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The asymptotic analysis of hepatitis B virus transmission using an epidemic model

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Abstract

In the current work, we study the temporal dynamics of hepatitis B keeping in view the various routs of transmission and infectious periods. We develop the epidemic model to study its qualitative analysis, while keeping in view the asymptomatic and symptomatic infections periods. We then discuss the well-possedness of the proposed epidemic problem. Particularly, we show the bounded-ness and positivity as well as existence of solution to the proposed epidemic problem. We also calculate the basic reproductive number and discuss the local and global dynamical properties of the considered model. For the local stability, we use the linear stability approach, while for global stability the well known LaSalle's principle are used. Finally, all the theoretical parts have been supported with the help of graphical representation.

Keywords: Hepatitis B model epidemic problem basic reproductive number linear stability analysis local and global dynamics numerical simulation.
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1. Introduction

The inflammation of liver due to hepatitis B virus (HBV) infection leads to cirrhosis, a severe form of liver scarring, which change the structural of the host cells and damage the liver's ability to function properly. HBV attacks hepatocytes, which produce liver infection at the same time as the liver clearing the virus from the body. HBV infection has distinct periods of infection (chronic and acute). The infection

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upto 180 days refers to the acute stage. During this stage the body immunity have the ability to finish the infectious hepatitis B virus, but some time lead to long illness and a more serious phase, called the chronic period of the disease. If an individual have the infection of HBV for more than 180 days causes life long or chronic illness. Usually in this stage often, the individuals have no history of acute illness. The life long illness in the chronic period of the disease dissipate of cirrhosis of liver, which causes liver failure and cancer [1]. The different sources of transferring HBV includes: sharing of blades tooth brushes and razors, secretion of vaginal and semen etc. [2, 3, 4, 5]. In addition, another transmission source is vertical transmission i.e., from infected mother to newborn baby [6]. Worldwide billion of people are infected with the contagious infection of HBV, in which only in China (93) million of people are infected [7, 8]. The most effective control measure for the preventive mechanism of HBV which provide immunity for at least 25 years is the hepatitis B vaccination [9, 10].

The infectious disease epidemiology is a rich filed and have a wealthy literature. Numerous of researchers investigated the temporal dynamics of distinct infectious diseases to learn the dynamics and provide the control analysis. Hepatitis B is one of the leading cause of death, therefore got the attention of researches in order to develop different epidemiological models [11, 12, 13, 14, 15]. A simple basic model has been presented to explore the dynamics of HBV by Anderson in the United Kingdom [16]. Williams et al. investigated the HBV dynamics in order to present the control analysis for the infection [17]. Further, the control mechanism with prediction has been presented by Medley et al. [18]. In addition, a model with age effect to evaluate the vaccination in China has been presented by Zhao et al. [19]. The concept of classical SIR model has been used to suggest control implantation by Bakare et al. [20]. Some control strategies with the aid of epidemiological models have been presented by Kamyad et al. [21]. To evaluate the various endemic states of HBV, a model studied in [22]. Similarly the dynamics of HBV with the aid of mathematical model investigated by Zhang et al. [23]. Recently, Kyere et al. [24] and Khan et al. [25] studied different models to evaluate the effect of involved parameters in the spreading of disease and used suitable control strategies for eliminating of the disease.

Although, the literature revalues that numerous of research articles have been presented which provide valuable outputs while investigating the dynamics and control of HBV. We noted that hepatitis B has many infectious periods and transmission routs. Asymptomatic and symptomatic population are very significant. Especially, in case of asymptomatic the individuals have no clear symptoms while spreads the disease. In the proposed work, we extend the work reported in [25] in order to investigate the dynamics of HBV keeping in view the disease various infection periods and source of transferring. Nevertheless, the reported work present a useful contribution to the epidemiology of HBV, however we would like to improve in order to incorporate new classes and parameters. We formulate the epidemic problem with new phenomenon by enriching the literature more feasible and interesting. We formulate the model along with the categorization of two infected compartments, asymptomatic and symptomatic with the aid of probabilistic transmission approach. We prove the basic properties to make the model feasible and well possed. We also show the existence analysis using the approach of cauchy abstract problem. Further calculating the equilibria of the proposed model, we first find the basic reproductive number and then on the basis of equilibria, we discuss the stabilities. We than prove that the proposed model is globally and locally asymptotically stable. The methods of dynamical systems are used to show the stabilities of the reported model. Particularly, we use the LaSalle's invariance rule and linear stability approach. Also, we execute the model to show the graphical representation of the analytical results for verification purposes.

2. The model

We describe the model and its formulation keeping in view the HBV characteristics to incorporate the new parameters and classes that are not proposed in the work reported in [25]. We assume the different groups of susceptible, chronic, acute and recovered individuals which are respectively symbolized by $w(t)$, $y(t)$, $x(t)$ and $z(t)$. We also put the following constraints.

- The constants and epidemic variables are taken to be non-negative.

- The different infections periods (chronic and acute) are taken.
- The different sources of transmission i.e. from acute and chronic are considered while formulating the model.
- Since, HBV vaccine provides indefinite immunity, so the vaccination of susceptible individuals are taken.
- The probability based disease transmission are taken. In addition, if the susceptible individuals after successful interaction with infectious individuals leads to acute group with probability r, than the $(1-r)$th portion of the susceptible individuals goes to chronic stage.
- In acute period often the immune system are able to clear the virus and get recovered, so a natural recovery is taken with probability s, while for those who leads to the life long illnesses are taken to the chronic period with probability $(1-s)$.
- Newborn are taken to be susceptible, while the death occurs due to disease is taken only in the chronic group of the model.

Thus, with the help of above constraints, we develop a mathematical model which looks like:

$$\begin{cases} \dfrac{dw(t)}{dt} = \Pi - \alpha x(t)w(t) - \alpha\gamma_0 y(t)w(t) - (\nu+\mu_0)w(t), \\ \dfrac{dx(t)}{dt} = r\left\{\alpha x(t)w(t) + \alpha\gamma_0 w(t)y(t)\right\} - \left\{\mu_0+\beta\right\}x(t), \\ \dfrac{dy(t)}{dt} = (1-r)\left\{\alpha x(t)w(t) + \alpha\gamma_0 w(t)y(t)\right\} + \beta s x(t) - \left\{\mu_0+\mu_1+\gamma_2\right\}y(t), \\ \dfrac{dz(t)}{dt} = (1-s)\beta x(t) + \nu w(t) + \gamma_2 y(t) - \mu_0 z(t), \end{cases} \tag{1}$$

with initial population sizes

$$x(0) \geq 0,\ w(0) > 0,\ z(0) > 0,\ y(0) \geq 0, \tag{2}$$

where Π denote the totally susceptible newborn and the parameter α represent is the transmission rate of HBV. γ_0 demonstrate the reduced transmission, while ν symbolize the rate of vaccination. In addition, the parameter μ_0 represent the death rate and β is taken to be the disease recovery rate. We also assume that μ_1 is the portion of death occurred from the HBV infection and γ_2 is the recovery rate of chronic population.

3. Biological and mathematical feasibility

We discuss the biological and mathematical feasibility of the considered epidemic problem. For this purposes we have some results in the following.

Proposition 3.1. *For the proposed model given by Eqn.(1) the non negatively orthant R^4_+ is positively invariant.*

Proof. Let $S = (w, x, y, z)^T$ and re-writing the system (1) as

$$\frac{dS(t)}{dt} = CS + A, \tag{3}$$

$$C = \begin{pmatrix} -R_{11} & 0 & 0 & 0 \\ \alpha\gamma_0 Y & -R_{22} & 0 & 0 \\ (1-r)(\alpha X + \alpha\gamma_0 Y) & s\beta & -R_{33} & 0 \\ \nu & (1-s)\beta & \gamma_2 & -R_{44} \end{pmatrix}, \quad A = \begin{pmatrix} \Pi \\ 0 \\ 0 \\ 0 \end{pmatrix}, \tag{4}$$

where

$$R_{11} = \alpha x + \alpha\gamma_0 y + \mu_0\nu,\ R_{22} = \mu_0 + \beta - \alpha\gamma w,\ R_{33} = \mu_0 + \mu_1 + \gamma_2,\ R_{44} = \mu_0. \tag{5}$$

Clearly, all the elements of C are non-negative except the main diagonal entries and also satisfies Metzler matrix properties, therefore C denote a Metzler matrix also $B \geq 0$. Hence, the proposed model (3) is positively invariant in non-negative orthant R^4_+.

$$C = B^1(0, t_+) \times B^1(0, t_+) \times B^1(0, t_+) \times B^1(0, t_+),\quad t_+ > 0, \tag{6}$$

Define the norm on C as

$$\sum_{i=1}^{4} \|\Psi_i\| \text{ for } \Psi(t) = (\Psi_1(t), \Psi_2(t), \Psi_3(t), \Psi_4(t))^T \in S. \tag{7}$$

where state space is in the form

$$\Omega := \{(w, x, y, z) \in C_+ \text{ where } 0 \leq N \leq 1\}, \tag{8}$$

where in Eqn.(8), $N(t) = x(t) + w(t) + z(t) + y(t)$ and $C_+ = B^1_+(0, t_+) \times B^1_+(0, t_+) \times B^1_+(0, t_+) \times B^1_+(0, t_+)$ denotes the positive cone $B^1(0, t_+)$ and $(\Psi_1, \Psi_2, \Psi_3, \Psi_4) = (w, x, y, z)$. We consider a linear operator which is denoted by R and given by

$$(R\Psi)(t) = (R_1, R_2, R_3, R_4), \tag{9}$$

where

$$\begin{aligned}
R_1 &= \left(-\frac{d\Psi_1}{dt} - (\mu_0 + \nu)\Psi_1, 0, 0, 0 \right),\\
R_2 &= \left(0, -\frac{d\Psi_2}{dt} - (\mu_0 + \beta)\Psi_2, 0, 0 \right),\\
R_3 &= \left(0, s\beta\Psi_2, -\frac{d\Psi_3}{dt} - (\mu + \mu_1 + \gamma_2)\psi_2, 0 \right),\\
R_4 &= \left(\nu\Psi_1, (1-s)\beta\Psi_2, \gamma_2\Psi_3, -\frac{d\Psi_4}{dt} - \mu_0\Psi_4 \right),
\end{aligned} \tag{10}$$

and the domain $D(R)$ is

$$D(R) = \{\Psi \in C \text{ where, } \Psi_i \in MC[0, t_+), \Psi(0) = (\Psi_i(0), \text{ for} i = 1...4)\}, \tag{11}$$

along with $\Psi_1(0) = w(0), \Psi_2(0) = x(0), \Psi_3(0) = y(0)$ and $\Psi_4(0) = z(0)$. Also the function $MC[0, t_+)$ in Eqn.(11) on $[0, t_+)$ denote absolutely continuous function. We take the nonlinear operation $P : C \to C$ by

$$(P\Psi)(t) = \begin{pmatrix} \Pi - \alpha\Psi_1\Psi_2 - \alpha\gamma_0\Psi_1\Psi_3 \\ r(\alpha\Psi_1\Psi_2 + \alpha\gamma_0\Psi_1\Psi_3) \\ (1-r)(\alpha\Psi_1\Psi_2 + \alpha\gamma_0\Psi_1\Psi_3) \\ 0 \end{pmatrix}, \tag{12}$$

and $u(t) = (w, x, y, z)$, then Eqn.(1) in the form of abstract Cauchy problem is written as

$$\frac{du(t)}{dt} = R(u(t)) + P(u(t)), \mu(0) = \mu_0 \in C, \tag{13}$$

and $\mu_0(t) = (w(0), x(0), y(0), z(0))^T$. To obtain the required results for R and P we follow Inaba and Webb. □

Lemma 3.2. *The linear operator R generates B_0 semi group e^{Rt} and w.r.t semi flow defined by e^{Rt} the space Ω is positive invariant.*

Lemma 3.3. *On C The operator P is continuously differentiable.*

Theorem 3.4. *A unique continues mild solution $\mu(t,\mu_0) \in C_+$ exist for Eqn.(13) and a maximal interval of existence $[0,t_+]$ for each $\mu_0 \in C_+$ and every $\mu_0 \in C_0$, such that*

$$\mu(t) = e^{Rt}\mu_0 + \int_0^t e^{t-\tau} S(\mu(\tau))d\tau. \tag{14}$$

Proposition 3.5. *Let $w(0) > 0$, $x(0) > 0$, $y(0) > 0$ and $z(0) > 0$ then the solution of system (1) is positive for every $t > 0$ if it is exist.*

Proof. Suppose the system (1) has solution in $I \subset [0,\infty)$, then the solution for the first equation in system given by (1) for $\forall\ \ t \in I$ is given by

$$w(t) = \Pi t \exp\left\{-\int_0^t \big(\alpha x(s) + \alpha\gamma_0 y(s) + \mu_0\nu\big)ds\right\} + C\exp\left\{-\int_0^t \big(\alpha x(s) + \alpha\gamma_0 y(s) + \mu_0\nu\big)ds\right\}. \tag{15}$$

So, $w(t) > 0$ for every $t \in I$. In the similar way, we can prove that $x(t)$, $y(t)$ and $z(t)$ have non-negative solution. □

4. Steady states analysis

In this section, we discuss the existence of equilibrium points for proposed model as well as the existence of backward bifurcation. We also find one of the important quantity R_0 for qualitative analysis of the model given by (1). The mathematical model given by (1) of the HBV is studied for the equilibrium points such as endemic equilibrium states and disease free. Consider D_2 represents the equilibrium point of the proposed model, where the population is taken consider to be noninfectious. This equilibrium point refer as disease free and for model (1) is given by $D_2 = \big(w^0, x^0, y^0, z^0\big)$, where $w^0 = \frac{\Pi}{\mu_0+\nu}$, $y^0 = x^0 = 0$ and $z^0 = \frac{\Pi\nu}{\mu_0(\mu_0+\nu)}$. For model (1) we find the basic reproduction number by using Driessche and Watmough method, to find the reproduction quantity, suppose that $J = (x,y)^T$, then the proposed system (1) implies

$$\left.\frac{dX}{dt}\right|_{D_2} = F_1 - V_1, \tag{16}$$

where

$$F_1 = \begin{bmatrix} r\alpha w^0 & r\alpha\gamma_0 w^0 \\ (1-r)\alpha w^0 & (1-r)\alpha\gamma_0 w^0 \end{bmatrix}, \quad V_1 = \begin{bmatrix} \mu_0+\beta & 0 \\ -s\beta & \mu_0+\mu_1+\gamma_2 \end{bmatrix}.$$

The spectral radius of $\rho(FV^{-1})$, i.e. $R_0 = R_1 + R_2 + R_3$, is the required reproduction quantity, where

$$R_1 = \frac{(1-r)w^0\alpha\gamma_0}{q_3},\ R_2 = \frac{w^0\alpha r}{q_2},\ R_3 = \frac{w^0\alpha\beta\gamma_0 rs}{q_2 q_3}, \tag{17}$$

where $q_1 = \nu + \mu_0$, $q_2 = \mu_0 + \beta$ and $q_3 = \gamma_2 + \mu_0 + \mu_1$. For stability of the given model with regard to local and global investigation we have the following results.

Theorem 4.1. *The disease free equilibrium point D_2 of the model given by (1) is stable locally, if $R_0 < 1$ while the disease free equilibrium point D_2 is unstable locally if $R_0 > 1$.*

Proof.

$$J_0\Big|_{D_2} = \begin{pmatrix} -q_1 & -\alpha w^0 & -\alpha\gamma_0 w^0 \\ 0 & r\alpha w^0 - q_2 & r\alpha\gamma_0 w^0 \\ 0 & (1-r)\alpha w^0 + s\beta & (1-r)\alpha\gamma_0 w^0 - q_3 \end{pmatrix}.$$

Clearly one eigenvalue of J is $-q_1 < 0$ which is negative to show that remaining eigenvalues are also we take the reduce matrix given by.

$$J_1\Big|_{D_2} = \begin{pmatrix} r\alpha W^0 - q_2 & r\alpha\gamma_0 w^0 \\ (1-r)\alpha W^0 + s\beta & (1-r)\alpha\gamma_0 w^0 - q_3 \end{pmatrix}.$$

Here we need to show that $H_0 := trace(J_1) < 0$ and $det(J_1) > 0$, so

$$trace(J_1) = -q_2(1-R_2) - q_3(1-R_1), \tag{18}$$

and

$$det(J_1) = q_2 q_3 \left\{ 1 - \left(\frac{(1-r)w^0\alpha\gamma_0}{q_3} + \frac{w^0\alpha r}{q_2} + \frac{w^0\alpha\beta\gamma_0 rs}{q_2 q_3} \right) \right\}, \tag{19}$$

which implies that

$$det(J_1) = q_2 q_3 (1 - R_0). \tag{20}$$

Noted that H_0 holds, if $R_0 < 1$, thus one can conclude that the stability D_2 of the proposed system is locally stable whenever $R_0 < 1$. □

Theorem 4.2. *The DFE point D_2 of the model (1) is stable globally asymptotically if $R_0 < 1$ and unstable whenever $R_0 > 1$.*

Proof. Let

$$F(t) = (w - w^0) + x + y. \tag{21}$$

By using values from the model (1), the derivative of $F(t)$ takes the form

$$\frac{dF(t)}{dt} = \Pi - (\nu + \mu_0)w - (\mu_1 + \mu_0 + \gamma_2) - \mu_0 x - \beta(1-s)x. \tag{22}$$

After using the value of X^0 some mathematical arrangement Eqn.(22) becomes

$$\frac{dF(t)}{dt} = -(\mu_0 + \nu)(w - w^0) - (\mu_0 + \mu_1 + \gamma_2) - \mu_0 x - \beta(1-s)x. \tag{23}$$

It is clear from Eqn.(23) that $\frac{dF(t)}{dt} < 0$ and $\frac{dF(t)}{dt} = 0$ if and only if $x = x^0$, $w = w^0$, $y = y^0$, $z = z^0$. So according to the LaSalles invariant principle the DFE point$E^0(X^0, 0, 0, Z^0)$ is stable globally asymptotically. □

Suppose D_3 represents the endemic equilibrium point and let $x = x^*$, $w = w^*$, $y = y^*$ and $z = z^*$ then the endemic equilibrium becomes

$$\begin{aligned} w^* &= \frac{q_2 q_2}{\alpha(rq_3 + \beta s\gamma_0 r + q_2\gamma_0(1-r))}, \\ x^* &= rq_1 q_3 \left(\frac{R_0 - 1}{\alpha(rq_3 + \beta s\gamma_0 r + q_2\gamma_0(1-r))} \right), \\ y^* &= q_1 \left(q_2(1-r) + r\beta s \right) \left(\frac{R_0 - 1}{\alpha(rq_3 + \beta s\gamma_0 r + q_2\gamma_0(1-r))} \right), \\ z^* &= \frac{(1-s)\beta x^* + \nu w^* + \gamma_2 y^*}{\mu_0}. \end{aligned} \tag{24}$$

Theorem 4.3. *The endemic state D_3 of the proposed model (1) is stable locally, if $R_0 > 1$, while D_3 is locally unstable whenever $R_0 < 1$.*

Proof.

$$J^*\Big|_{D_3} = \begin{pmatrix} -\alpha x^* - \alpha\gamma_0 y^* - q_1 & -\alpha w^* & -\alpha\gamma_0 w^* \\ r(\alpha x^* + \alpha\gamma_0 y^*) & r\alpha w^* - q_2 & r\alpha\gamma_0 w^* \\ (1-r)(\alpha x^* + \alpha\gamma_0 y^*) & (1-r)\alpha w^* + s\beta & (1-r)\alpha\gamma_0 w^* - q_3 \end{pmatrix}.$$

Calculating the characteristic polynomial we obtain

$$P(\lambda) = \lambda^3 + P_1\lambda^2 + P_2\lambda + P_3 \tag{25}$$

where

$$\begin{aligned} P_1 &= q_1 + q_2 + q_3 + x^*\alpha + y^*\alpha\gamma_0 - (1-r)w^*\alpha r - w^*\alpha\gamma_0, \\ P_2 &= q_1q_2 + q_1q_3 + q_2q_3 + x^*\alpha q_2 + x^*\alpha q_3 + y^*\alpha\gamma_0 q_2 + y^*\alpha\gamma_0 q_3 - w^*\alpha q_1 r - w^*\alpha q_3 r \\ &\quad - (1-r)w^*\alpha\gamma_0 q_1 - (1-r)w^{\alpha}\gamma_0 q_2 - w^{\alpha}\beta\gamma_0 rs, \\ P_3 &= q_1q_2q_3 + x^*\alpha q_2 q_3 + y^*\alpha\gamma_0 q_2 q_3 - w^*\alpha q - 1q_3 r - (1-r)w^*\alpha\gamma_0 q_1 q_2 - w^*\alpha\beta\gamma_0 q_1 rs. \end{aligned}$$

Roots of Eqn.(25) are negative whenever Δ_3 is positive, i.e the determinant of the Hurwitz matrix of order 3 is positive, which looks like

$$\Delta_3 = \begin{pmatrix} P_1 & 1 & 0 \\ P_3 & P_2 & P_1 \\ 0 & 0 & P_3 \end{pmatrix}.$$

Thus, the result of local stability around the disease endemic state hold whenever $\Delta_3 > 0$ and $R_0 > 0$. □

Theorem 4.4. *The EE point D_3 of the proposed system given by (1) is globally stable, if $R_0 > 1$ and unstable if $R_0 < 1$.*

Proof. Let

$$H(t) = \{(w - w^*) + (x - x^*) + (y - y^*)\}^2. \tag{26}$$

Taking the time dynamics of $H(t)$ and using model (1) with some mathematical arrangement we may leads to

$$\begin{aligned} \frac{dH(t)}{dt} = -\{(w - w^*) + (x - x^*) + (y - y^*)\}^2 \big[(\mu_0 + \nu)(w - w^0) \\ + (\mu_0 + \mu_1 + \gamma_2) + \mu_0 x - \beta(1-s)x\big]. \end{aligned} \tag{27}$$

Eqn.(27) implies that $\frac{dH(t)}{dt} < 0$ and consequently $\frac{dH(t)}{dt} = 0$ whenever $x = x^*$, $w = w^*$, $y = y^*$, $z = z^*$. So according to LaSalles invariant principle EE point is stable globally asymptotically. □

5. Numerical simulation

Here we perform the numerical simulations to realize the time-related dynamical behavior keep in touch with the hepatitis B model given by (1). It is necessary to show the feasibility of this work and find out that the analytical results are valid with the aid of large-scale numerical simulation. We show here the numerical verification of the our analytical findings as carried out in the previous sections. For, this we use numerical method i.e., 4th order Runge-Kutta technique. We use the theory of linear stability analysis and perturb the initial sizes of the various compartments (w, x, y, z) of the considered model from the disease free state, the solutions curves tends to the equilibrium position irrespective of its initial sizes in the long term run as shown in Figure.1 to 4, which represents the graphical verification of the considered problem at disease free state. Moreover the biological interpretation describe that whenever R_0 is less then unity, each curve of w will tend to its equilibrium as shown in Figure.1 which analyze that there will be always

susceptible individuals. The dynamics of asymptomatic and symptomatic as well as recovered individuals are given in Figure.2–4, which reveals that the solution curves of the infected compartments will tend to its associated equilibrium position as time varies and remain stable i.e., goes to zero. However, the recovered population will always exists as shown in Figure.4.

Again to discuss the temporal dynamics of endemic state we use the linear stability approach and perturb the initial sizes of population form its endemic equilibrium. Clearly we observed that each trajectory of the compartmental population approaches to its associated steady states with the passage of time as shown in Figure.5–8. The biological interpretation reveals that whenever R_0 is not less then unity, then the number of compartmental individuals increases in the initial and reach to its endemic stage. It is very much clear that the dynamics of susceptible population is shown in Figure.5. The dynamics of the asymptomatic, symptomatic and recovered population at endemic states are shown in Figure.6, 7 and 8, which verify that there will be always infected (asymptomatic and symptomatic) as well as recovered population. All these results suggest that if $R_0 > 1$ and applying no proper control mechanism, the disease attains its endemic stage. So a special attention is required to keep the value of R_0 less as much as possible.

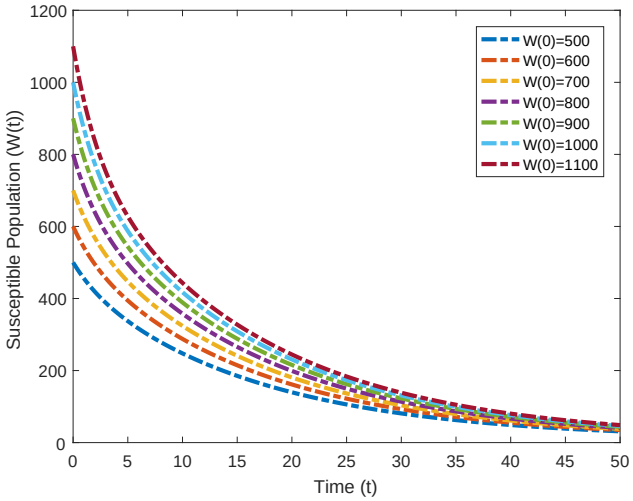

Figure 1: The concern graph represent the dynamics of $(w(t))$ around disease free state for various initial population sizes, and parameters values: $\Pi = 0.6$, $\alpha = 0.0001$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $\beta = 0.1$, $v = 0.01$, $r = 0.5$, $s = 0.6$, $\mu_1 = 0.02$
$, \gamma_2 = 0.4.$

6. Conclusion

In this work we discussed the time dynamics of the hepatitis B infectious virus by using the epidemic model. We categorized the various infectious periods of hepatitis B with probabilistic rate of disease transmission. We formulated the model and studied the basic properties in the form of positivity and bounded-ness to make the epidemic problem meaningful. We also discussed the existence analysis of the problem using the cauchy abstract problem. Moreover, calculated the basic reproductive number and performed the dynamical properties of the model. Particularly, we showed the local dynamics of the model equilibria by using the linear stability analysis while discussed the global dynamics with the help of LaSalle's principle. The conditions for local and global dynamics are derived in the form of threshold parameter and then visualized graphically using the large scale numerical simulation. We discussed the detailed dynamics of the model graphically and supported our analytical findings.

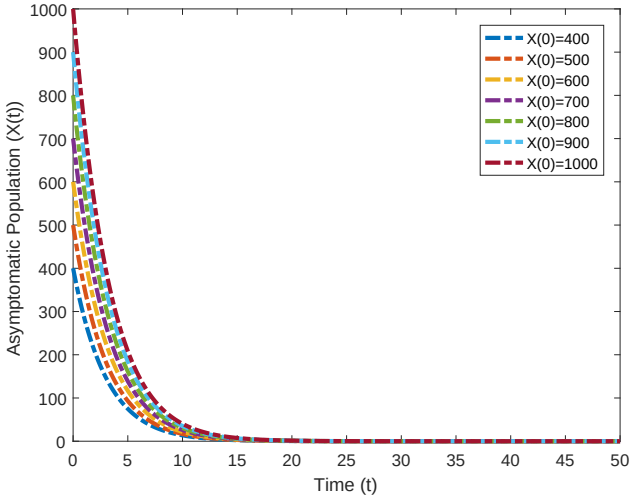

Figure 2: The concern graph show the disease free dynamics of $x(t)$ for various value of initial sizes and parameters values, $\Pi = 0.6$, $\alpha = 0.0001$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $v = 0.01$, $\beta = 0.1$, $r = 0.5$, $s = 0.6$, $\mu_1 = 0.02$
$, \gamma_2 = 0.4.$

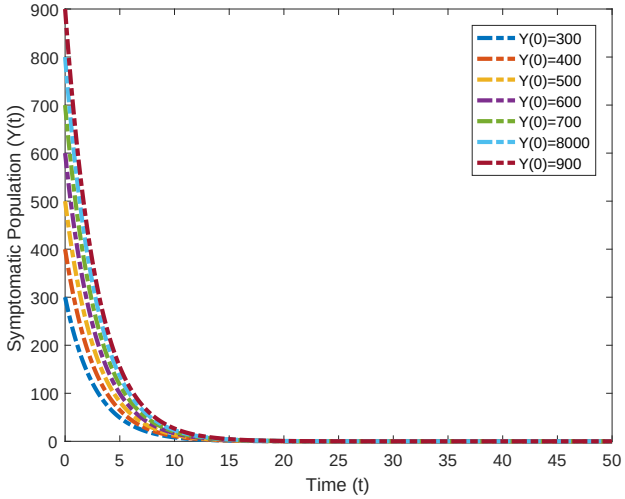

Figure 3: The graph show the time dynamics $y(t)$ around disease free point and various population sizes while value of epidemic parameters are $\Pi = 0.6$, $\alpha = 0.0001$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $v = 0.01$, $r = 0.5$, $\beta = 0.1$, $\mu_1 = 0.02$
$, s = 0.6, \gamma_2 = 0.4.$

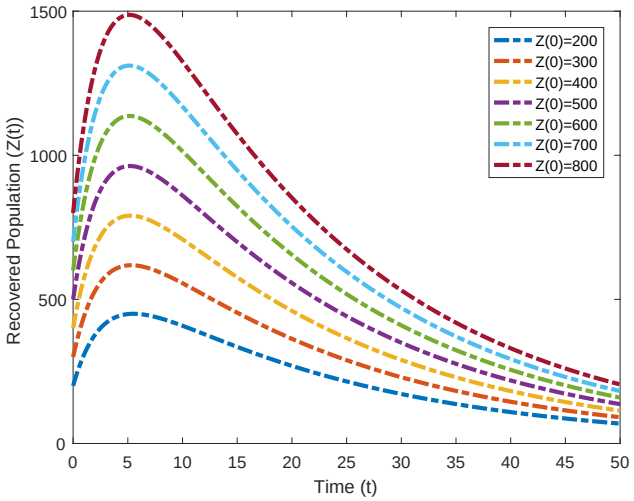

Figure 4: The graph visualizes the temporal dynamics $z(t)$ at disease free state and different initial conditions. Moreover, the epidemic parameters values are as: $\Pi = 0.6$, $\alpha = 0.0001$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $v = 0.01$, $r = 0.5$, $\gamma_2 = 0.4$, $\beta = 0.1$, $s = 0.6$, $\mu_1 = 0.02$.

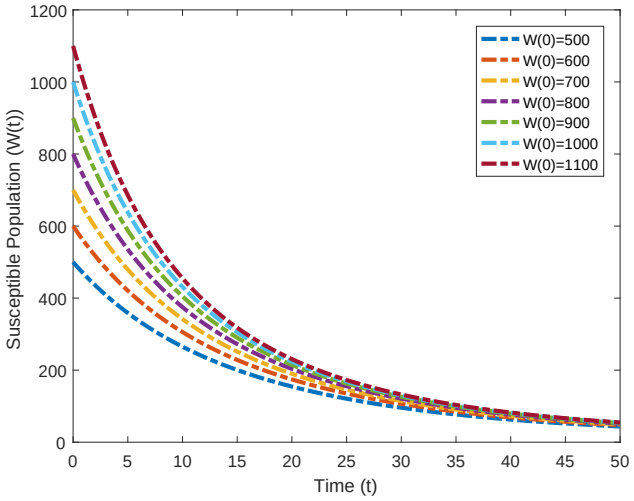

Figure 5: The graph visualizes the disease endemic dynamics of $w(t)$ for different value of initial sizes and parameters values, $\Pi = 0.8$, $\alpha = 0.00005$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $\beta = 0.001$, $v = 0.001$, $r = 0.5$, $s = 0.2$, $\gamma_2 = 0.02$, $\mu_1 = 0.006$.

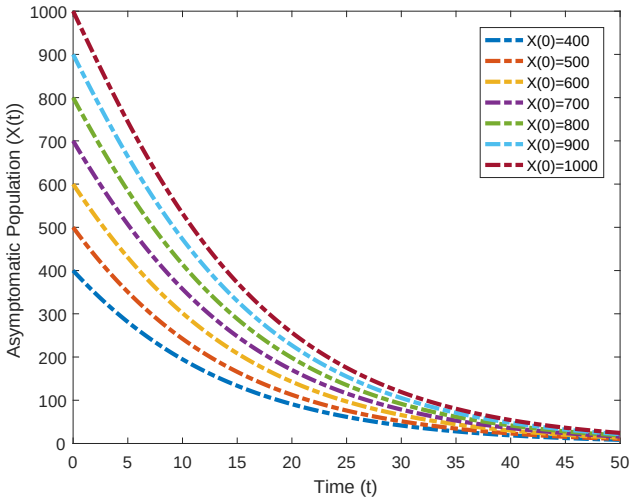

Figure 6: The graph demonstrate the dynamics $x(t)$ around endemic point and various population sizes while value of epidemic parameters are $\Pi = 0.8$, $\alpha = 0.00005$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $\beta = 0.001$, $v = 0.001$, $r = 0.5$, $s = 0.2$, $\gamma_2 = 0.02$, $\mu_1 = 0.006$..

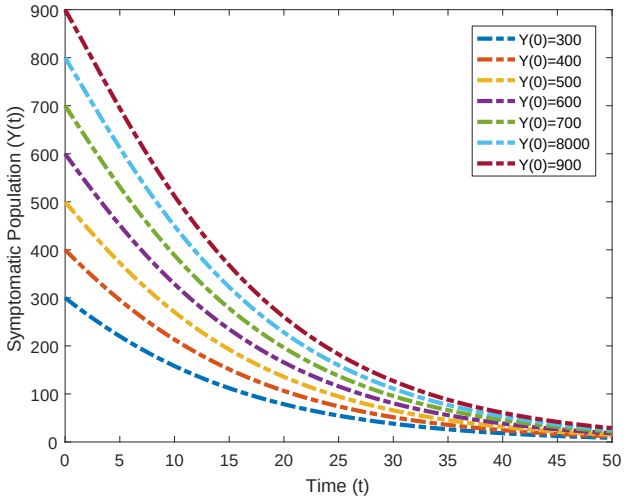

Figure 7: The graph demonstrate the temporal dynamics $y(t)$ at endemic state and different initial conditions. Moreover, the epidemic parameters values are as: $\Pi = 0.8$, $\alpha = 0.00005$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $\beta = 0.001$, $v = 0.001$, $r = 0.5$, $s = 0.2$, $\gamma_2 = 0.02$, $\mu_1 = 0.006$.

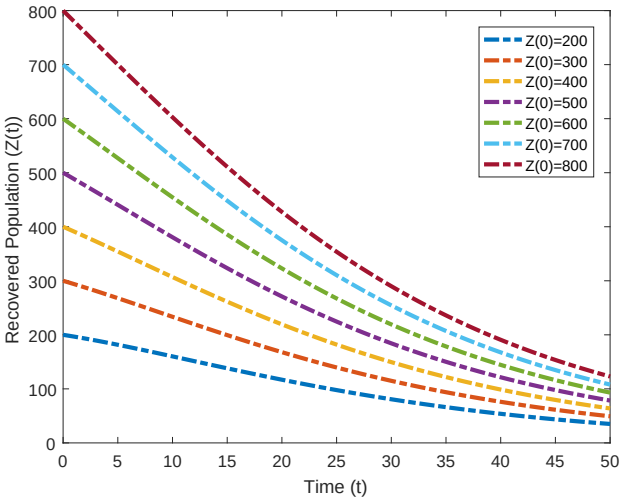

Figure 8: The graph demonstrate the temporal dynamics $z(t)$ at endemic state and different initial conditions. Moreover, the epidemic parameters values are as: $\Pi = 0.8$, $\alpha = 0.00005$, $\gamma_0 = 0.01$, $\mu_0 = 0.05$, $\beta = 0.001$, $v = 0.001$, $r = 0.5$, $s = 0.2$, $\gamma_2 = 0.02$, $\mu_1 = 0.006$.

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Declarations

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- Author's contribution: All the authors equally contributed towards this work.

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