Natural and Applied Sciences International Journal (NASIJ)

https://doi.org/10.47264/idea.nasij/2.1.6

Vol.2, No.1,(January-December 2021), 70-79

https://www.ideapublishers.org/index.php/nasij

Research Article

THE ANALYSIS OF HEPATITIS B VIRUS (HBV) TRANSMISSION USING AN EPIDEMIC MODEL

TAHIR KHAN[1], RAHMAN ULLAH[1†], GUL ZAMAN[2], IMTIAZ AHMAD[2]

1. DEPARTMENT OF MATHEMATICS AND STATISTICS, WOMAN UNIVERSITY SAWABI, KHYBER PAKHTUNKHWA,
2. DEPARTMENT OF MATHEMATICS, UNIVERSITY OF MALAKAND, CHAKDARA, DIR (LOWER), KHYBER PAKHTUNKHAWA, PAKISTAN

Email address: rahman_yzi@yahoo.com

Published Date: 17 December 2021

ABSTRACT. In this article, we propose an epidemic problem of hepatitis B with vaccination. So to do this, first we presents the model formulation and prove that the solutions are bounded and positive. We obtain the disease free equilibrium and calculate *the basic reproduction number* (R_0). The *reproductive number* will be used to find the endemic state of the model. We discuss the qualitative analysis of the proposed problem and show that whenever, $R_0 < 1$ then the disease free equilibrium is stable locally and globally. Moreover, whenever, $R_0 > 1$, then the endemic state is asymptotically stable. We derive sufficient conditions for both the equilibria and its stabilities. Further more numerical simulation are carried out to illustrate the feasibility of the obtained results and verified that with actual data, we are in the position to put down the hepatitis B infection form the community. We also highlight the role of epidemic parameters in the disease propagation. Our numerical works verified the analytical results. Finally some important conclusion are given at the end of the article.

1. INTRODUCTION

The infection of hepatitis B causes liver disease. Mostly hepatitis caused by a virus, bacterial infections or continuous exposure to drugs or alcohol [1]. This infection B has multiple phases: acute and chronic. The first one refers to the initial 200 days after some one exposed to the virus. The immune system usually able to vanish the virus in acute phase, and so the recovery within some months is possible. But for some one if the infection remains it leads to the chronic position. This stage of hepatitis B refers to the illness occurs if the hepatitis B virus remain in the body and with the passage of time, the infection develop serious health complications. Most of the time often there has no history of acute illness for a person with chronic illness. This actually produce scarring of liver and become the reason of liver cancer and failure. There are multiple routes of spreading in the case of hepatitis B, in which semen, blood, and vaginal secretions etc., are significant [2, 3, 4]. Also this virus can be transmitted from mother to child at abortion time. Hepatitis B virus also transferred from sexual contact and razers sharing [5]. The mode of transmission are similar for both the HBV and HIV, however the HBV virus is fifty to hundreds time more infectious. According to World Health Organization (WHO), the infection of hepatitis B is a major and sever health problem all over the world. Because 350 to 400 million people are infected world wide with this contagious infection. Only in China there are 93 million population have been suffered due to hepatitis B virus infections [6, 7]. Every year almost ten thousands people catch this disease by passing. Since this is a major health issue

2010 *Mathematics Subject Classification.* 65M06, 65M12, 65T60.

Key words and phrases. Mathematical model, hepatitis B virus, *basic reproduction number*, stabilities, simulation.

around the world and so for its prevalence high priority strategies are discovered [8]. Effective vaccine are available with 95 percent effective antibodies [9].

The mathematical modeling of different infectious disease has a rich field and a numbers of research articles have been studied by various authors (see for detail, [10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 22, 23, 24, 25, 26, 27]). Similarly modeling of HBV has also a rich literature and different mathematician and biologists investigated various epidemiological models to study and forecast the future spread of the disease. Especially in the last some decade the field of epidemiological models are frequently used. A mathematical model has been used by Medley for minimizing the HBV in Newzeland [28, 29]. Andreson et al., used a simple model to describe the carries effect on the transmission of HBV [30]. Moreover, a model has been proposed by Zhao at al [31] to discuss the prediction of HBV using an age structured model.

In this work, we establish a HBV model. To do this, we split the total population according to the characteristic and so into five epidemiological groups of, $S(t)$, the susceptible, $E(t)$, the exposed, $A(t)$, the acute, $B(t)$, the chronic and $R(t)$, the recovered/removed individuals. Once we formulate the model, we prove the positivity and bounded-ness. The equilibria and the *basic reproduction number* of the proposed model are calculated to show the stabilities. We prove the local as well as global dynamics of the model. Linearization along with Herwitz-criteria are used to discuss the local dynamics, while for the global dynamics the classic Lyapnov function theory. Finally we discuss the numerical findings and trying to verify all the analytical works numerically. We also presents some sensitive analysis of important parameters.

2. Mathematical model and its analysis

We formulate a mathematical model for the HBV transmission incorporating the exposed group and dividing the infected class in two group of $A(t)$ and $B(t)$, which shows respectively the acute and the chronic stages. We use $N(t)$ to symbolize the total population and consequently divide into five groups. In which the susceptible, $S(t)$ are those individuals who have a chance to got the infection, while the exposed $E(t)$ represents those who are not infectious, and the infected $A(t)$ represent those individuals which are infective with acute hepatitis B. Moreover, the infected $B(t)$ are those individual, which are infected with chronic hepatitis and recovered/removed $R(t)$ represents the recovered with permanent immunity. Thus the compartmental mathematical model is represented analytically by the following nonlinear system of five differentials equations:

$$\begin{aligned}
\frac{dS(t)}{dt} =& \Pi - \alpha S(t)B(t) - (v+\mu_0)S(t),\\
\frac{dE(t)}{dt} =& \alpha S(t)B(t) - (\mu_0+\beta)E(t),\\
\frac{dA(t)}{dt} =& \beta E(t) - (\mu_0+\gamma_1+\gamma)A(t),\\
\frac{dB(t)}{dt} =& \gamma A(t) - (\mu_0+\gamma_2+\mu_1)B(t),\\
\frac{dR(t)}{dt} =& vS(t) + \gamma_1 A(t) + \gamma_2 B(t) - \mu_0 R(t),
\end{aligned} \tag{2.1}$$

with

$$S(0) > 0,\ E(0) \geq 0,\ A(0) \geq 0,\ B(0) \geq 0,\ R(0) > 0. \tag{2.2}$$

In Eq.(2.1), Π represent the recruitment rate (assumed susceptible), α is the disease progression rate from susceptible to exposed, β is the progression rate from exposed class acute, γ is the rate at which infected with acute hepatitis B individuals moves to the chronic group. γ_1 and γ_2 are the recovery rates of acute and chronic groups respectively. μ_0 is the natural death rate and μ_1 the rate of deaths occur from the infection. The vaccination rate is v.

Let $N(t)$ represents the sum of all epidemiological groups then $N = S + E + A + B + R$. So the initial sizes and the considered problem, make sure that $N(t) \geq 0$, hence the total population $N(t)$ is bounded and remain positive for $t > 0$. Thus, we have the following result.

Proposition 1. *Let $X = (S, E, A, B, R)$ be the solution of the problem as stated by model (2.1), then X is positive and bounded.*

Proof. The differentiability of the right-hand side of the proposed model (2.1) implies the existence of a unique maximal solution for any associated Cauchy problem, then the 1st equation solution of problem (2.1) looks like

$$\begin{aligned} S(t) = S(0)\exp\left[-\left\{(v+\mu_0)t + \int_0^t \alpha B(x)dx\right\}\right] \\ + \exp\left[-\left\{(v+\mu_0)t + \int_0^t B(x)dx\right\}\right]\int_0^t \Pi \exp\left[\left\{-(v+\mu_0)y + \int_0^l B(u)du\right\}\right]dy > 0. \end{aligned} \tag{2.3}$$

Similarly, the solution of the 2nd equation of the problem as stated by Eqn.(2.1) implies that

$$A(t) = A(0)\exp\left[-\{(\beta+\mu_0)t\}\right] + \exp\{-(\beta+\mu_0)t\}\int_0^t \alpha B(x)dx \exp\{-(\beta+\mu_0)\}ydy \geq 0. \tag{2.4}$$

Furthermore, the solution of the 3rd, 4th and 5th equations of the proposed model implies that $A(t), B(t) \geq 0$ and $R(t) > 0$, which shows that the solution (X) of system (2.1) is positive.

Now to show that the solution is bounded we differentiate $N(t)$ and using model (2.1), then $\frac{dN}{dt} + \mu_0 N = \Pi - \mu_1 B$ implies that $\frac{dN}{dt} + \mu_0 N \leq \Pi$. The integration of both sides and applying the differential inequality by following [32], we obtain $0 < N(S, E, A, B, R) \leq \frac{\Pi}{\mu_0} + ce^{-\mu_0 t}$. Letting $t \to \infty$, then $0 < N(S, E, A, B, R) \leq \frac{\Pi}{\mu_0}$, which prove that the solutions of the problem (2.1) are bounded and confined in the region given by

$$\Omega = \left\{(S, E, A, B, R) \in R_+^5 : N = \frac{\Pi}{\mu_0} + \xi\right\} \tag{2.5}$$

for any $\xi > 0$ and for $t \to \infty$. □

3. Steady state analysis

We study the temporal dynamical behavior of the problem (2.1), so the right hand side is equating to zero of all the equations. Direct calculation give us, that the model (2.1) has always a disease free equilibrium, let us denoted by F_0 and given by $F_0 = (S_0, E_0, A_0, B_0, R^0)$, where $S_0 = \frac{\Pi}{v+\mu_0}$, $R^0 = \frac{v\Pi}{\mu_0(v+\mu_0)}$ and $E_0 = A_0 = B_0$.

In order to use the disease-free state we figure out the *basic reproductive number*. Because in epidemiological models this quantity is an important role. To find it we follow [33], therefore assuming

$\chi = (E(t), A(t), B(t))$ so from Eqn.(2.1), we may write

$$\frac{d\chi}{dt} = \bar{F} - \bar{V},$$

where the matrices are define as

$$\bar{F} = \begin{bmatrix} \alpha S(t)B(t) \\ 0 \\ 0 \end{bmatrix}, \ \bar{V} = \begin{bmatrix} (\mu_0 + \beta)E(t) \\ (\gamma + \gamma_1 + \mu_0)A(t) - \beta E(t) \\ (\mu_0 + \gamma_2 + \mu_1)B(t) - \gamma A(t) \end{bmatrix}.$$

Taking the linearization of $\bar{F}$ and $\bar{V}$ at F_0 we get

$$F = \begin{bmatrix} 0 & 0 & \alpha S_0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}, \ V = \begin{bmatrix} q_2 & 0 & 0 \\ -\beta & q_3 & 0 \\ 0 & -\gamma_1 & q_4 \end{bmatrix},$$

where $q_2 = \mu_0 + \beta$, $q_3 = \mu_0 + \gamma + \gamma_1$ and $q_4 = \mu_0 + \mu_1 + \gamma_2$. Thus the *basic reproduction number* (R_0) is defined to be $R_0 = \rho(\bar{K}) = \rho(FV^{-1})$, which becomes

$$R_0 = \frac{\alpha S_0 \beta \gamma_1}{q_2 q_3 q_4}. \tag{3.1}$$

Similarly to find the endemic equilibrium, solving Eqn.(2.1) simultaneously for S, E, A, B and R at steady states respectively, thus we will obtain the unique positive endemic equilibrium denoted by F_* and define as $F_* = (S^*, E^*, A^*, B^*, R^*)$ where

$$\begin{aligned} S^* &= \frac{1}{\alpha\beta\gamma} q_2 q_4, \ E^* = \frac{1}{\alpha\beta\gamma} q_4 q_1 \{R_0 - 1\}, \ A^* = \frac{1}{\alpha\gamma} \frac{q_1 q_4}{q_3} \{R_0 - 1\}, \\ B^* &= \frac{1}{\alpha} q_1 \{R_0 - 1\}, \ R^* = \frac{1}{\mu_0} \{\gamma_1 A^* + \gamma_2 B^* + v S^*\}, \end{aligned} \tag{3.2}$$

and $q_1 = \mu_0 + v$. Obviously, if R_0 is less then one, than the endemic equilibrium dose not exists, but if $R_0 > 1$, the unique endemic positive states exists.

We now study the asymptotic stability of our proposed model at both disease free and endemic equilibrium. We use the Jacobian and the classic Lyapunov function theory to perform the analysis. Regarding the stability analysis, we prove the following.

Theorem 1. *The disease-free state (F_0) of the model (2.1) is asymptotically stable locally and globally whenever $R_0 < 1$, while unstable if $R_0 > 1$.*

Proof. Linearizing Eqn.(2.1) at F_0, we obtain a Jacobian matrix (J_0) is defined by

$$J_0 = \begin{pmatrix} -(\mu_0 + v) & 0 & 0 & \frac{-\alpha b}{\mu_0 + v} & 0 \\ 0 & -(\mu_0 + \beta) & 0 & \frac{\alpha b}{\mu_0 + v} & 0 \\ 0 & \beta & -(\mu_0 + \gamma_1 + \gamma) & 0 & 0 \\ 0 & 0 & \gamma & -(\mu_0 + \gamma_1 + \gamma) & 0 \\ v & 0 & \gamma_1 & \gamma_2 & -\mu_0 \end{pmatrix}. \tag{3.3}$$

The characteristic equation of the matrix J_0 as stated by Eqn.(3.3) looks like

$$\{\lambda + \mu_0\} \{\lambda + (\mu_0 + v)\} \left\{\lambda^3 + a_1\lambda^2 + a_2\lambda + a_3\right\} = 0, \tag{3.4}$$

where

$$
\begin{aligned}
a_1 &= \beta + 3\mu_0 + \mu_1 + \gamma_1 + \gamma_2 + \gamma,\\
a_2 &= 3\mu_0^2 + 2\beta\mu_0 + \beta\mu_1 + \beta\gamma_1 + \beta\gamma_2 + 2\mu_0\mu_1 + \beta\gamma + 2\mu_0\gamma_1 + 2\mu_0\gamma_2 + \mu_1\gamma_1\\
&\quad + 2\mu_0\gamma + \mu_1\gamma + \gamma_1\gamma_2 + \gamma\gamma_2,\\
a_3 &= \mu_0^2\gamma_1 + \mu_0^2\gamma + \beta\mu_0\gamma_1 + \beta\mu_1\gamma_1 + \beta\mu_0\gamma + \beta\mu_1\gamma + \beta\gamma_1\gamma_2 + \mu_0\mu_1\gamma + \beta\gamma\gamma_2\\
&\quad + \mu_0\mu_1\gamma + \mu_0\gamma_1\gamma_2 + \mu_0\gamma\gamma_2 + \mu_0(\mu_0+\beta)(\mu_0+\mu_1+\gamma_2)\{1-R_0\}.
\end{aligned}
$$

There are five roots of the Eqn.(3.4), which shows that matrix J_0 have five corresponding eigenvalues. Two roots are $\lambda_1 = -\mu_0$ and $\lambda_2 = -(\mu_0 + v)$ and are negative. The rest of the roots are obtained from the below equation

$$\psi(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3. \tag{3.5}$$

Clearly roots of the above equation are negative or negative real parts, if the Routh-Hewritz criteria holds (for detail see [34]). So $a_3 > 0$ and $a_1 > 0$, and $a_3 < a_1a_2$ if and only if the value of R_0 is less then one, which shows that if $R_0 < 1$, then roots will be negative. But on the other hand whenever $R_0 > 1$ the above equation roots will be negative and positive, which shows that F_0 is unstable saddle point.

Now for global dynamics the problem (2.1) at F_0, we construct the Lyapunov function given by

$$L(t) = k_1(S - S_0) + k_2E(t) + k_3A(t) + k_4B(t), \tag{3.6}$$

where k_i for $i = 1, 2...4$ are some constant and will be chosen latterly. Calculating the temporal derivative of the above function and then using model (2.1), we obtain

$$
\begin{aligned}
\frac{dL}{dt} &= k_1(\Pi - \alpha S(t)B(t) - q_1S(t)) + k_2(\alpha S(t)B(t) - q_2E(t)) + k_3(\beta E(t) - q_3A(t))\\
&\quad + k_4(\gamma A(t) - q_4B(t)).
\end{aligned}
$$

Using $k_1 = k_2 = q_3q_4$, $k_3 = k_4 = \alpha S_0\gamma_1$ and $S_0 = \frac{\Pi}{\mu_0+v}$, so the above equation take the following form

$$
\begin{aligned}
\frac{dL}{dt} &= -q_1q_2q_3(S - S_0) - q_2q_3q_4\left\{1 - \frac{\alpha\beta S_0\gamma_1}{q_2q_3q_4}\right\}E(t) - \alpha\gamma S_0(\mu_0+\gamma_1)A(t) - q_4\alpha\gamma S_0B(t),\\
\frac{dL}{dt} &= -q_1q_2q_3(S - S_0) - q_2q_3q_4\left\{1 - R_0\right\}E(t) - \alpha\gamma S_0(\mu_0+\gamma_1)A(t) - q_4\alpha\gamma S_0B(t).
\end{aligned}
$$

Thus $\frac{dL}{dt} < 0$ and $\frac{dL}{dt} = 0$ if and only if $S = S_0$, $E = E_0$, $A = A_0$ and $B = B_0$. Following the LaSalle invariant principle [35], therefore the disease free equilibrium $F_0 = (S_0, 0, 0, 0)$ is globally stable. □

Theorem 2. *The endemic equilibrium state F_* of the model (2.1) is locally and globally asymptotically stable, if the following condition are satisfied,*

1. $R_0 > 1$
2. $\alpha\beta\Pi\gamma q_3q_4 > q_1q_2$
3. $q_4 > 1$

and unstable otherwise.

Proof. Linearizing the model (2.1) about F_* gives a Jacobian matrix symbolized by J_1, such that

$$J_1 = \begin{pmatrix} -\frac{\alpha\beta\Pi\gamma}{q_2q_3} & 0 & 0 & -\frac{q_2q_3}{\beta\gamma} & 0 \\ \frac{\alpha\beta\Pi\gamma}{q_2q_3} - q_1 & -q_2 & 0 & \frac{q_2q_3}{\beta\gamma} & 0 \\ 0 & \beta & -q_4 & 0 & 0 \\ 0 & 0 & \gamma & -q_3 & 0 \\ v & 0 & \gamma_1 & \gamma_2 & -\mu_0 \end{pmatrix}, \tag{3.7}$$

where

$$q_1 = v + \mu_0,\ q_2 = \beta + \mu_0,\ q_3 = \gamma_2 + \mu_1 + \mu_0,\ q_4 = \gamma_1 + \gamma + \mu_0.$$

Now we find the characteristic equation of the Jacobian matrix (J_1), which looks like

$$\{\lambda + \mu_0\}\{\lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4\} = 0, \tag{3.8}$$

where

$$\begin{aligned} b_1 &= q_2 + q_3 + q_4 + \alpha\beta\Pi\gamma\frac{q_3}{q_2}, \\ b_2 &= (\alpha\beta\Pi\gamma + q_2 + q_4)q_3 + q_2\gamma_2q_4 + \alpha\beta\Pi\gamma\frac{q_4^2}{q_2} + \alpha\beta\Pi\gamma\frac{q_3q_4}{q_2}, \\ b_3 &= \alpha\beta\Pi\gamma q_3^2 + \alpha\beta b\gamma q_2q_3 + q_4 - q_2q_3 + \alpha\beta\Pi\gamma\frac{q_3^2q_4}{q_2}, \\ b_4 &= \alpha\beta\Pi\gamma q_3^2q_4 - q_1q_2q_3. \end{aligned}$$

It is clear from Eqn.(3.8) that there are five corresponding eigenvalues of J_1. One of them is $\lambda_1 = -\mu_0$ is negative, while the rest four roots can be obtained by solving the equation given below

$$\xi(\lambda) = \lambda^4 + b_1\lambda^3 + b_2\lambda^2 + b_3\lambda + b_4. \tag{3.9}$$

Following the Routh-Hewritz criteria [34] these eigenvalues are negative, if $b_i > 0$ for $i = 1, \ldots, 4$, and $b_1b_2b_3 > b_4$. So for $R_0 > 1$, $\alpha\beta b\gamma q_3q_4 > q_1q_2$ and $q_4 > 1$, we obtain $b_i > 0$ for $i = 1, \ldots, 4$, and $b_3^2 + b_1^2b_4 < b_1b_2b_3$. Thus eigenvalues of J_1 are negative if and only if the conditions stated above form 1 to 3 are satisfied.

To establish the global dynamics at $F_* = (S^*, E^*, A^*, B^*, R^*)$, we construct the Laypnavo function given by

$$V(t) = \frac{1}{2}\left\{(S - S^*) + (E - E^*) + (A - A^*) + (B - B^*)\right\}^2. \tag{3.10}$$

Differentiating and using Eqn.(2.1) in the derived result, we get

$$\begin{aligned} \frac{dV}{dt} &= (S - S^*) + (E - E^*) + (A - A^*) + (B - B^*)\big\{\Pi - (v + \mu_0)S \\ &\quad - \mu_0E - (\gamma_1 + \mu_0)A - (\mu_0 + \gamma_2 + \mu_1)B\big\}. \end{aligned}$$

Now using the endemic equilibrium, the above equation can be written as

$$\begin{aligned} \frac{dV}{dt} &= (S - S^*) + (E - E^*) + (A - A^*) + (B - B^*)\big\{(\mu_0 + \beta)E^* \\ &\quad + \frac{1}{\alpha\beta\gamma}(\mu_0 + \beta)(\mu_0 + v)(\mu_0 + \mu_1 + \gamma_2) - (\mu_0 + v)S - \mu_0E \\ &\quad - (\mu_0 + \gamma_1)A - (\mu_0 + \mu_1 + \gamma_2)B\big\}. \end{aligned}$$

Simplification with little re-arrangement of the above equation yields

$$\begin{aligned}\frac{dV}{dt} &= \{(E-E^*)+(S-S^*)+(B-B^*)+(A-A^*)\}\left\{\mu_0(E^*-E)\right.\\ &+\frac{1}{\alpha\gamma}(\mu_0+\mu_1+\gamma_2)(\mu_0+v)+(\mu_0+\beta)(S^*-S)-\mu_0 E\\ &\left.-(\mu_0+\gamma_1)A-(\mu_0+\mu_1+\gamma_2)B\right\},\end{aligned}$$

implies that

$$\begin{aligned}\frac{dV}{dt} &= -\{(S-S^*)+(A-A^*)+(E-E^*)+(B-B^*)\}\left\{\mu_0(E-E^*)\right.\\ &\left.+(\mu_0+\beta)(S-S^*)+(\mu_0+\gamma_1)A+(\gamma_2+\mu_0+\mu_1)(B-B^*)\right\}.\end{aligned}$$

Hence $\frac{dV}{dt} < 0$ whenever $R_0 > 1$ and consequently $\frac{dV}{dt} = 0$ if and only if $S = S^*$, $E = E^*$, $A = A^*$, $B = B^*$, $R = R^*$. Therefore, the Lassales invariance principle implies that F_* is stable globally asymptotically. □

4. Numerical simulations

We demonstrate the numerical simulations of the developed model (2.1). This verify the analytical findings via utilizing the numerical analysis due to the complexity of the analytical solution. We chose the parameters value with biologically feasibility. Also it is very easy to isolate the behaviour/effect of the interaction between the different compartments, which is one of the significant advantage of the numerical analysis. We assume the set of parameters $A_1 = \{\Pi, \alpha, \beta, \mu_0, \mu_1, \gamma, \gamma_1, \gamma_2, v\}$. In which some parameters value are are taken from the literature and some are assumed. The values of the parameter in A_1 are as follows:

$$\Pi = 10, \alpha = 0.4, \beta = 0.05, \mu_0 = .2, \mu_1 = 0.02, \gamma = 0.23, \gamma_1 = 0.8, \gamma_2 = 0.2, v = 0.98.$$

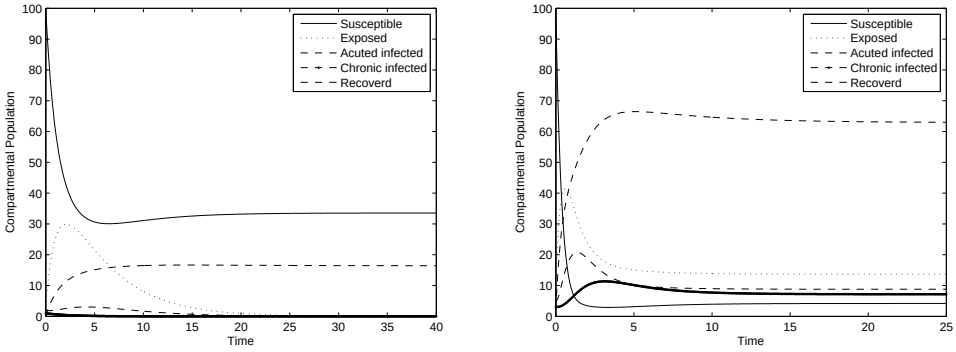
For the parameter set A_1 the problem (2.1) has only a disease-free state and it is stable see Fig.1a. The value of α and β are chosen in such a way that $R_0 < 1$. Moreover, again for another set of parameter $A_2 = \{\Pi, \alpha, \beta, \mu_0, \mu_1, \gamma, \gamma_1, \gamma_2, v\}$, whose all values are equal except α, β, μ_1 and γ. In this case all the listed parameters values would be definitely greater then the above, so the values of the parameters in A_2 are as follows:

$$\Pi = 10, \alpha = 0.5, \beta = 0.9, \mu_0 = 0.2, \mu_1 = 0.09, \gamma = 0.4, \gamma_1 = 0.8, \gamma_2 = 0.2, v = 0.98.$$

For the parameter set A_2 the proposed model (2.1) has two equilibria, disease free, endemic equilibrium and the endemic equilibrium is stable locally asymptotically see Fig.1b. Furthermore at the parameter set A_2, $R_0 > 1$ and the condition at Theorem.4 that is $\alpha\beta\Pi\gamma q_3 q_4 > q_1 q_2$, $q_4 > 1$ satisfied, which ensure the verification of analytical result at Theorem.4.

5. Conclusion

In this work, we developed an epidemic problem for the dynamics of HBV. Therefore we divided the host population into five groups of susceptible, exposed, infected with acute hepatitis, infected with chronic hepatitis and the recovered, then formulated the model with this new features. After formulating the model, we proved the bounded-ness of the solutions of the proposed problem and find R_0. We find the steady states



(A) Solution curves at disease free state (B) Solution curves at endemic state

FIGURE 1. The plot visualizes the solution curves at the disease-free and endemic states with respective set of parameters A_1 and A_2.

i.e., the infected and uninfected steady states. The disease free equilibrium (F_0) is locally stable whenever $R_0 < 1$, while the endemic state (F_*) is locally stable if $R_0 > 1$. Moreover, all the flows along the axes of susceptible and recovered are always attractor to the DFE (F_0), but the flow along the axes of the infected compartment that is exposed, infected with acute hepatitis B (A) and the infected with chronic Hepatitis B B depend on the value of R_0. So, if $R_0 < 1$ then the axes of infected compartment E, A and B are attractor towards the DFE (F_0). But when the *basic reproduction number* acroses one that is $R_0 > 1$, the axes of these compartment do not attract the DFE (F_0) and repels from it. Furthermore for global stabilities we established the Lyapunov function and proved that the dynamics of the considered problem are investigated by R_0 completely. Finally given the graphical visualizations to the analytical results to verify the results. We believe that this assumption, extension and the new analysis are plausible biologically and mathematically.

REFERENCES

[1] Alter, M. J., Evatt, B. L., Margolis, H. S., Biswas, R., Epstein, J. S., Feinstone, S. M., ... & Hoofnagel, J. H. (1991). Public health service interagency guidelines for screening donors of blood, plasma, organs, tissues, and semen for evidence of hepatitis B and hepatitis C. American Journal of Infection Control, 19(5), 32A-41A.

[2] Lavanchy, D. (2004). Hepatitis B virus epidemiology, disease burden, treatment, and current and emerging prevention and control measures. Journal of viral hepatitis, 11(2), 97-107.

[3] Lok, A. S., Heathcote, E. J., & Hoofnagle, J. H. (2001). Management of hepatitis B: 2000—summary of a workshop. Gastroenterology, 120(7), 1828-1853.

[4] McMahon, B. J. (2005, February). Epidemiology and natural history of hepatitis B. In Seminars in liver disease (Vol. 25, No. S 1, pp. 3-8). Published in 2005 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA.

[5] Chang, M. H. (2007, June). Hepatitis B virus infection. In Seminars in fetal and neonatal medicine (Vol. 12, No. 3, pp. 160-167). WB Saunders.

[6] Libbus, M. K., & Phillips, L. M. (2009). Public health management of perinatal hepatitis B virus. Public health nursing, 26(4), 353-361.

[7] Williams, R. (2006). Global challenges in liver disease. Hepatology, 44(3), 521-526.

[8] Maynard, J. E., Kane, M. A., & Hadler, S. C. (1989). Global control of hepatitis B through vaccination: role of hepatitis B vaccine in the Expanded Programme on Immunization. Clinical Infectious Diseases, 11(Supplement$_3$) S574-S578.

[9] Shepard, C. W., Simard, E. P., Finelli, L., Fiore, A. E., & Bell, B. P. (2006). Hepatitis B virus infection: epidemiology and vaccination. Epidemiologic reviews, 28(1), 112-125.
[10] Zaman, G., Kang, Y. H., & Jung, I. H. (2008). Stability analysis and optimal vaccination of an SIR epidemic model. BioSystems, 93(3), 240-249.
[11] Zaman, G., Kang, Y. H., & Jung, I. H. (2009). Optimal treatment of an SIR epidemic model with time delay. BioSystems, 98(1), 43-50.
[12] Li, X. P., Gul, N., Khan, M. A., Bilal, R., Ali, A., Alshahrani, M. Y., ... & Islam, S. (2021). A new Hepatitis B model in light of asymptomatic carriers and vaccination study through Atangana–Baleanu derivative. Results in Physics, 29, 104603.
[13] Li, X. P., Wang, Y., Khan, M. A., Alshahrani, M. Y., & Muhammad, T. (2021). A dynamical study of SARS-COV-2: A study of third wave. Results in Physics, 29, 104705.
[14] Li, X. P., Al Bayatti, H., Din, A., & Zeb, A. (2021). A vigorous study of fractional order COVID-19 model via ABC derivatives. Results in Physics, 29, 104737.
[15] Li, Y. X., Rauf, A., Naeem, M., Binyamin, M. A., & Aslam, A. (2021). Valency-based topological properties of linear hexagonal chain and hammer-like benzenoid. Complexity, 2021.
[16] Li, Y. X., Muhammad, T., Bilal, M., Khan, M. A., Ahmadian, A., & Pansera, B. A. (2021). Fractional simulation for Darcy-Forchheimer hybrid nanoliquid flow with partial slip over a spinning disk. Alexandria Engineering Journal, 60(5), 4787-4796.
[17] Xiong, P. Y., Khan, M. I., Gowda, R. P., Kumar, R. N., Prasannakumara, B. C., & Chu, Y. M. (2021). Comparative analysis of (Zinc ferrite, Nickel Zinc ferrite) hybrid nanofluids slip flow with entropy generation. Modern Physics Letters B, 2150342.
[18] Li, Y. X., Alshbool, M. H., Lv, Y. P., Khan, I., Khan, M. R., & Issakhov, A. (2021). Heat and mass transfer in MHD Williamson nanofluid flow over an exponentially porous stretching surface. Case Studies in Thermal Engineering, 26, 100975.
[19] Xiong, P. Y., Hamid, A., Chu, Y. M., Khan, M. I., Gowda, R. P., Kumar, R. N., ... & Qayyum, S. (2021). Dynamics of multiple solutions of Darcy–Forchheimer saturated flow of Cross nanofluid by a vertical thin needle point. The European Physical Journal Plus, 136(3), 1-22.
[20] Xiong, P. Y., Almarashi, A., Dhahad, H. A., Alawee, W. H., Issakhov, A., & Chu, Y. M. (2021). Nanoparticles for phase change process of water utilizing FEM. Journal of Molecular Liquids, 334, 116096.
[21] Chen, S. B., Jahanshahi, H., Abba, O. A., Solís-Pérez, J. E., Bekiros, S., Gómez-Aguilar, J. F., ... & Chu, Y. M. (2020). The effect of market confidence on a financial system from the perspective of fractional calculus: Numerical investigation and circuit realization. Chaos, Solitons & Fractals, 140, 110223.
[22] Din, A., & Li, Y. (2021). Stationary distribution extinction and optimal control for the stochastic hepatitis B epidemic model with partial immunity. Physica Scripta, 96(7), 074005.
[23] Din, A., LI, Y., Khan, F. M., Khan, Z. U., & Liu, P. (2021). On Analysis of fractional order mathematical model of Hepatitis B using Atangana–Baleanu Caputo (ABC) derivative. Fractals, 2240017.
[24] Din, A., & Li, Y. (2021). Lévy noise impact on a stochastic hepatitis B epidemic model under real statistical data and its fractal–fractional Atangana–Baleanu order model. Physica Scripta, 96(12), 124008.
[25] Khan, T., Zaman, G., & Chohan, M. I. (2017). The transmission dynamic and optimal control of acute and chronic hepatitis B. Journal of biological dynamics, 11(1), 172-189.
[26] Khan, T., Khan, A., & Zaman, G. (2018). The extinction and persistence of the stochastic hepatitis B epidemic model. Chaos, Solitons & Fractals, 108, 123-128.
[27] Khan, T., Ullah, Z., Ali, N., & Zaman, G. (2019). Modeling and control of the hepatitis B virus spreading using an epidemic model. Chaos, Solitons & Fractals, 124, 1-9.
[28] Mann, J., & Roberts, M. (2011). Modelling the epidemiology of hepatitis B in New Zealand. Journal of theoretical biology, 269(1), 266-272.
[29] Thornley, S., Bullen, C., & Roberts, M. (2008). Hepatitis B in a high prevalence New Zealand population: a mathematical model applied to infection control policy. Journal of Theoretical Biology, 254(3), 599-603.
[30] Anderson, R. M., & May, R. M. (1992). Infectious diseases of humans: dynamics and control. Oxford university press.
[31] Zhao, S., Xu, Z., & Lu, Y. (2000). A mathematical model of hepatitis B virus transmission and its application for vaccination strategy in China. International journal of epidemiology, 29(4), 744-752.
[32] Birkhoff, G., & Rota, G. C. (1982). Ordinary Differential Equation, Ginn. and Co., Boston,.[18] May RM (2001). Stability and Complexity in Model Ecosystems.
[33] Lashari, A. A., & Zaman, G. (2011). Global dynamics of vector-borne diseases with horizontal transmission in host population. Computers & Mathematics with Applications, 61(4), 745-754.

[34] Allen, L. J. (2007). An introduction to mathematical biology. Upper Saddle River, New Jersey.
[35] Astolfi, A. (2009). Stability of Dynamical Systems-Continuous, Discontinuous, and Discrete Systems (by Michel, AN et al.; 2008)[Bookshelf]. IEEE Control Systems Magazine, 29(1), 126-127.