrix} I_B \\ I_V \end{pmatrix}$$

and

$$\bar{G}(Y,Z) = \begin{pmatrix} \beta_1 I_B[S_0-S]+\sigma PS \\ \beta_2 I_C[S_0-S]+\beta_3 I_C[V_0-V]+(1-\sigma)PS \end{pmatrix}.$$

In model (5), $S_0+V_0 \le \frac{b}{\mu}$, is the bound for the population, clearly, $S, V, I_B, I_C \le \frac{b}{\mu}$. Then, $\bar{G}(Y,Z) \ge 0$. Thus, the disease free equilibrium point e_0 is globally asymptotically stable for $\mathcal{R}_0 < 1$.

Theorem 6.2. *The HBVFE, is global asymptotically stable when $\mathcal{R}_{0B} \le 1 < \mathcal{R}_0$, otherwise is unstable.*

Proof. To discuss that, we consider the following Lyapunov function

$$L_1 = \int_{S_1}^{S}(1-\frac{S_1}{x})dx + \int_{V_1}^{V}(1-\frac{V_1}{x})dx + I_B + \int_{I_{C1}}^{I_C}(1-\frac{I_{C1}}{x})dx. \tag{29}$$

The derivative of $L_1(t)$ corresponding to the model solutions is

$$\frac{dL_1}{dt} = (1-\frac{S_1}{S})\frac{dS}{dt} + (1-\frac{V_1}{V})\frac{dV}{dt} + \frac{dI_B}{dt} + (1-\frac{I_{C1}}{I_C})\frac{dI_C}{dt}. \tag{30}$$

In following from direct simplify

$$\begin{aligned} \left(1-\frac{S_1}{S}\right)\frac{dS}{dt} &= (1-\tfrac{S_1}{S})[b(1-\theta I_B)-(\mu+\epsilon+b+\beta_1 I_B+\beta_2 I_C)S+\psi V] \\ &= (1-\tfrac{S_1}{S})[-\theta b I_B-(\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C)S+\psi V \\ &\quad +(\mu+\epsilon+p+\beta_2 I_{C1})S_1-\psi V_1] \\ &= \beta_2 S_1 I_{C1}[1-\tfrac{S_1}{S}-\tfrac{SI_C}{S_1 I_{C1}}+\tfrac{I_C}{I_{C1}}]+\beta_1 S_1 I_B[1-\tfrac{S}{S_1}-\tfrac{\theta b}{\beta_1 S_1}+\tfrac{\theta b}{\beta_1 S}] \\ &\quad +(\mu+\epsilon+p)S_1[1-\tfrac{S_1}{S}-\tfrac{S}{S_1}+1]+\tfrac{\psi S_1 V_1}{S}[1-\tfrac{V}{V_1}-\tfrac{s}{S_1}+\tfrac{VS}{V_1 S_1}], \end{aligned} \tag{31}$$

$$\begin{aligned} \left(1-\frac{V_1}{V}\right)\frac{dV}{dt} &= (1-\tfrac{V_1}{V})[\epsilon S-(\mu+\psi+\beta_3 I_C)V] \\ &= (1-\tfrac{V_1}{V})[\epsilon S-(\mu+\psi+\beta_3 I_C)V-\epsilon S_1+(\mu+\psi+\beta_3 I_{C1})V_1] \\ &= \beta_3 V_1 I_{C1}[1-\tfrac{V_1}{V}-\tfrac{VI_C}{V_1 I_{C1}}+\tfrac{I_C}{I_{C1}}]+\tfrac{\epsilon S_1 V_1}{V}[1-\tfrac{S}{S_1}-\tfrac{V}{V_1}+\tfrac{SV}{S_1 V_1}] \\ &\quad +(\mu+\psi)V_1[1-\tfrac{V_1}{V}-\tfrac{V}{V_1}+1], \end{aligned} \tag{32}$$

$$\frac{dI_B}{dt} = [b\theta+\beta_1 S-\mu-d_1-r_1]I_B, \tag{33}$$

$$\begin{aligned} \left(1-\frac{I_{C1}}{I_C}\right)\frac{dI_C}{dt} &= (1-\tfrac{I_{C1}}{I_C})[(\beta_2 S+\beta_3 V-\mu-d_2-r_2)I_C+pS] \\ &= (1-\tfrac{I_{C1}}{I_C})[(\beta_2 S+\beta_3 V-\mu-d_2-r_2)I_C+pS \\ &\quad -(\beta_2 S_1+\beta_3 V_1-\mu-d_2-r_2)I_{C1}-pS_1] \\ &= \tfrac{\beta_2 S_1 I_{C1}^2}{I_C}[1-\tfrac{I_C}{I_{C1}}-\tfrac{SI_C}{S_1 I_{C1}}+\tfrac{SI_C^2}{S_1 I_{C1}^2}] \\ &\quad +\tfrac{\beta_3 V_1 I_{C1}^2}{I_C}[1-\tfrac{I_C}{I_{C1}}-\tfrac{VI_C}{V_1 I_{C1}}+\tfrac{VI_C^2}{V_1 I_{C1}^2}] \\ &\quad +(\mu+d_2+r_2)[1-\tfrac{I_{C1}}{I_C}-\tfrac{I_C}{I_{C1}}+1] \\ &\quad +\tfrac{pS_1 I_{C1}}{I_C}[1-\tfrac{S}{S_1}-\tfrac{I_C}{I_{C1}}+\tfrac{SI_C}{S_1 I_{C1}}]. \end{aligned} \tag{34}$$

Now, putting the Eqs. (31)–(34) in to Eq. (30), we get

$$\begin{aligned} \tfrac{dL_1}{dt} &= \beta_2 S_1 I_{C1}[1-\tfrac{S_1}{S}+\tfrac{I_C}{I_{C1}}(1-\tfrac{S}{S_1})]+\beta_1 S_1 I_B[1-\tfrac{S}{S_1}+\tfrac{\theta b}{\beta_1 S}(1-\tfrac{S}{S_1})] \\ &\quad +(\mu+\epsilon+p)S_1 I_C[2-\tfrac{S_1}{S}-\tfrac{S}{S_1}]+\tfrac{\psi S_1 V_1}{S}[1-\tfrac{V}{V_1}+\tfrac{VS}{V_1 S_1}(1-\tfrac{SV_1}{S_1 V})] \\ &\quad +\beta_3 V_1 I_{C1}[1-\tfrac{V_1}{V}+\tfrac{I_C}{I_{C1}}(1-\tfrac{V}{V_1})]+\tfrac{\epsilon S_1 V_1}{V}[1-\tfrac{V}{V_1}+\tfrac{VS}{V_1 S_1}(1-\tfrac{SV_1}{S_1 V})] \\ &\quad +(\mu+\psi)V_1[2-\tfrac{V_1}{V}-\tfrac{V}{V_1}]+(\mu+d_2+r_2)[2-\tfrac{I_{C1}}{I_C}-\tfrac{I_C}{I_{C1}}] \\ &\quad +\tfrac{pS_1 I_{C1}}{I_C}[1-\tfrac{I_C}{I_{C1}}+\tfrac{SI_C}{S_1 I_{C1}}(1-\tfrac{I_C S_1}{I_{C1} S})]+[b\theta+\beta_1 S-\mu-d_1-r_1]I_B \\ &\quad +\tfrac{\beta_2 S_1 I_{C1}^2}{I_C}[1-\tfrac{I_C}{I_{C1}}+\tfrac{SI_C^2}{S_1 I_{C1}^2}(1-\tfrac{I_{C1}}{I_C})]+\tfrac{\beta_3 V_1 I_{C1}^2}{I_C}[1-\tfrac{I_C}{I_{C1}}+\tfrac{VI_C^2}{V_1 I_{C1}^2}(1-\tfrac{I_{C1}}{I_C})]. \end{aligned} \tag{35}$$

Obviously, in Eq. (35),

$$\left[1-\frac{I_C}{I_{C1}}+\frac{SI_C^2}{S_1I_{C1}^2}(1-\frac{I_{C1}}{I_C})\right]\le 0,$$
$$\left[1-\frac{S}{S_1}+\frac{\theta b}{\beta_1 S}(1-\frac{S}{S_1})\right]\le 0,$$
$$\left[1-\frac{V}{V_1}+\frac{VS}{V_1S_1}(1-\frac{SV_1}{S_1V})\right]\le 0,$$
$$\left[1-\frac{V_1}{V}+\frac{I_C}{I_{C1}}(1-\frac{V}{V_1})\right]\le 0,$$
$$\left[b\theta+\beta_1 S-\mu-d_1-r_1\right]I_B\le 0.$$

Therefore, using the result of LaSalle Invariance principle, the HB-VFE has a basin of attraction under $\mathcal{R}_{0B}\le 1<\mathcal{R}_0$.

Theorem 6.3. *The HCVFE, is global asymptotically stable when $\mathcal{R}_{0C}\le 1<\mathcal{R}_0$, otherwise is unstable.*

Proof. To discuss that, we consider the following Lyapunov function

$$L_2=\int_{S_2}^{S}(1-\frac{S_2}{x})dx+\int_{V_2}^{V}(1-\frac{V_2}{x})dx+\int_{I_{B2}}^{I_B}(1-\frac{I_{B2}}{x})dx+I_C. \tag{36}$$

The derivative of $L_2(t)$ corresponding to the model solutions is

$$\frac{dL_2}{dt}=(1-\frac{S_2}{S})\frac{dS}{dt}+(1-\frac{V_2}{V})\frac{dV}{dt}+(1-\frac{I_{B2}}{I_B})\frac{dI_B}{dt}+\frac{dI_C}{dt}. \tag{37}$$

In following from direct simplify

$$\begin{aligned}\left(1-\tfrac{S_2}{S}\right)\tfrac{dS}{dt}&=(1-\tfrac{S_2}{S})[b(1-\theta I_B)-(\mu+\epsilon+b+\beta_1 I_B+\beta_2 I_C)S+\psi V]\\&=(1-\tfrac{S_2}{S})[-\theta b I_B-(\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C)S+\psi V\\&\quad+\theta b I_{B2}+(\mu+\epsilon+p+\beta_1 I_{B2})S_2-\psi V_2]\\&=\beta_1 S_2 I_{B2}[1-\tfrac{S_2}{S}-\tfrac{SI_B}{S_2I_{B2}}+\tfrac{I_B}{I_{B2}}]+(\mu+\epsilon+p)[1-\tfrac{S_2}{S}-\tfrac{S}{S_2}+1]\\&\quad+\tfrac{\psi V_2S_2}{S}[1-\tfrac{V}{V_2}-\tfrac{S}{S_2}+\tfrac{VS}{V_2S_2}]+b\theta I_{B2}[1-\tfrac{S_2}{S}-\tfrac{I_B}{I_{B2}}+\tfrac{S_2I_B}{SI_{B2}}]\\&\quad+\beta_2 I_C S_2[1-\tfrac{S}{S_2}],\end{aligned} \tag{38}$$

$$\begin{aligned}\left(1-\tfrac{V_2}{V}\right)\tfrac{dV}{dt}&=(1-\tfrac{V_2}{V})[\epsilon S-(\mu+\psi+\beta_3 I_C)V]\\&=(1-\tfrac{V_2}{V})[\epsilon S-(\mu+\psi+\beta_3 I_C)V-\epsilon S_2+(\mu+\psi)V_2]\\&=\tfrac{\epsilon S_2V_2}{V}[1-\tfrac{S}{S_2}-\tfrac{V}{V_2}+\tfrac{SV}{S_2V_2}]+(\mu+\psi)[1-\tfrac{V_2}{V}-\tfrac{V}{V_2}+1]\\&=\beta_3 I_C V_2[1-\tfrac{V}{V_2}],\end{aligned} \tag{39}$$

$$\begin{aligned}\left(1-\tfrac{I_{B2}}{I_B}\right)\tfrac{dI_B}{dt}&=(1-\tfrac{I_{B2}}{I_B})[(b\theta+\beta_1 S-\mu-d_1-r_1)I_B+\sigma pS]\\&=(1-\tfrac{I_{B2}}{I_B})[(b\theta+\beta_1 S-\mu-d_1-r_1)I_B+\sigma pS\\&\quad-(b\theta+\beta_1 S_2-\mu-d_1-r_1)I_{B2}-pS_2]\\&=\tfrac{\beta_1 S_2 I_{B2}^2}{I_B}[1-\tfrac{I_B}{I_{B2}}+\tfrac{SI_B^2}{S_2I_{B2}^2}-\tfrac{S}{I_B}S_2I_{B2}]\\&\quad+(\mu+d_1+r_1)I_{B2}[1-\tfrac{I_{B2}}{I_B}-\tfrac{I_B}{I_{B2}}+1]\\&\quad+\tfrac{pS_2I_{B2}}{I_B}[1-\tfrac{\sigma S}{S_2}-\tfrac{I_B}{I_{B2}}+\tfrac{\sigma SI_B}{S_2I_{B2}}]+\tfrac{b\theta I_{B2}^2}{I_B}[1-\tfrac{I_B}{I_{B2}}-\tfrac{I_B}{I_{B2}}+\tfrac{I_B^2}{I_{B2}^2}],\end{aligned} \tag{40}$$

$$\frac{dI_C}{dt}=[\beta_2 S+\beta_3 V-\mu-d_2-r_2], \tag{41}$$

Thus, by putting Eqs. (38)–(41) in to Eq. (37), we get

$$\begin{aligned}\tfrac{dL_2}{dt}&=\beta_1 S_2 I_{B2}[1-\tfrac{S_2}{S}+\tfrac{I_B}{I_{B2}}(1-\tfrac{S}{S_2})]+(\mu+\epsilon+p)S_2[2-\tfrac{S_2}{S}-\tfrac{S}{S2}]\\&\quad+\tfrac{\psi V_2S_2}{S}[1-\tfrac{S}{S_2}+\tfrac{VS}{V_2S_2}(1-\tfrac{S_2}{S})]+b\theta I_{B2}[1-\tfrac{S_2}{S}+\tfrac{S_2I_B}{SI_{B2}}(1-\tfrac{S}{S_2})]\\&\quad+\beta_2 I_C S_2[1-\tfrac{S}{S_2}]+\tfrac{\epsilon S_2V_2}{V}[1-\tfrac{S}{S_2}+\tfrac{VS}{V_2S_2}(1-\tfrac{S_2}{S})]+\beta_3 I_C V_2[1-\tfrac{V}{V_2}]\\&\quad+(\mu+\psi)V_2[2-\tfrac{V_2}{V}-\tfrac{V}{V_2}]+(\mu+d_1+r_1)I_{B2}[2-\tfrac{I_{B2}}{I_B}-\tfrac{I_B}{I_{B2}}]\\&\quad+\tfrac{pS_2I_{B2}}{I_B}[1-\tfrac{I_B}{I_{B2}}+\tfrac{\sigma SI_B}{S_2I_{B2}}(1-\tfrac{I_{B2}}{I_B})]+[\beta_2 S+\beta_3 V-\mu-d_2-r_2]I_C\\&\quad+\tfrac{\beta_1 S_2 I_{B2}^2}{I_B}[1-\tfrac{I_B}{I_{B2}}+\tfrac{SI_B^2}{S_2I_{B2}^2}(1-\tfrac{I_{B2}}{I_B})]+\tfrac{b\theta I_{B2}^2}{I_B}[1-\tfrac{I_B}{I_{B2}}+\tfrac{I_B^2}{I_{B2}^2}(1-\tfrac{I_{B2}}{I_B})].\end{aligned} \tag{42}$$

Obviously, in Eq. (42),

$$\left[1-\frac{S}{S_2}+\frac{VS}{V_2S_2}(1-\frac{S_2}{S})\right]\le 0,$$
$$\left[1-\frac{I_B}{I_{B2}}+\frac{\sigma SI_B}{S_2I_{B2}}(1-\frac{I_{B2}}{I_B})\right]\le 0,$$
$$\left[1-\frac{V}{V_2}\right]\le 0,$$
$$\left[\beta_2 S+\beta_3 V-\mu-d_2-r_2\right]I_C\le 0.$$

Therefore, using the result of LaSalle Invariance principle, the HCVFE has a basin of attraction under $\mathcal{R}_{0C}\le 1<\mathcal{R}_0$.

Theorem 6.4. *The EE, is global asymptotically stable when $\mathcal{R}_0>1$, otherwise is unstable.*

Proof. To discuss that, we consider the following Lyapunov function

$$L_3=\int_{S_3}^{S}(1-\frac{S_3}{x})dx+\int_{V_3}^{V}(1-\frac{V_3}{x})dx+\int_{I_{B3}}^{I_B}(1-\frac{I_{B3}}{x})dx+\int_{I_{C3}}^{I_C}(1-\frac{I_{C3}}{x})dx. \tag{43}$$

The derivative of $L_3(t)$ corresponding to the model solutions is

$$\frac{dL_3}{dt}=(1-\frac{S_3}{S})\frac{dS}{dt}+(1-\frac{V_3}{V})\frac{dV}{dt}+(1-\frac{I_{B3}}{I_B})\frac{dI_B}{dt}+(1-\frac{I_{C3}}{I_C})\frac{dI_C}{dt}. \tag{44}$$

In following from direct calculation

$$\begin{aligned}\left(1-\tfrac{S_3}{S}\right)\tfrac{dS}{dt}&=\beta_1 S_3 I_{B3}[1-\tfrac{S_3}{S}-\tfrac{SI_B}{S_3I_{B3}}+\tfrac{I_B}{I_{B3}}]+\beta_2 S_3 I_{C3}[1-\tfrac{S_3}{S}-\tfrac{SI_C}{S_3I_{C3}}+\tfrac{I_C}{I_{C3}}]\\&\quad+(\mu+\epsilon+p)S_3[1-\tfrac{S_3}{S}-\tfrac{S}{S_3}+1]+\tfrac{\psi S_3V_3}{S}[1-\tfrac{V}{V_3}-\tfrac{S}{S_3}+\tfrac{SV}{S_3V_3}]\\&\quad+\tfrac{b\theta S_3I_B}{S}[1-\tfrac{I_{B3}}{I_B}-\tfrac{S}{S_3}+\tfrac{SI_{B3}}{S_3I_B}],\end{aligned} \tag{45}$$

$$\begin{aligned}\left(1-\tfrac{V_3}{V}\right)\tfrac{dV}{dt}&=\beta_3 V_3 I_{C3}[1-\tfrac{V_3}{V}-\tfrac{VI_C}{V_3I_{C3}}+\tfrac{I_C}{I_{C3}}]+(\mu+\psi)V_3[1-\tfrac{V_3}{V}-\tfrac{V}{V_3}+1]\\&\quad+\tfrac{\epsilon S_3V_3}{V}[1-\tfrac{S}{S_3}-\tfrac{V}{V_3}+\tfrac{SV}{S_3V_3}],\end{aligned} \tag{46}$$

$$\begin{aligned}\left(1-\tfrac{I_{B3}}{I_B}\right)\tfrac{dI_B}{dt}&=\tfrac{\beta_1 S_3 I_{B3}^2}{I_B}[1-\tfrac{I_B}{I_{B3}}-\tfrac{SI_B}{S_3I_{B3}}+\tfrac{SI_B^2}{S_3I_{B3}^2}]\\&\quad+(\mu+d_1+r_1)I_{B3}[1-\tfrac{I_{B3}}{I_B}-\tfrac{I_B}{I_{B3}}+1]\\&\quad+\tfrac{b\theta I_{B3}^2}{I_B}[1-\tfrac{I_B}{I_{B3}}-\tfrac{I_B}{I_{B3}}+\tfrac{I_B^2}{I_{B3}^2}]+\tfrac{p\sigma S_3I_{B3}}{I_B}[1-\tfrac{S}{S_3}-\tfrac{I_B}{I_{B3}}+\tfrac{SI_B}{S_3I_{B3}}],\end{aligned} \tag{47}$$

$$\begin{aligned}\left(1-\tfrac{I_{C3}}{I_C}\right)\tfrac{dI_C}{dt}&=\tfrac{\beta_2 S_3 I_{C3}^2}{I_C}[1-\tfrac{I_C}{I_{C3}}-\tfrac{SI_C}{S_3I_{C3}}+\tfrac{SI_C^2}{S_3I_{C3}^2}]\\&\quad+\tfrac{\beta_3 V_3 I_{C3}^2}{I_C}[1-\tfrac{I_C}{I_{C3}}-\tfrac{VI_C}{V_3I_{C3}}+\tfrac{VI_C^2}{V_3I_{C3}^2}]\\&\quad+(\mu+d_2+r_2)I_{C3}[1-\tfrac{I_{C3}}{I_C}-\tfrac{I_C}{I_{C3}}+1]\\&\quad+\tfrac{(1-\sigma)pS_3I_{C3}}{I_C}[1-\tfrac{S}{S_3}-\tfrac{I_C}{I_{C3}}+\tfrac{SI_C}{S_3I_{C3}}].\end{aligned} \tag{48}$$

Now, by putting Eqs. (45)–(48) in to Eq. (44), we get

$$\begin{aligned}\tfrac{dL_3}{dt}&=\beta_1 S_3 I_{B3} I_C I_B[1-\tfrac{S_3}{S}+\tfrac{I_B}{I_{B3}}(1-\tfrac{S}{S_3})]+\beta_2 S_3 I_{C3}[1-\tfrac{S_3}{S}+\tfrac{I_C}{I_{C3}}(1-\tfrac{S}{S_3})]\\&\quad+\beta_3 V_3 I_{C3}[1-\tfrac{V_3}{V}+\tfrac{I_C}{I_{C3}}(1-\tfrac{V}{V_3})]+\tfrac{\psi S_3V_3}{S}[1-\tfrac{V}{V_3}+\tfrac{SV}{S_3V_3}(1-\tfrac{V_3}{V})]\\&\quad+\tfrac{\epsilon S_3V_3}{V}[1-\tfrac{V}{V_3}+\tfrac{SV}{S_3V_3}(1-\tfrac{V_3}{V})]+(\mu+\epsilon+p)S_3[2-\tfrac{S_3}{S}-\tfrac{S}{S_3}]\end{aligned}$$

$$+(\mu+\psi)V_3[2-\tfrac{V_3}{V}-\tfrac{V}{V_3}]+(\mu+d_1+r_1)I_{B3}[2-\tfrac{I_{B3}}{I_B}-\tfrac{I_B}{I_{B3}}]$$
$$+(\mu+d_2+r_2)I_{C3}[2-\tfrac{I_{C3}}{I_B}-\tfrac{I_B}{I_{B3}}]+\tfrac{b\theta S_3 I_B}{S}[1-\tfrac{I_{B3}}{I_B}+\tfrac{SI_{B3}}{S_3 I_B}(1-\tfrac{I_B}{I_{B3}})]$$
$$+\tfrac{b\theta I_{B3}^2}{I_B}[1-\tfrac{I_B}{I_{B3}}+\tfrac{I_B^2}{I_{B3}^2}(1-\tfrac{I_B}{I_{B3}})]+\tfrac{\sigma p S_3 I_{B3}}{I_B}[1-\tfrac{I_B}{I_{B3}}+\tfrac{SI_B}{S_3 I_{B3}}(1-\tfrac{I_{B3}}{I_B})]$$
$$+\tfrac{(1-\sigma)pS_3 I_{C3}}{I_C}[1-\tfrac{I_C}{I_{C3}}+\tfrac{SI_C}{S_3 I_{C3}}(1-\tfrac{I_{C3}}{I_C})]+\tfrac{\beta_1 S_3 I_{B3}^2}{I_B}[1-\tfrac{I_B}{I_{B3}}+\tfrac{SI_B^2}{S_3 I_{B3}^2}(1-\tfrac{I_{B3}}{I_B})]$$
$$+\tfrac{\beta_2 S_3 I_{C3}^2}{I_C}[1-\tfrac{I_C}{I_{C3}}+\tfrac{SI_C^2}{S_3 I_{C3}^2}(1-\tfrac{I_{C3}}{I_C})]+\tfrac{\beta_3 V_3 I_{C3}^2}{I_C}[1-\tfrac{I_C}{I_{C3}}+\tfrac{VI_C^2}{V_3 I_{C3}^2}(1-\tfrac{I_{C3}}{I_C})]. \tag{49}$$

Now, in Eq. (49) if the following

$$\left[1-\frac{S_3}{S}+\frac{I_B}{I_{B3}}(1-\frac{S}{S_3})\right]\le 0$$
$$\left[1-\frac{I_B}{I_{B3}}+\frac{SI_B^2}{S_3 I_{B3}^2}(1-\frac{I_{B3}}{I_B})\right]\le 0$$
$$\left[1-\frac{I_C}{I_{C3}}+\frac{VI_C^2}{V_3 I_{C3}^2}(1-\frac{I_{C3}}{I_C})\right]\le 0$$
$$\left[1-\frac{V}{V_3}+\frac{SV}{S_3 V_3}(1-\frac{V_3}{V})\right]\le 0$$

Therefore, using the result of LaSalle Invariance principle, the HB-VFE has a basin of attraction under $\mathcal{R}_0>1$, otherwise is unstable.

7. Fractional-order viral hepatitis model

In this part, we apply a fractional-order derivative effect on the viral hepatitis disease. We study the dynamic behaviour by using Caputo–Fabrizio derivative with fractional-order. We therefore replace the derivatives in the model (5) under consideration with a fractional derivative to maintain the dimension of both sides of the equations of the proposed model taking the κ power of each variable.

7.1. Preliminaries

Definition 7.1.1 (*See Ref. 26*)**.** The Caputo fractional derivative for order $x>0$ is defined in below

$$D_t^x f(t)=\frac{1}{\Gamma(n-x)}\int_a^t (t-\xi)^{n-x-1} f^{(n)}(\xi)d\xi,$$

where $n-1<x\le n,\quad n\in N,\quad f\in C^{n-1}[0,t]$.

Definition 7.1.2 (*See Ref. 26*)**.** The Atangana–Baleanu fractional derivative for a given function for order x in Caputo sense are defined in below

$$D_t^x f(t)=\frac{B(x)}{1-x}\int_a^t \frac{df(\xi)}{d\xi}E_x[-\frac{x}{1-x}(t-\xi)^x]d\xi,$$

where $B(x)=(1-x)+\frac{x}{\Gamma(x)}$ is a normalisation function and $E_\alpha(.)$ is the Mittag-Leffler function.

Definition 7.1.3 (*See Ref. 26*)**.** Atangana–Baleanu fractional integral order x is defined by

$$I_t^x(f(t))=\frac{1-x}{B(x)}f(t)+\frac{x}{B(x)\Gamma(x)}\int_a^t f(\xi)(t-\xi)^{x-1}d\xi,$$

if $f(t)$ is a constant, integral will be resulted with zero.

Definition 7.1.4 (*See Ref. 26*)**.** the Laplace transforms for the Atangana–Baleanu fractional operator of order x, where $0<x\le 1$ is given by

$$LD^x f(t)(s)=\frac{B(x)}{1-x}\frac{S^x Lf(t)(s)-s^{x-1}f(a)}{s^x+\frac{x}{1-x}}.$$

Definition 7.1.5 (*See Ref. 26*)**.** The function f is satisfied the Hölder continuous function if and only if there $C,\upsilon\in\Re^+$ be constants such that $\|f(x)-f(y)\|\le C\|x-y\|^{\upsilon}$.

7.2. Fractional-order model

We utilised the Caputo–Fabrizio (CF) fractional derivatives instead of classical derivatives. In this matter, the model (5) in previous section became the following model of fractional-order type:

$$\begin{aligned}
D^\kappa S(t) &= b(1-\theta I_B)-(\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C)S+\psi V,\\
D^\kappa V(t) &= \epsilon S-(\mu+\psi+\beta_3 I_C)V,\\
D^\kappa I_B(t) &= (b\theta+\beta_1 S-\mu-d_1-r_1)I_B+\sigma pS,\\
D^\kappa I_C(t) &= (\beta_2 S+\beta_3 V-\mu-d_2-r_2)I_C+(1-\sigma)pS.
\end{aligned} \tag{50}$$

Here, $0<\kappa<1$ and D^κ represented to the fractional derivative in the (CF) operator. While, the all parameters still the same meaning such as model (5) as well as the all equilibrium points are the similar in the both models (5) and (50).

8. Numerical simulations

In this section of the present article is devoted to the numerical results of the dynamics of viral hepatitis disease. In this work, four categories of the species are considered; the susceptible population, vaccinated population, viral B hepatitis infected population and viral C hepatitis infected population. These four species are interconnected with each other. Moreover, the impact of embedded parameters is shown on the dynamics of all species. Here, we chose the parameters values range according to several references also the current research takes into account the Caputo sense fractional differential operator as state as in follows $b=100;\theta=1\times10^{-5};\beta_1=1\times10^{-6};\beta_2=1\times10^{-6};\beta_3=1\times10^{-6};\epsilon=0.5;\mu=0.01;p=0.1;\psi=0.2;d_1=2\times10^{-5};d_2=1\times10^{-4};r_1=1\times10^{-6};r_2=0.002;\sigma=0.002$, with initial data (150,750,25,15) (blue curve) and (500,1500,100,4000) (red curve) in Fig. 3. Then, we get $\mathcal{R}_0=1.4>1$, and hence the solution approaches $e_3=(700,1500,16,6400)$.

Next, we consider that $\sigma=1$ then $\mathcal{R}_{0C}=0.8<1<\mathcal{R}_0=1.22$, then the solution approaches $e_2=(650,1550,7700,0)$. This result is shown in Fig. 4.

For $\sigma=0$ we get $\mathcal{R}_{0B}=0.3<1<\mathcal{R}_0=1.22$, and then the solution tends to $e_1=(650,1500,0,6400)$, which agrees with the global stability of e_1 in this case. This result is shown in Fig. 5.

Now, for the same data used with take values of parameters p,β_1 and β_3, so that $p=0,\beta_1=1\times10^{-7}$ and $\beta_3=1\times10^{-7}$ respectively, the trajectories of model (5) approaches to the $e_0=(2900,7000,0,0)$ and $\mathcal{R}_0=0.431<1$, are drawn in Fig. 6. Also, the effect of vaccine and external sources of disease are drawn in Figs. 7–9 respectively. While, the discussion and influence of a fractional-order model we show that by Figs. 10–13.

Finally, the results in Fig. 14 show the relation between the reproduction number $\mathcal{R}_0$ and some parameters such as $\beta_1,\beta_2,\beta_3,\epsilon,\psi,\mu,d_1,d_2$ and r_2. Hence, this relationship has an impact on disease behaviour.

9. Conclusion

In this study, we looked at an epidemiological mathematical model of diseases caused by hepatitis virus types B and C. It is believed that our model considers four groups: the vulnerable population, the immunised population, the population infected with viral B and C hepatitis respectively. These four species are related to one another by some assumption was wrote through four ordinary differential equations. It was our goal to examining how co-transmission as (direct contact, vertically, external sources of disease), vaccination and the different fractional orders affect the dynamics of such a system. The boundedness, uniqueness and positivity of the solution were investigated. It was established that various equilibrium points exist. The topics of local and global stability were covered according to the basic reproduction number. Finally, model (5) was solved, the theoretical conclusion was verified, and the control set of parameters

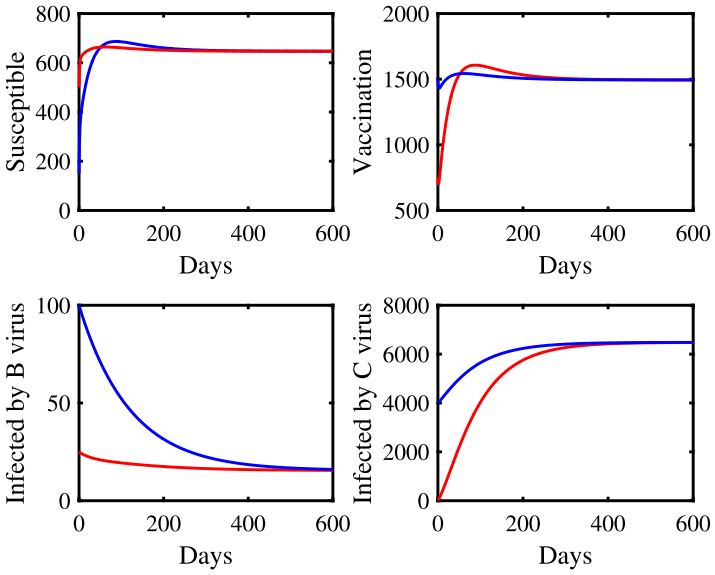

Fig. 3. Graph stability of EE point.

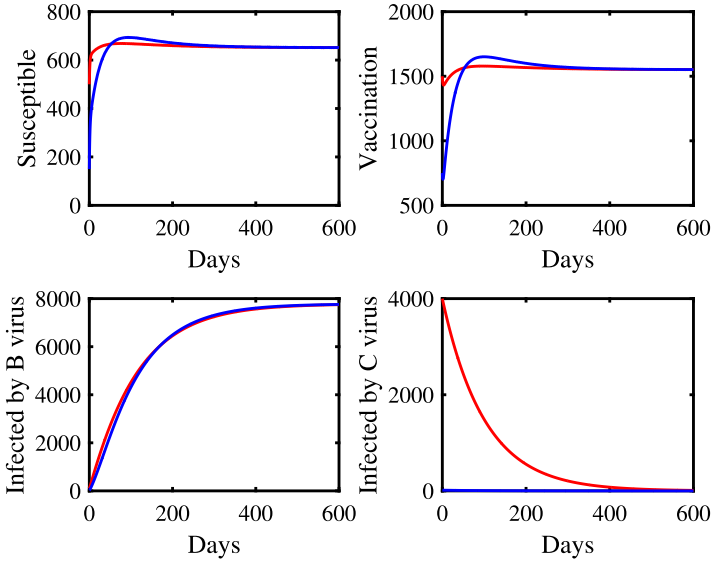

Fig. 4. Graph stability of HCVFE point.

was specified using numerical simulation. The following results were obtained.

Model (5) asymptotically approaches EE point from several sets of initial points if $\mathcal{R}_0 > 1$. Due to $\sigma = 1$, we get the $\mathcal{R}_{0C} < 1$ and the dynamical of model (5) converges to a HCVFE point. While, the value of $\sigma = 0$, we have, the value of $\mathcal{R}_{0B} < 1$ and the dynamical of model (5) converges to a HBVFE point. When, declining contact rates, the EE gradually converges to a UVHE point as the (β_1, β_3, p) rates declining, demonstrating the influence of contact rate on the dynamics of the system. For the vaccination rate of system (5), it was noted that with an increase in vaccine rate, the system dynamics still converge to EE, but the number of infected are decreasing. However, lowering the vaccination rate or increasing the vaccination failure rate then the number of infected are increase.

The numerical results confirm that fractional-order differential equations describe biological systems better and have plentiful dynamics when compared with standard integer-order models. Meanwhile, comparing the different fractional orders, we observe that the viral hepatitis infected solutions converge faster to their equilibrium points as kappa is increased to 1.

Finally, we presented the global sensitivity analysis of the model in order to point out the dominant and the most influential parameters of the model. From these results, it is found that the most crucial parameters are the contact rates $(\beta_1, \beta_2, \beta_3, p)$ and the vaccination rate (ϵ, ψ).

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

The authors confirm that the data supporting the findings of this study are available within the article cited there in.

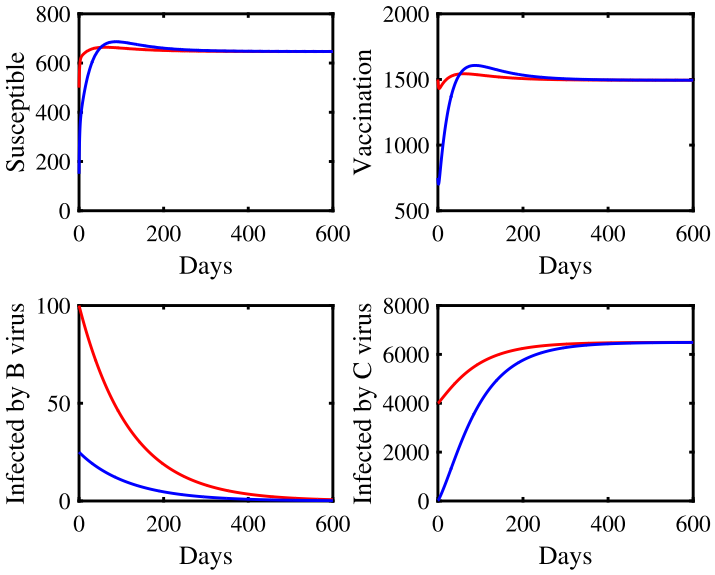

Fig. 5. Graph stability of HBVFE point.

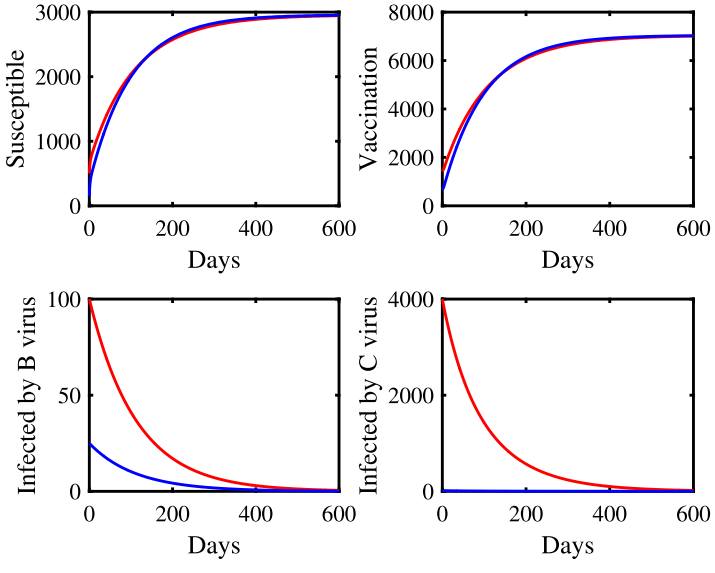

Fig. 6. Graph stability of UVHE point.

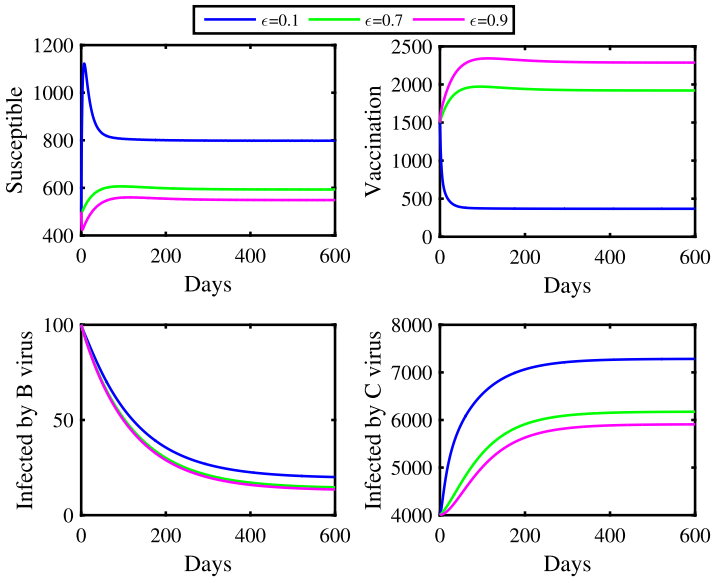

Fig. 7. Effect of vaccination rate on population.

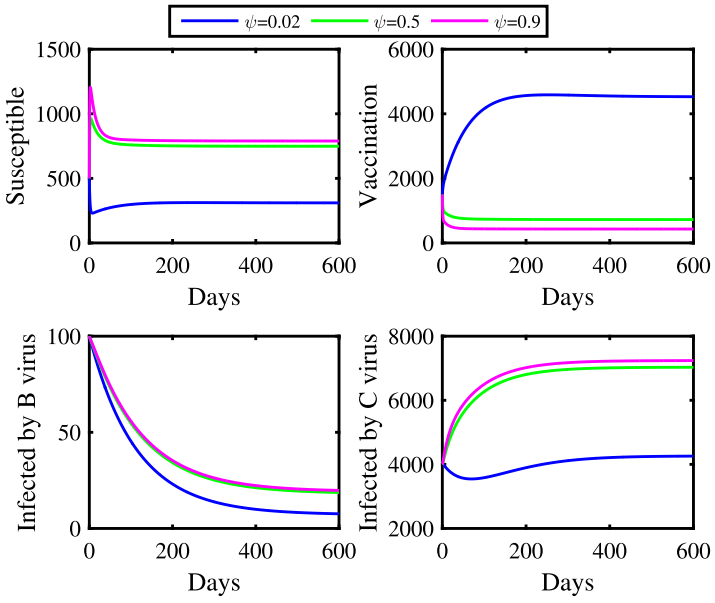

Fig. 8. Effect of failure of vaccine on population.

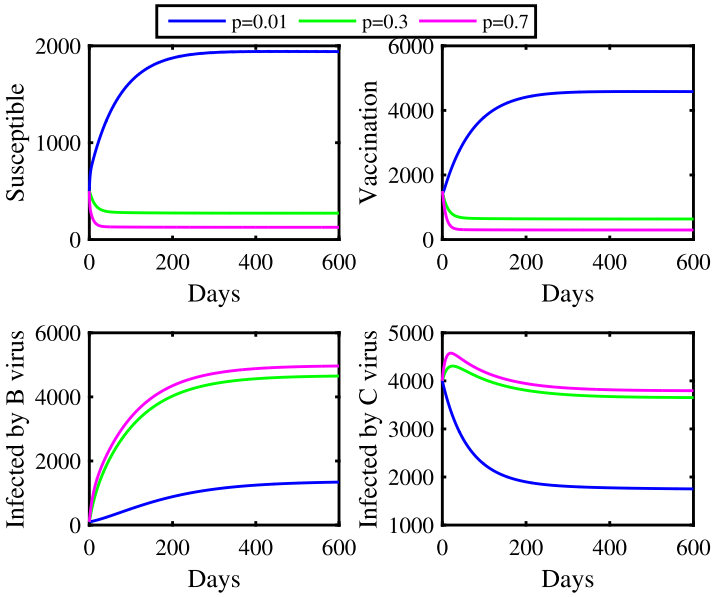

Fig. 9. Effect of external source of infection on population.

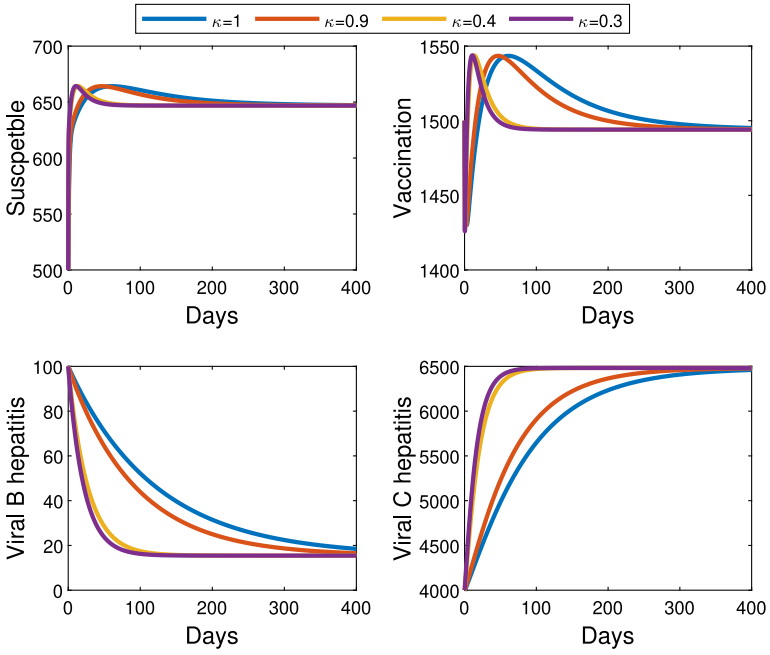

Fig. 10. Effect of fractional-order on EE point with different values of κ.

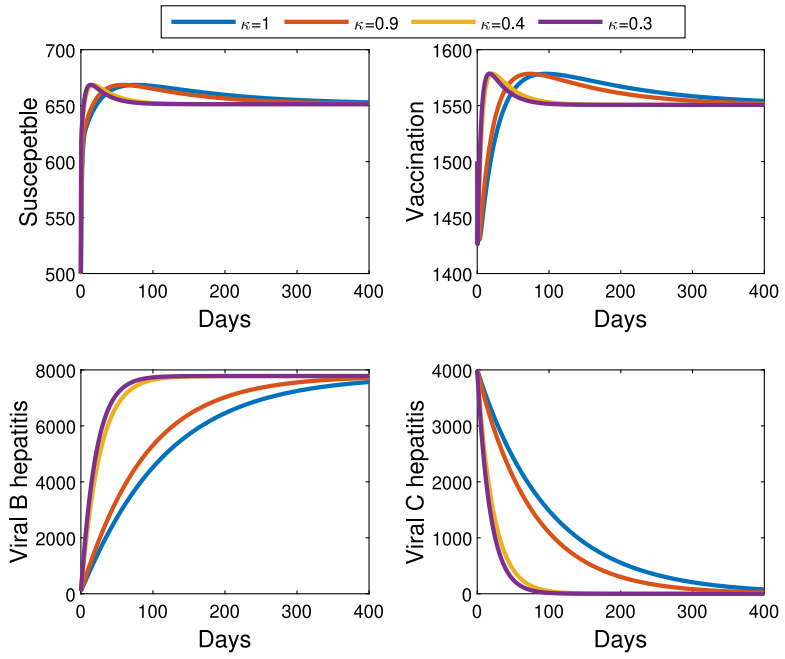

Fig. 11. Effect of fractional-order on HCVFE point with different values of κ.

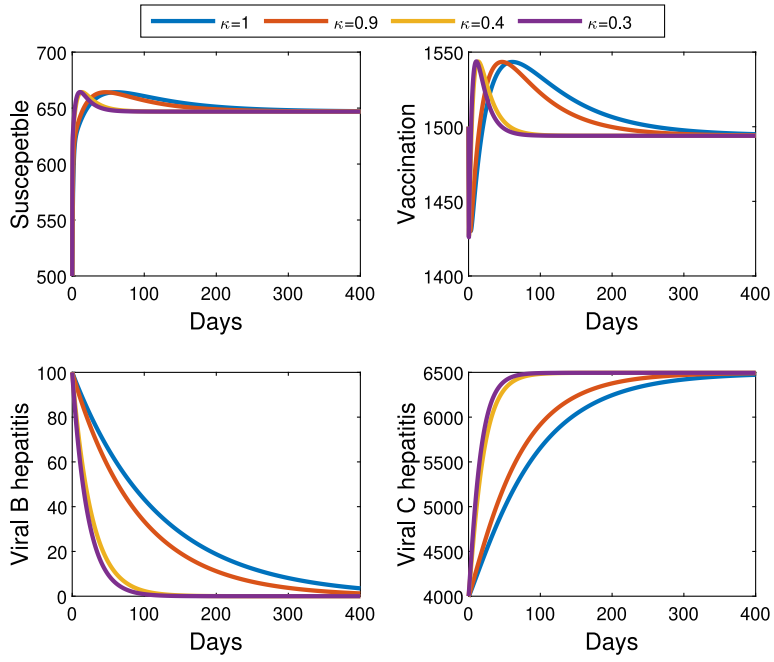

Fig. 12. Effect of fractional-order on HBVFE point with different values of κ.

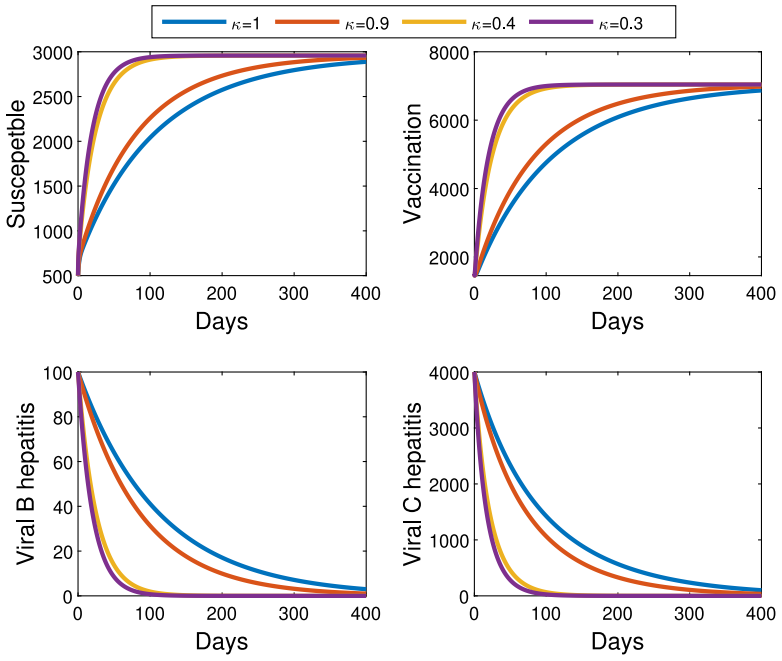

Fig. 13. Effect of fractional-order on UVHE point with different values of κ.

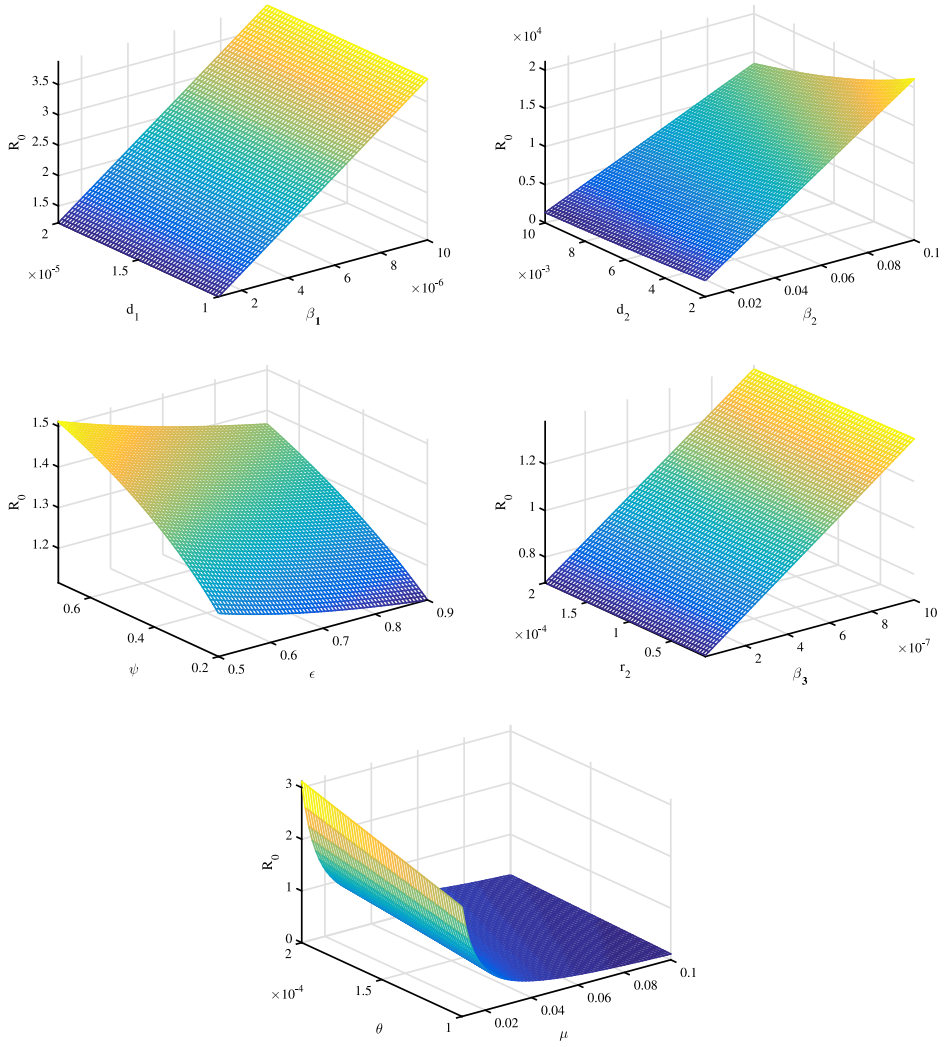

Fig. 14. Effect of some parameters on the reproduction number value.

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