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

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ABSTRACT. In this article, we propose an epidemic problem of hepatitis B with vaccination. So to do this,
first we present 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 razors 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
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:

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

with

$$S(0) > 0, \quad E(0) \geq 0, \quad A(0) \geq 0, \quad B(0) \geq 0, \quad R(0) > 0.$$  

(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. γ₁ and γ₂ are the recovery rates of acute and chronic groups respectively. μ₀ is the natural death rate and μ₁ the rate of deaths occur from the infection. The vaccination rate is ν.

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

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

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

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

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

\[
\Omega = \left\{ (S, E, A, B, R) \in R^5_+ : N = \frac{\Pi}{\mu_0} + \xi \right\}
\]

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 \( S_0 = \frac{\Pi}{v+\mu_0} \), \( R_0 = \frac{\mu_1}{\mu_0(v+\mu_0)} \) 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
\[
\frac{d\chi}{dt} = F - V,
\]
where the matrices are defined as
\[
F = \begin{bmatrix}
\alpha S(t)B(t) \\
0 \\
0
\end{bmatrix}, \quad V = \begin{bmatrix}
(\mu_0 + \beta)E(t) \\
(\gamma + \gamma_1 + \mu_0)A(t) - \beta E(t) \\
(\mu_0 + \gamma_2 + \mu_1)B(t) - \gamma A(t)
\end{bmatrix}.
\]
Taking the linearization of \( F \) and \( V \) at \( F_0 \) we get
\[
F = \begin{bmatrix}
0 & 0 & \alpha S_0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{bmatrix}, \quad V = \begin{bmatrix}
q_2 & 0 & 0 \\
-\beta & q_3 & 0 \\
0 & -\gamma_1 & q_4
\end{bmatrix},
\]
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 \( R_0 = \rho(K) = \rho(FV^{-1}) \), which becomes
\[
R_0 = \frac{\alpha S_0\beta \gamma_1}{q_2 q_3 q_4}.
\] (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
\[
S^* = \frac{1}{\alpha \beta \gamma} q_2 q_4, \quad E^* = \frac{1}{\alpha \beta \gamma} q_4 q_1 \{R_0 - 1\}, \quad A^* = \frac{1}{\alpha \gamma} q_4 \{R_0 - 1\}, \\
B^* = \frac{1}{\alpha} q_1 \{R_0 - 1\}, \quad R^* = \frac{1}{\mu_0} \{\gamma_1 A^* + \gamma_2 B^* + vS^*\},
\] (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
\[
J_0 = \begin{bmatrix}
-(\mu_0 + v) & 0 & 0 & -\frac{\alpha b}{\mu_0 + v} & 0 \\
0 & -(\mu_0 + \beta) & 0 & \frac{\alpha b}{\mu_0 + v} & 0 \\
0 & -\beta & -(\mu_0 + \gamma_1 + \gamma) & 0 & 0 \\
0 & 0 & \gamma & -(\mu_0 + \gamma_1 + \gamma) & 0 \\
v & 0 & \gamma_1 & \gamma_2 & -\mu_0
\end{bmatrix}.
\] (3.3)
The characteristic equation of the matrix \( J_0 \) as stated by Eqn.(3.3) looks like
\[
\lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 = 0,
\] (3.4)
where
\[
a_1 = \beta + 3\mu_0 + \mu_1 + \gamma_1 + \gamma_2 + \gamma,
\]
\[
a_2 = 3\mu_0^2 + 2\beta\mu_0 + \beta\mu_1 + \beta\gamma_1 + \beta\gamma_2 + 2\mu_0\mu_1 + \beta\gamma + 2\mu_0\gamma_1 + 2\mu_0\gamma_2 + \mu_1\gamma_1 + 2\mu_0\gamma + \mu_1\gamma + \gamma_1\gamma_2 + \gamma\gamma_2,
\]
\[
a_3 = \mu_0^2\gamma_1 + \mu_0^2\gamma + \beta\mu_0\gamma_1 + \beta\mu_0\gamma + \beta\mu_1\gamma + \beta\gamma_1\gamma_2 + \mu_0\mu_1\gamma + \beta\gamma\gamma_2 + \mu_0\mu_1\gamma + \mu_0\gamma_1\gamma_2 + \mu_0\gamma_2 + \mu_0(\mu_0 + \beta)(\mu_0 + \mu_1 + \gamma_2)\{1 - R_0\}.
\]

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 \(\lambda_2 = -(\mu_0 + \nu)\) and are negative. The rest of the roots are obtained from the below equation
\[
\psi(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3.
\] (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 than 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
\[
L(t) = k_1(S - S_0) + k_2E(t) + k_3A(t) + k_4B(t),
\] (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
\[
\frac{dL}{dt} = k_1(\Pi - \alpha S(t)B(t) - q_1S(t)) + k_2(\alpha S(t)B(t) - q_2E(t)) + k_3(\beta E(t) - q_3A(t)) + k_4(\gamma A(t) - q_4B(t)).
\]

Using \(k_1 = k_2 = q_3q_4\), \(k_3 = k_4 = \alpha S_0\gamma_1\) and \(S_0 = \frac{\Pi}{\mu_0 + \nu}\), so the above equation take the following form
\[
\frac{dL}{dt} = -q_1q_2q_3(S - S_0) - q_2q_3q_4\left\{1 - \frac{\alpha\beta S_0\gamma_1}{q_2q_3q_4}\right\} E(t) - \alpha\gamma S_0(\mu_0 + \gamma_1)A(t) - q_4\alpha\gamma S_0B(t),
\]
\[
\frac{dL}{dt} = -q_1q_2q_3(S - S_0) - q_2q_3q_4\left\{1 - R_0\right\} E(t) - \alpha\gamma S_0(\mu_0 + \gamma_1)A(t) - q_4\alpha\gamma S_0B(t).
\]

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

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

(3.7)

where

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

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

\[
\{ \lambda + \mu_0 \} \{ \lambda^4 + b_1 \lambda^3 + b_2 \lambda^2 + b_3 \lambda + b_4 \} = 0,
\]

(3.8)

where

\[
b_1 = q_2 + q_3 + q_4 + \alpha \beta \Pi \gamma \frac{q_3}{q_2},
\]

\[
b_2 = (\alpha \beta \Pi \gamma + q_2 + q_4)q_3 + q_2 \gamma_2 q_4 + \alpha \beta \Pi \gamma \frac{q_4^2}{q_2} + \alpha \beta \Pi \gamma \frac{q_2 q_4 q_1}{q_2},
\]

\[
b_3 = \alpha \beta \Pi \gamma q_3^2 + \alpha \beta \gamma q_2 q_3 + q_4 - q_2 q_3 + \alpha \beta \Pi \gamma \frac{q_2^2 q_4}{q_2},
\]

\[
b_4 = \alpha \beta \Pi \gamma q_3^2 q_4 - q_1 q_2 q_3.
\]

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

\[
\xi(\lambda) = \lambda^4 + b_1 \lambda^3 + b_2 \lambda^2 + b_3 \lambda + b_4.
\]

(3.9)

Following the Routh-Hewritz criteria [34] these eigenvalues are negative, if \( b_i > 0 \) for \( i = 1, \ldots, 4 \), and \( b_1 b_2 b_3 > b_4 \). So for \( R_0 > 1 \), \( \alpha \beta \gamma q_3 q_4 > q_1 q_2 \) and \( q_4 > 1 \), we obtain \( b_i > 0 \) for \( i = 1, \ldots, 4 \), and \( b_3^2 + b_1^2 b_3 < b_1 b_2 b_3 \). 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 \( F_e = (S^*, E^*, A^*, B^*, R^*) \), we construct the Laypnavo function given by

\[
V(t) = \frac{1}{2} \{(S - S^*) + (E - E^*) + (A - A^*) + (B - B^*)\}^2.
\]

(3.10)

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

\[
\frac{dV}{dt} = (S - S^*) + (E - E^*) + (A - A^*) + (B - B^*)\{\Pi - (v + \mu_0)S - \mu_0 E - (\gamma_1 + \mu_0)A - (\mu_0 + \gamma_2 + \mu_1)B\}.
\]

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

\[
\frac{dV}{dt} = (S - S^*) + (E - E^*) + (A - A^*) + (B - B^*)\{(\mu_0 + \beta)E^* + \frac{1}{\alpha \beta \gamma}(\mu_0 + \beta)(\mu_0 + v)(\mu_0 + \mu_1 + \gamma_2) - (\mu_0 + v)S - \mu_0 E - (\mu_0 + \gamma_1)A - (\mu_0 + \mu_1 + \gamma_2)B\}.
\]
Simplification with little re-arrangement of the above equation yields
\[
\frac{dV}{dt} = \{(E - E^*) + (S - S^*) + (B - B^*) + (A - A^*)\} \{\mu_0(E^* - E)
+ \frac{1}{\alpha\gamma}(\mu_0 + \mu_1 + \gamma_2)(\mu_0 + v) + (\mu_0 + \beta)(S^* - S) - \mu_0E
- (\mu_0 + \gamma_1)A - (\mu_0 + \mu_1 + \gamma_2)B\},
\]
implies that
\[
\frac{dV}{dt} = -\{(S - S^*) + (A - A^*) + (E - E^*) + (B - B^*)\} \{\mu_0(E - E^*)
+ (\mu_0 + \beta)(S - S^*) + (\mu_0 + \gamma_1)A + (\gamma_2 + \mu_0 + \mu_1)(B - B^*)\}.
\]
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 Lasalle's invariance principle implies that \(F_s\) 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 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 \(\alpha\) and \(\beta\) 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 \(\alpha, \beta, \mu_1\) and \(\gamma\). 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 \(\alpha\beta\Pi\gamma_3q_4 > q_1q_2, q_4 > 1\) 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
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 ($F_\ast$) 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 crosses 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


