Dynamical Analysis of Transmission of Hepatitis B and C Viruses with External Source of disease by Mathematical Model

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Dynamical Analysis of Transmission of Hepatitis B and C Viruses with External Source of disease by Mathematical Model

The Hepatitis B and C Viruses with External Source of disease

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Abstract In this paper, we develop a deterministic mathematical model of the Hepatitis B and C viruses transmission in population, which allows transmission by two way vertical such as from pregnant mother to fetus and horizontal. Also, by two way through direct contact and due to the external source of infective such as blood transfusion or other. In reality, we know that there is a vaccination against the hepatitis B virus but so far, there is no vaccine against the hepatitis C virus this is why it is considered more dangerous than hepatitis B. Furthermore, we study the vaccination effect with the failure in the vaccine. We propose an SVI_BI_CR model using a system of ordinary differential equations. First the major basic analysis, like the uniqueness, boundedness and positivity of the solution for the proposed model. Second the existence of all biological equilibrium points, basic reproduction number and stability analysis of all equilibrium points. The numerical simulation indicated to confirm the analytic results and the government must apply all control strategies in combating hepatitis virus at short periods of time.

Keywords Epidemic Model · Hepatitis B and C Viruses · Vaccination; External Sources of infection · Vertical and Horizontal transmission · Stability.

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1 Introduction

There are several types of viruses that infect humans and cause many diseases, such as Encephalitis, HPV (Human papilloma virus), HIV, Yellow fever, Impetigo, Poliomyelitis, Chicken pox, Measles, Mumps, HBV (hepatitis B virus), HCV (hepatitis C virus) and lastly COVID-19. In fact, viral hepatitis is considered one of the serious global epidemic diseases caused by HBV. It mainly attacks the liver, it may be a chronic disease or lead to death and sometimes it turns into liver cancer. According to reports from the World Health Organization (WHO) in 2020, about 240 million people in the world are living with chronic liver infection and approximately 0.78 million died. While, in another reports in 2017, the hepatitis affects the lives of 257 million people and death 56000 people every year [1,2].

According to the mechanism of transmission of hepatitis, it is one of the infectious diseases that is transmitted from one person to another, and the most transmissible method is sexual intercourse through semen, blood and vaginal excretion as well as by external sources of infective for example sharing unhygienic needles or razors, transfusion of infected human blood, tattoo and dialysis machines, therefor all above ways to spreading is called horizontal transmissions. Also, this infection transmits from mother to newborn baby at the time of birth but called the vertical transmission [3–5]. Since there is no effective treatment yet, the vaccination that was discovered in 1982, is very important and is necessary today because infection with hepatitis has a high mortality rate. The vaccine protects children from serious infection with the hepatitis and enhances the immune system against infection [6].

There are five primary types of hepatitis, which are A, B, C, D and E. The types A and E come through contamination of food or water, and not turn into chronic hepatitis and are completely cured of them and do not leave traces of the liver. As for the other three B, C and D, they are come through blood and its derivatives, and sexual relations and them turns into chronic liver infections that and leave an impact and damage to the liver.

Some related literature and works regarding the Hepatitis are considered in [7]-[11]. In addition, Gul et al. [12] studied symptomatic carrier effect on the dynamics of hepatitis B virus. Saad et al. [13] studied hepatitis C virus by fractional order modeling. The authors in [14] proposed a new mathematical model for the transmission of HBV due to sexual. Khatun et al. [15] introduced a mathematical modeling of hepatitis B virus with immune responses. In [16], Din et al. formulated a model of hepatitis B epidemic with stochastic analysis and delay effect. Lu et al. [17] studied and analyzed a modeling of hepatitis transmission in China through homosexual and heterosexual. In this paper, we formulate a mathematical model to analyze the infection transmission of hepatitis B and C viruses. Different from previous studies of hepatitis mod-

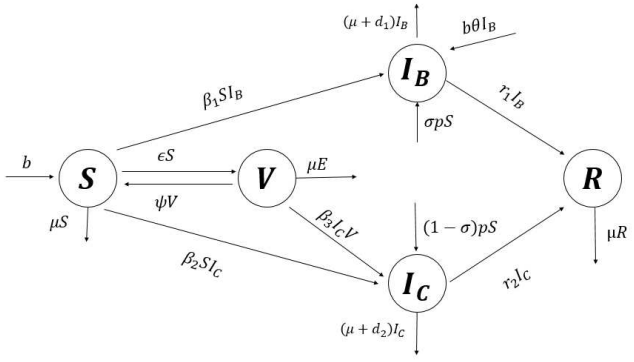

Fig. 1 Diagram of Hepatitis Model.

els, we focus on the vertical transmission of hepatitis B which does not exist in hepatitis C. As well as, we discuss the failure of the vaccination against hepatitis B virus and the risk of not being available against hepatitis C. This work is ordered as follows. The model divided the population into five groups is formulated, and then the basic properties of solutions of the proposed model such as uniqueness, boundedness and positivity are investigated. Also, we calculate the basic reproduction number of the model. Finally, we analyze and study the local and global stability about the all four equilibrium points of the model.

2 Model Formulation

In this section, we present the proposed mathematical model of HBV and HCV infection disease. To create the model, we assume that the total human population $N(t)$ is divided into five epidemiological sub-compartments namely and denoted by susceptible $S(t)$; vaccination $V(t)$; infected by hepatitis B virus $I_B(t)$; infected by hepatitis C virus $I_C(t)$ and recovered $R(t)$ respectively. Such that $N(t) = S(t) + V(t) + I_B(t) + I_C(t) + R(t)$. The transmission dynamics of HBV and HCV is given by the following diagram of model and the nonlinear differential equations.

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

with the initial conditions for model (1) satisfy:

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

In above model, recruitment rate is b and the natural death in all population is μ. The horizontal transmission of disease by contact rates and denoted by $\beta_i, i = 1, 2, 3$. For vertical transmission, we assume that a fraction θ of newborns from infected by hepatitis B virus class are infected and it is denoted by $(0 \leq \theta \leq 1)$. The both hepatitis B and C are also spread by external sources denoted by $p \geq 0$ with fraction $(0 \leq \sigma \leq 1)$. The susceptible class is vaccinated at per capita rate $\epsilon > 0$. The vaccine provides temporary immunity (failure in vaccine), that is denoted by $(0 \leq \psi \leq 1)$. The disease mortality rates in the HBV and HCV are d_1 and d_2, respectively. The HBV and HCV individuals are recovered at the rates r_1 and r_2, respectively.

3 Basic Properties of Solutions

The hepatitis model (1), will be meaningful epidemiologically if the solutions of (1) with non-negative initial conditions (2) will remain non-negative for all time $t > 0$.
In this section, we investigate the existence, uniqueness, boundedness and positivity of the solution in the following theorems.

3.1 Existence and uniqueness

Theorem 3.1 There exists a unique solution of model (1), in a positively invariant set, that remains for all finite time $t \geq 0$.
Proof. The right-hand side of all equations is continuous in the convex domain $E = (t, S(t), V(t), I_B(t), I_C(t), R(t))$ of $(5 + 1)$-dimensional space $\Re_+^{5+1}$, with continuous partial derivatives. So problem (1) has a unique solution in $\Re_+^5$, which exists for a given finite time $t \in [0, +\infty)$ and initial conditions (2).

3.2 Boundedness

Theorem 3.2 All solutions of model (1) which initiate in $\Re_+^5$, are uniformly bounded.
Proof. As the total population size are $N = S + V + I_B + I_C + R$, so from model (1) we get

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

Then $\dot{N} \leq b - \mu N$. Hence, $\mathrm{N(t)} \leq N(0)e^{-\mu t} + \frac{b}{\mu}(1 - e^{-\mu t})$, which shows that

$$N(t) \leq \max\{N(0), \frac{b}{\mu}\}. \tag{4}$$

This implies the the boundedness of solutions.

3.3 Positivity

Theorem 3.3 All solutions $S(t), V(t), I_B(t), I_C(t), R(t)$ of model (1), starting from positive initial conditions (2), remain positive for all finite time $t \geq 0$.

Proof. We have
$\dot{S}\mid_{S=0} = b(1-\theta I_B) + \psi V > 0, \quad forall \quad I_B, V \geq 0,$
$\dot{V}\mid_{V=0} = \epsilon S > 0, \quad forall \quad S > 0,$
$\dot{I_B}\mid_{I_B=0} = \sigma pS \geq 0, \quad forall \quad S > 0,$
$\dot{I_C}\mid_{I_C=0} = (1-\sigma)pS \geq 0, \quad forall \quad S > 0,$
$\dot{R}\mid_{R=0} = r_1 I_B + r_2 I_C \geq 0, \quad forall \quad I_B, I_C \geq 0.$
This leads to the non-negativity of solutions.

On the other hand, since the $5th$ equation of model (1), is independent on the other equations rest, so model (1) we can be reduced to the following system:

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

4 Existence of equilibrium point and basic reproduction number of model (5)

Obviously, model (5) has a disease free equilibrium point when $I_B = I_C = 0$ with $p = 0$ and denoted by $e_0 = (S_0, V_0, 0, 0)$, where

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

Then we define the basic reproduction number $\mathcal{R}_0$ of the model (5) is obtained by using the next generation technique [18], and is given by

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

where

- $\mathcal{R}_{0B} = \frac{b\theta\mu(\mu+\epsilon)(\mu+\epsilon+\psi)+b\beta_1[\mu(\mu+\epsilon+\psi)+\epsilon\psi]}{\mu(\mu+\epsilon)(\mu+\epsilon+\psi)(\mu+d_1+r_1)}$,
  is the basic reproduction number for HBV infected.
- $\mathcal{R}_{0C} = \frac{b\beta_2[\mu(\mu+\epsilon+\psi)+\epsilon\psi]+\beta_3\epsilon b(\mu+\epsilon)}{\mu(\mu+\epsilon)(\mu+\epsilon+\psi)(\mu+d_2+r_2)}$,
  is the basic reproduction number for HCV infected.

Now, model (5) has a hepatitis B virus-free equilibrium point when $I_B = 0$ under the condition $\sigma = 0$ and denoted by $e_1 = (S_1, V_1, 0, I_{C1})$, where.

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

Also, we can calculated the positive value of I_{C1} from the polynomial as follow

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

here
$A_1 = -\beta_2\beta_3(\mu + d_2 + r_2) < 0,$
$A_2 = b\beta_2\beta_3 - (\mu + d_2 + r_2)(\mu\beta_2 + \beta_3(\mu + \epsilon + p) + \psi\beta_2),$
$A_3 = b\beta_3(\epsilon + p) - [\mu(\mu + \epsilon + p) + \psi(\epsilon + p)(\mu + d_2 + r_2)],$
$A_4 = bp\beta_3 > 0.$
Then, we have a unique positive root of I_{C1} in equation (9), if and only if $A_2 < 0$ or $A_3 > 0$.

Clearly, model (5) has a hepatitis C virus-free equilibrium point when $I_C = 0$ under the condition $\sigma = 1$ and denoted by $e_2 = (S_2, V_2, I_{B2}, 0)$, where.

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

It easy see that, the all values of S_2 and V_2 became a positive under the following condition

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

While, we can calculated the positive value of I_{C1} from the polynomial as follow

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

here
$B_1 = -\beta_1(\mu + \psi)(\mu + d_1 + r_1) < 0,$
$B_2 = b[\mu\theta(\mu + \epsilon + p) + \psi\theta(\mu + p) + \beta_1(\mu + \psi)] - bp\theta(\mu + \psi) - (\mu + d_1 + r_1)[\psi(\mu + p) + \mu(\mu + \epsilon + p)],$
$B_3 = bp(\mu + \psi) > 0.$
Then, we have a unique positive root of I_{B2} in equation (12), if and only if $B_2 < 0$ or $B_2 > 0$.

Lastly, model (5) has the endemic equilibrium point and denoted by $e_3 = (S_3, V_3, I_{B3}, I_{C3})$, where.

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

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

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

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

Now, if $I_C \to 0$, we get the isoclines becomes

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

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

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

Then, equation (16) intersects the I-axis at the positive $\widetilde{I_B}$, while equation (17) intersects the I-axis at the positive $\widehat{I_B} = \frac{1}{\theta}$. Clearly, the equations (14) and (15) have a unique positive intersection point and then e_3 exists uniquely, under the following conditions

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

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

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

5 Local Stability Analysis

In the following of this section, we investigate the local stability of model (5) around all equilibrium points using the linearization method. It easy we can calculating the general Jacobian matrix of model (5) about an arbitrary point $e^* = (S, V, I_B, I_C)$ and can be written in below

$$J(e^*) = \begin{pmatrix} -(\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C) & \psi & -(b\theta+\beta_1 S) & -\beta_2 S \\ \epsilon & -(\mu+\psi+\beta_3 I_C) & 0 & -\beta_3 V \\ \beta_1 I_B + \sigma p & 0 & b\theta+\beta_1 S-(\mu+d_1+r_1) & 0 \\ \beta_2 I_C + (1-\sigma)p & \beta_3 I_C & 0 & \beta_2 S+\beta_3 V-(\mu+d_2+r_2) \end{pmatrix}. \tag{21}$$

Now, rewrite the equation (21) around a disease free equilibrium point e_0 has the following result

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

Then, the characteristic polynomial of matrix (22) 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
$T_0 = 2\mu + \epsilon + \psi > 0,$
$D_0 = \mu(\mu + \psi + \epsilon) > 0.$

Consequently, equation (23) has following roots, which represent the eigenvalues of matrix (22)

$$\begin{cases} \lambda_S = -(\mu + \epsilon + \psi), \\ \lambda_V = -\mu, \\ \lambda_{I_B} = b\theta + \beta_1 S_0 - (\mu + d_1 + r_1), \\ \lambda_{I_C} = \beta_2 S_0 + \beta_3 V_0 - (\mu + d_2 + r_2). \end{cases}$$

Clearly, λ_S and λ_V have always negative real parts, while λ_{I_B} and λ_{I_C} are negative real parts when $\mathcal{R}_0 < 1$. Hence, e_0 is locally asymptotically stable. However, it is a saddle point otherwise.

Again, rewrite the equation (21) but around a hepatitis B virus-free equilibrium point e_1 has 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

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

Then, the characteristic polynomial of matrix (24) is

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

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 of $b\theta + \beta_1 S_1 - (\mu + d_1 + r_1)$ in equation (25) can has negative real part if $m_{33} < 0$. The remaining eigenvalues can be obtained from the cubic equation (25). Clearly, if it satisfies the *Routh − Hurtwiz* criterion conditions that $M_i > 0$ for $i = 1, 3$ and $\Delta = M_1M_2 - M_3 > 0$. So, $M_i > 0$ for $i = 1, 3$ with $\Delta > 0$ can be easily confirm 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 locally asymptotically stable.

Now, the equation (21) around a hepatitis C virus-free equilibrium point e_2 can be written as following

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

Here

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

Then the characteristic polynomial of matrix (26) is

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

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

Clearly, in equation (27) one of the eigenvalues is $\beta_2 S_2 + \beta_3 V_2 - (\mu + d_2 + r_2)$ has negative real part if $w_{44} < 0$. The other eigenvalues can be find from the cubic equation (27). Clearly, if it satisfies the $Routh - Hurtwiz$ criterion conditions that $W_i > 0$ for $i = 1, 3$ and $\Delta = W_1W_2 - W_3 > 0$. So, $W_i > 0$ for $i = 1, 3$ with $\Delta > 0$ can be easily confirm 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 locally asymptotically stable.

Finally, about endemic equilibrium point e_3 the equation (21) becomes below

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

Here

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

Obviously, according to $Gersgorin$ theorem [19] if the condition $\mid q_{ii} \mid > \sum_{i=1, i\neq j}^{4} \mid q_{ij} \mid$, holds. Then the all eigenvalues of matrix (28) can be obtained in the left half plane and exists in the region $\Lambda = \cup\{\upsilon^* \in \Theta :\mid \upsilon^* - q_{ii} \mid < \sum_{i=1, i\neq j}^{4} \mid q_{ij} \mid\}$, where Λ represents the union of all the discs centered at q_{ii} with radius q_{ij}. Therefore, since $q_{ii} < 0$ for all $i = 1, 2, 3, 4$ under the condition of the Gersgorin theorem with all the conditions $\mu + d_1 + r_1 > 2(b\theta + \beta_1 S_3)$ and $\mu + d_2 + r_2 > 2(\beta_2 S_3 + \beta_3 V_3)$, it is easy to confirm that all the eigenvalues of matrix (28) have a negative real parts and then e_3 is locally asymptotically stable. Otherwise it is saddle point.

6 Global Stability Analysis

Now, we investigate the global stability of all equilibrium points of the our model (5). First, we prove that of the disease free point e_0 by using Castillo-Chavez method for inform see [20]. The remaining three points can be prove the global stability with help of LaSalle through define the *Lyapunov* function for inform see [21].

Theorem 6.1: If $\mathcal{R}_0 < 1$, the e_0 of proposed model (5) is globally asymptotically stable.

Proof. Let $Y = (S, V)$ be the uninfected individuals and $Z = (I_B, I_C)$ represent to the HBV and HCV infected and $e_0 = (S_0, V_0, 0, 0)$ represents to the disease free equilibrium point. We have

$\frac{dX}{dt}$= 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 $K(Y, 0) = 0$, it becomes

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

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

BZ-$\bar{G}(Y, Z) = \begin{pmatrix} b\theta + \beta_1 S_0 - (\mu + d_1 + r_1) & 0 \\ 0 & \beta_2 S_0 + \beta_3 V_0 - (\mu + d_2 + r_2) \end{pmatrix} \cdot \begin{pmatrix} I_B \\ I_V \end{pmatrix}$

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

Such that

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

$Z = \begin{pmatrix} I_B \\ I_V \end{pmatrix}$

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

Theorem 6.2: If $\mathcal{R}_{0B} \leq 1 < \mathcal{R}_0$, the e_1 of proposed model (5) is global asymptotically stable.

Proof. To confirm the result, we choose the *Lyapunov* function as follows

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

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

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

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

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

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

Clearly, by substituting equations (31)-(34) in to equation (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 equation (35),

$$[1 - \frac{I_C}{I_{C1}} + \frac{SI_C^2}{S_1 I_{C1}^2}(1 - \frac{I_{C1}}{I_C})] \leq 0,$$

$$[1 - \frac{S}{S_1} + \frac{\theta b}{\beta_1 S}(1 - \frac{S}{S_1})] \leq 0,$$

$$[1-\frac{V}{V_1}+\frac{VS}{V_1S_1}(1-\frac{SV_1}{S_1V})]\leq 0,$$
$$[1-\frac{V_1}{V}+\frac{I_C}{I_{C1}}(1-\frac{V}{V_1})]\leq 0,$$
$$[b\theta+\beta_1 S-\mu-d_1-r_1]I_B\leq 0.$$

Hence, the largest invariant subset $\frac{dL_1}{dt}=0$, is e_1. Then, according the result of *Lasalle* that HBV free of model (5) is globally asymptotically stable, for $\mathcal{R}_{0B}\leq 1<\mathcal{R}_0$ and it becomes unstable for $\mathcal{R}_{0B}>1$.

Theorem 6.3: If $\mathcal{R}_{0C}\leq 1<\mathcal{R}_0$, the e_2 of proposed model (5) is global asymptotically stable.

Proof. To confirm the result, we choose the *Lyapunov* function as follows

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

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

$$\begin{aligned}(1-\tfrac{S_2}{S})\tfrac{dS}{dt}&=(1-\tfrac{S_2}{S})[b(1-\theta I_B)-(\mu+\epsilon+b+\beta_1 I_B+\beta_2 I_C)S+\psi V]\\&=(1-\tfrac{S_2}{S})[-\theta bI_B-(\mu+\epsilon+p+\beta_1 I_B+\beta_2 I_C)S+\psi V+\theta bI_{B2}+(\mu+\epsilon+p+\beta_1 I_{B2})S_2-\psi V_2]\\&=\beta_1 S_2 I_{B2}[1-\tfrac{S_2}{S}-\tfrac{SI_B}{S_2I_{B2}}+\tfrac{I_B}{I_{B2}}]+(\mu+\epsilon+p)[1-\tfrac{S_2}{S}-\tfrac{S}{S_2}+1]\\&\quad+\tfrac{\psi V_2S_2}{S}[1-\tfrac{V}{V_2}-\tfrac{S}{S_2}+\tfrac{VS}{V_2S_2}]+b\theta I_{B2}[1-\tfrac{S_2}{S}-\tfrac{I_B}{I_{B2}}+\tfrac{S_2B}{SI_{B2}}]\\&\quad+\beta_2 I_C S_2[1-\tfrac{S}{S_2}],\end{aligned} \tag{38}$$

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

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

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

Now, by substituting equations (38)-(41) in to equation (37), we get

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

Obviously, in equation (42),

$$[1-\frac{S}{S_2}+\frac{VS}{V_2S_2}(1-\frac{S_2}{S})]\leq 0,$$

$$[1-\frac{I_B}{I_{B2}}+\frac{\sigma SI_B}{S_2I_{B2}}(1-\frac{I_{B2}}{I_B})]\leq 0,$$

$$[1-\frac{V}{V_2}]\leq 0,$$

$$[\beta_2 S+\beta_3 V-\mu-d_2-r_2]I_C\leq 0.$$

Thus, the largest invariant subset $\frac{dL_2}{dt}=0$, is e_2. Then, according the result of *Lasalle* that HCV free of model (5) is globally asymptotically stable, for $\mathcal{R}_{0C}\leq 1<\mathcal{R}_0$ and it becomes unstable for $\mathcal{R}_{0C}>1$.

Theorem 6.4: If $\mathcal{R}_0>1$, the e_3 of proposed model (5) is global asymptotically stable.

Proof. To confirm the result, we choose the *Lyapunov* function as follows

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

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

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

$$(1-\frac{V_3}{V})\frac{dV}{dt} = \beta_3 V_3 I_{C3}[1-\frac{V_3}{V}-\frac{VI_C}{V_3 I_{C3}}+\frac{I_C}{I_{C3}}]+(\mu+\psi)V_3[1-\frac{V_3}{V}-\frac{V}{V_3}+1] \\ +\frac{\epsilon S_3 V_3}{V}[1-\frac{S}{S_3}-\frac{V}{V_3}+\frac{SV}{S_3 V_3}], \tag{46}$$

$$(1-\frac{I_{B3}}{I_B})\frac{dI_B}{dt} = \frac{\beta_1 S_3 I_{B3}^2}{I_B}[1-\frac{I_B}{I_{B3}}-\frac{SI_B}{S_3 I_{B3}}+\frac{SI_B^2}{S_3 I_{B3}^2}]+(\mu+d_1+r_1)I_{B3}[1-\frac{I_{B3}}{I_B}-\frac{I_B}{I_{B3}}+1] \\ +\frac{b\theta I_{B3}^2}{I_B}[1-\frac{I_B}{I_{B3}}-\frac{I_B}{I_{B3}}+\frac{I_B^2}{I_{B3}^2}]+\frac{p\sigma S_3 I_{B3}}{I_B}[1-\frac{S}{S_3}-\frac{I_B}{I_{B3}}+\frac{SI_B}{S_3 I_{B3}}], \tag{47}$$

$$(1-\frac{I_{C3}}{I_C})\frac{dI_C}{dt} = \frac{\beta_2 S_3 I_{C3}^2}{I_C}[1-\frac{I_C}{I_{C3}}-\frac{SI_C}{S_3 I_{C3}}+\frac{SI_C^2}{S_3 I_{C3}^2}]+\frac{\beta_3 V_3 I_{C3}^2}{I_C}[1-\frac{I_C}{I_{C3}}-\frac{VI_C}{V_3 I_{C3}}+\frac{VI_C^2}{V_3 I_{C3}^2}] \\ +(\mu+d_2+r_2)I_{C3}[1-\frac{I_{C3}}{I_C}-\frac{I_C}{I_{C3}}+1]+\frac{(1-\sigma)pS_3 I_{C3}}{I_C}[1-\frac{S}{S_3}-\frac{I_C}{I_{C3}}+\frac{SI_C}{S_3 I_{C3}}]. \tag{48}$$

Now, by substituting equations (45)-(48) in to equation (44), we get

$$\frac{dL_3}{dt} = \beta_1 S_3 I_{B3} I_C I_B[1-\frac{S_3}{S}+\frac{I_B}{I_{B3}}(1-\frac{S}{S_3})]+\beta_2 S_3 I_{C3}[1-\frac{S_3}{S}+\frac{I_C}{I_{C3}}(1-\frac{S}{S_3})] \\ +\beta_3 V_3 I_{C3}[1-\frac{V_3}{V}+\frac{I_C}{I_{C3}}(1-\frac{V}{V_3})]+\frac{\psi S_3 V_3}{S}[1-\frac{V}{V_3}+\frac{SV}{S_3 V_3}(1-\frac{V_3}{V})] \\ +\frac{\epsilon S_3 V_3}{V}[1-\frac{V}{V_3}+\frac{SV}{S_3 V_3}(1-\frac{V_3}{V})]+(\mu+\epsilon+p)S_3[2-\frac{S_3}{S}-\frac{S}{S_3}] \\ +(\mu+\psi)V_3[2-\frac{V_3}{V}-\frac{V}{V_3}]+(\mu+d_1+r_1)I_{B3}[2-\frac{I_{B3}}{I_B}-\frac{I_B}{I_{B3}}] \\ +(\mu+d_2+r_2)I_{C3}[2-\frac{I_{C3}}{I_B}-\frac{I_B}{I_{B3}}]+\frac{b\theta S_3 I_B}{S}[1-\frac{I_{B3}}{I_B}+\frac{SI_{B3}}{S_3 I_B}(1-\frac{I_B}{I_{B3}})] \\ +\frac{b\theta I_{B3}^2}{I_B}[1-\frac{I_B}{I_{B3}}+\frac{I_B^2}{I_{B3}^2}(1-\frac{I_B}{I_{B3}})]+\frac{\sigma p S_3 I_{B3}}{I_B}[1-\frac{I_B}{I_{B3}}+\frac{SI_B}{S_3 I_{B3}}(1-\frac{I_{B3}}{I_B})] \\ +\frac{(1-\sigma)pS_3 I_{C3}}{I_C}[1-\frac{I_C}{I_{C3}}+\frac{SI_C}{S_3 I_{C3}}(1-\frac{I_{C3}}{I_C})]+\frac{\beta_1 S_3 I_{B3}^2}{I_B}[1-\frac{I_B}{I_{B3}}+\frac{SI_B^2}{S_3 I_{B3}^2}(1-\frac{I_{B3}}{I_B})] \\ +\frac{\beta_2 S_3 I_{C3}^2}{I_C}[1-\frac{I_C}{I_{C3}}+\frac{SI_C^2}{S_3 I_{C3}^2}(1-\frac{I_{C3}}{I_C})]+\frac{\beta_3 V_3 I_{C3}^2}{I_C}[1-\frac{I_C}{I_{C3}}+\frac{VI_C^2}{V_3 I_{C3}^2}(1-\frac{I_{C3}}{I_C})]. \tag{49}$$

Now, in equation (49) if the following

$$[1-\frac{S_3}{S}+\frac{I_B}{I_{B3}}(1-\frac{S}{S_3})]\leq 0$$

$$[1-\frac{I_B}{I_{B3}}+\frac{SI_B^2}{S_3 I_{B3}^2}(1-\frac{I_{B3}}{I_B})]\leq 0$$

$$[1-\frac{I_C}{I_{C3}}+\frac{VI_C^2}{V_3 I_{C3}^2}(1-\frac{I_{C3}}{I_C})]\leq 0$$

$$[1-\frac{V}{V_3}+\frac{SV}{S_3 V_3}(1-\frac{V_3}{V})]\leq 0$$

Then, the largest invariant subset $\frac{dL_3}{dt}=0$, is e_3. Then, according the result of *Lasalle* that endemic equilibrium point of model (5) is globally asymptotically stable, for $\mathcal{R}_0>1$ and it becomes unstable for $\mathcal{R}_0<1$.

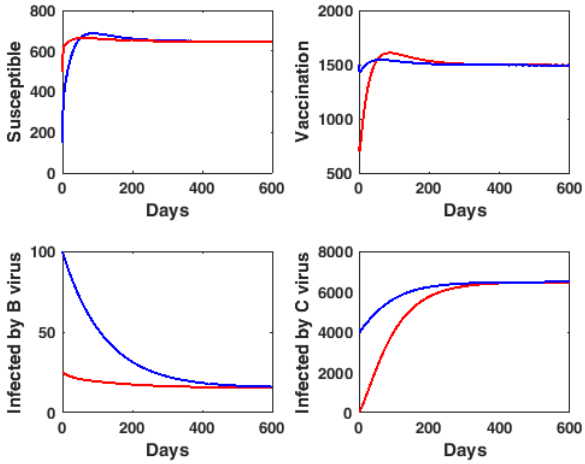

Fig. 2 The trajectory of model (5) approaches to globally asymptotically stable of endemic equilibrium point.

7 Numerical Simulation

To understand the influence of parameters and confirm the previous analytical results and compare, so we should investigate that through numerical simulation with choosing realistic values for parameters in this section. It is well known that the model parameters affect the dynamical behavior of the model greatly. This occurs due to either error in their measurement or missing the parameter values. Hence, the impact of varying the model's parameters on the behavior of the solution of the model is important. Therefore, the parameters that are having vital roles in the dynamics of the model will be given clear attention to understand their effects on the control of the spread of disease. We take the parameters values range adopt to some references as state in follows $b = 100; \theta = 1 \times 10^{-5}; \beta_1 = 1 \times 10^{-6}; \beta_2 = 1 \times 10^{-6}; \beta_3 = 1 \times 10^{-6}; \epsilon = 0.5; \mu = 0.01; p = 0.1; \psi = 0.2; d_1 = 2 \times 10^{-5}; d_2 = 1 \times 10^{-4}; r_1 = 1 \times 10^{-6}; r_2 = 0.002; \sigma = 0.002$, with time period to one year as well as two initial points (150,750,25,15) is (blue line) and (500,1500,100,4000) (red line). We show that the dynamic behavior of the model (5) approaches to the $e_3 = (700, 1500, 16, 6400)$ and $\mathcal{R}_0 = 1.4 > 1$, see Figure(2).

However, for the same data used with take value of parameter σ, so that $\sigma = 1$ we have, the trajectories of model (5) approaches to the $e_2 = (650, 1550, 7700, 0)$ and $\mathcal{R}_{0c} = 0.8 < 1 < \mathcal{R}_0 = 1.22$, are drawn in figure (3).

On the other hand, for the same data used with take value of parameter σ, so that $\sigma = 0$ we have, the trajectories of model (5) approaches to the $e_1 = (650, 1500, 0, 6400)$ and $\mathcal{R}_{0b} = 0.3 < 1 < \mathcal{R}_0 = 1.22$, are drawn in figure (4).

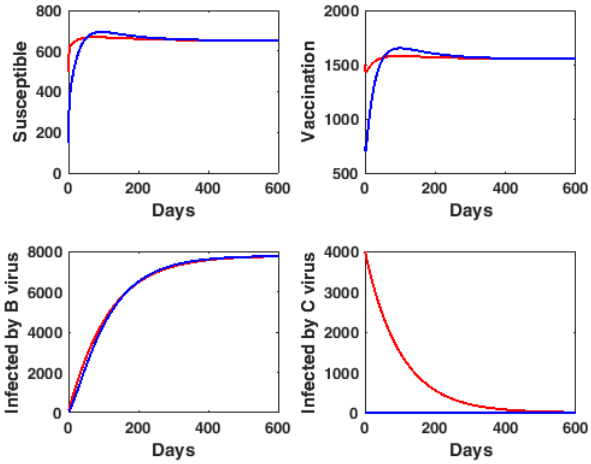

Fig. 3 The trajectory of model (5) approaches to globally asymptotically stable of hepatitis C virus-free equilibrium point.

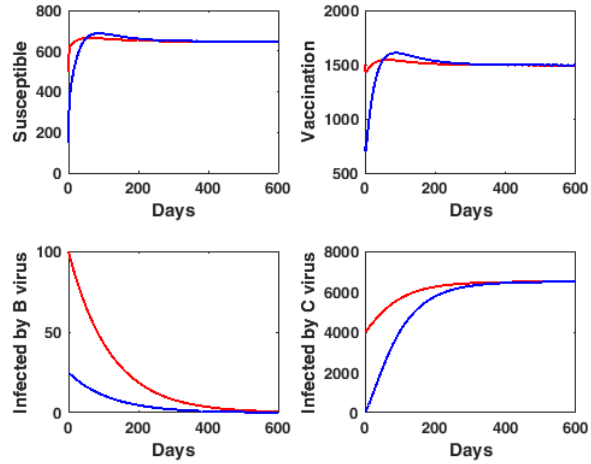

Fig. 4 The trajectory of model (5) approaches to globally asymptotically stable of hepatitis B virus-free equilibrium point.

Now, for the same data used with take values of parameters p, β_1 and β_3, so that $p = 0, \beta_1 = 1 \times 10^{-7}$ and $\beta_3 = 1 \times 10^{-7}$ 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 figure (5).

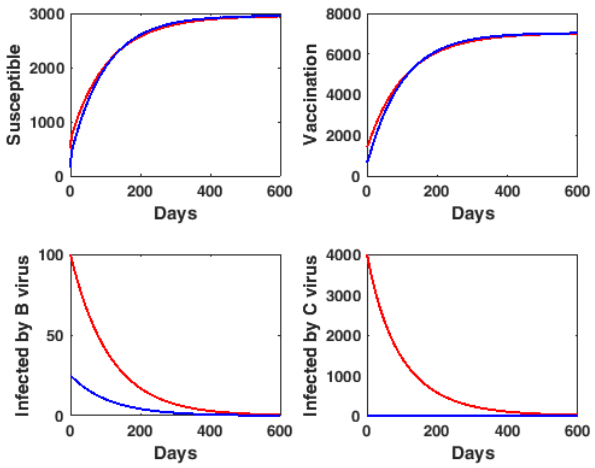

Fig. 5 The trajectory of model (5) approaches to globally asymptotically stable of disease-free equilibrium point.

8 Conclusion

In this article, we have introduced a new mathematical model of hepatitis B and C viruses, that outbreak by into two modes: vertical (the transmission from mothers to babies) and horizontal due to the direct contact between the susceptible individuals with infected individuals. As well as, through the external sources of infection. The incidence of infection is described by linear function. According to the results in some studies the hepatitis disease incidence rate has decreased significantly and steadily in children because of the intensive and strong immunization campaign. Also, one of the goals of the World Health Organization in the year 2030 is to reduce the rate of injuries to 90% and deaths to 65%. Therefore, we focused on the importance of vaccination against the disease and the need for efficiency of the vaccine. The proposed model has four equilibrium points namely disease-free equilibrium, hepatitis B virus-free equilibrium point, hepatitis C virus-free equilibrium point and endemic equilibrium. The stability analysis of the model shows that the disease-free equilibrium is globally asymptotically stable if the basic reproduction number $\mathcal{R}_0 < 1$, the condition of hepatitis B virus-free equilibrium point becomes global stability under the basic reproduction number for hepatitis B virus $\mathcal{R}_{0B} < 1$, the dynamical behavior of proposed model converges to the hepatitis C virus-free equilibrium point if the basic reproduction number for hepatitis C virus $\mathcal{R}_{0C} < 1$. The stability analysis of the model shows that the endemic equilibrium is globally asymptotically stable if the basic reproduction number is $\mathcal{R}_0 > 1$.

Conflict of interest

The authors declare that they have no conflicts of interest.

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