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DYNAMICS OF A DISCRETE HEPATITIS B VIRUS MODEL

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Abstract. Mathematical models are used to study the epidemic diseases to understand the dynamics of disease spreading. In biomathematics, mathematical modeling is considered as a powerful tool to help in interpreting the experimental results of biological phenomena involved in the spreading of disease in more precise way. By using these models, one can estimates the nature of the spread of Hepatitis B virus. So in this paper, we study dynamical properties of a discrete Hepatitis B virus (HBV) model. More precisely, local dynamical properties at equilibrium states are examined by basic reproduction number. Furthermore, we also studied rate of convergence, local and global dynamics at equilibrium states of a discrete HBV model. Finally, theoretical results are confirmed numerically.

Keywords: basic reproduction number; discrete HBV model; equilibrium states; numerical simulation.
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1. INTRODUCTION

Hepatitis B is a potentially life-threatening liver infection cause by the HBV. Infection with the hepatitis B virus affects people all over the world. The HBV is a double-stranded DNA virus from the hepadnavirus family with unique characteristics resembling retroviruses. Direct blood

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transfusion with infected person, other body fluids and contact with contaminated needles of an infected person are the ways to transmit the HBV. It is a potentially fatal condition that can results in liver cancer.

It might be beneficial to investigate anti-HBV infection therapy using mathematical models. In order to understand HBV dynamics, numerous models have been proposed by Pang et al. [1]. The basic infection model, which was developed by Pang et al. [1], is frequently used in order to study the dynamics of virus infection. In a basic model for HBV and HIV infection, Nowak & Bangham [2] proposed models that take the impact of cell-mediated immunity into account. A mathematical model that takes into account lytic as well as nonlytic immune responses was proposed and studied by Nowak & Bangham [2], quantitatively examined its overall dynamics. It is noted that dynamics of infection mathematical models have been extensively studied in recent years by researchers. For instance, Li & Chai [3] have investigated the behavior of HBV model with the effect of drug-resistant treatment:

$$
\begin{cases}
\dot{s} = \lambda - K_s v s - K_r y s - \alpha s, \\
\dot{I} = (1-u) K_s v s - \alpha I, \\
\dot{v} = N_s \alpha I - \delta v - f(C_N) v, \\
\dot{w} = u K_s v s + K_r y s - \alpha w, \\
\dot{y} = N_r \alpha w - \delta y - \beta(u) f(C_N) y,
\end{cases} \tag{1}
$$

where s, I, v, w, y respectively denote uninfected hepatocytes, infected hepatocytes with drug sensitive HBV, hepatocytes infected with drug resistant HBV and drug resistant HBV. λ is the growth rate of uninfected hepatocytes, death rates of viruses and hepatocytes respectively are δ and α while the infection rates of drug-resistant HBV on uninfected hepatocytes and drug-sensitive HBV are K_r and K_s, respectively. Moreover, total number of viruses produced over the course of a hepatocyte's life cycle by drug-resistant and drug-sensitive infected hepatocytes are respectively denoted by N_r and N_S, u denotes the mutation rate between drug-sensitive and drug-resistant hepatocytes, the rate at which medication therapy reduces HBV is expressed as $f(C_N)$, C_N stands for a patient's typical steady-state plasma concentration of nucleoside medications, and finally $\beta(u)$ is the drug therapy inhibition rate. Mouofo et al. [4] have first given the

mathematical formulation and then studied the dynamics of following within-host model of co-infection with Hepatitis B and D:

$$\begin{cases} \dot{s} = \lambda - d_s s - \frac{\beta(1-\eta)sv}{s+I}, \\ \dot{I} = \frac{\beta(1-\eta)sv}{s+I} - a_I I - \delta I w, \\ \dot{v} = k(1-\varepsilon)I - d_v v, \\ \dot{w} = \rho I w + pw - qw^2, \end{cases} \tag{2}$$

where uninfected liver cells is s, infected liver cells is I, v and w respectively represent number free virus and CTL cells. The death rates of cells s, I and free virus are d_s, a_I and dv, respectively whereas production of cells s is λ. Moreover, contact rate between s and v is β, cells I contain v at rate kI, cells I are detached at rate δw by CTL immune responses, virus-specific CTL cells proliferate at rate ρI by contacts with I, η is efficacy of inhibiting new virus infections and finally, p and q stand for the density-dependent rate of immune cell suppression and immune cell proliferation, respectively. Volinsky [5] has investigated the dynamics of following HBV model:

$$\begin{cases} \dot{s} = r - ds - (1-\eta)\beta vs, \\ \dot{I} = (1-\eta)\beta vs - aI - pIy, \\ \dot{v} = (1-\varepsilon)kI - uv - qvw, \\ \dot{w} = -hw + gvw, \\ \dot{y} = cIy - by + DG, \end{cases} \tag{3}$$

where I, v, w, y and s denotes infected cells, free virus, antibody response, cytotoxic T lymphocyte reaction and uninfected cells, respectively, cells s are produced, die and infected by the virus at rates r, ds and bvs respectively. Moreover, $CTLs$ kill the cells I at a rate pIy while growing and die rates are bvs and aI, respectively. Finally, the free viruses v are die, produce and neutralised at rate uv, kI and qvw by antibodies. For more recent investigation regarding the dynamics of HBV models, we refer the reader to the work of eminent researchers [6, 7, 8, 9, 10] and references cited therein.

Hereafter, mathematical model of viral replication for HBV is formulated. The detailed study of immune system have been fundamentally understand by the molecular methods. However, population dynamics of the immune response are studied by mathematical models because experimental approaches are unable to provide sufficient results for understanding the dynamics of HBV. We examine the fundamental dynamics of virus-host cell interaction and the effects of immune responses on viral burden and antigenic diversity. In order to survive and reproduce, viruses rely on their host cell. The balance of the antiviral immune response's beneficial and harmful effects relies on the amount of virus present, the tissues infected and the length of the infection. The host cell might be harmed directly by the virus or by immunological responses to the virus (see [11]). However, cytotoxic T lymphocytes (CTLs), which kill virus-infected cells, play a crucial role in antiviral defence in the majority of viral infections. T cells, Natural killer cells, antibodies and cytokines are crucial elements of a typical immune response to a virus. They are thought to be the primary host immunological component that restricts the extent of viral replication in vivo and consequently establishes virus burden. Now based on Figure 1, latter we write the model equations that describes how a virus interacts with host cells while replicating with three variables: free virus particles v, uninfected and infected cells s and I, respectively. The quantities v, I and s denote the abundance in the blood or tissue of the host while the system of differential equations describe how v, I and s changes with respect to the time. On assuming that cells s encounters free virus, and then turned in to cells I. The free virus particles are infect the cells s at the rate which is proportional to βsv where β is the constant rate represent the efficiency of this process, including the probability and rate of successful infection. kI denote the rate in which cells I produce free virus v whereas the cells I die at a rate δI. At the rate uv, free virus particles v are eradicate from the system, whereas d, δ and u denote death rates of cells s, I and free virus v, respectively. So, $\frac{1}{\delta}$ and $\frac{1}{u}$ are average life time of cells I and free virus, respectively. $\frac{k}{\delta}$ is the amount of virus produced from the cell I while τ and ds are the constant rates at which cells s are produced and then die, respectively. So based on these presumptions, one has the HBV mathematical model designated by system of differential

equations (see [2]):

$$\begin{cases} \dot{s} = \tau - ds - \beta sv, \\ \dot{I} = \beta sv - \delta I, \\ \dot{v} = kI - uv. \end{cases} \tag{4}$$

In the context of populations with non-overlapping generations, discrete-time models governed by difference equations are preferable to continuous ones. Discrete models can also produce effective computational results for numerical simulations. For instance, HBV model (4) after discretization, by Euler-forward formula, takes the following form:

$$\begin{cases} \frac{s_{t+1}-s_t}{h} = \tau - ds_t - \beta s_t v_t, \\ \frac{I_{t+1}-I_t}{h} = \beta s_t v_t - \delta I_t, \\ \frac{v_{t+1}-v_t}{h} = kI_t - uv_t, \end{cases} \tag{5}$$

where h is the step size. After simplification, the required discrete HBV model (5) takes the form:

$$\begin{cases} s_{t+1} = h\tau + (1-hd)s_t - \beta h s_t v_t, \\ I_{t+1} = (1-\delta h)I_t + h\beta s_t v_t, \\ v_{t+1} = (1-hu)v_t + hkI_t. \end{cases} \tag{6}$$

So, in this paper our aim is to investigate the dynamical properties of a three-dimensional discrete HBV model, which is depicted in (6). More precisely, our main contributions in this paper include:

- Investigation of equilibrium states of a discrete HBV model (6).
- Formation of basic reproduction number (BRN).
- Study of local behavior at equilibrium states.
- Study of global dynamics and rate of convergence of a discrete HBV model (6).
- Verification of theoretical results numerically.

The layout of the paper is as follows: In Section 2, we obtained existence of equilibrium states and basic reproduction number whereas local stability analysis at equilibrium states are investigated in Section 3. In Section 4, we studied convergence rate of HBV model (6). In Section

5, we have examined the global dynamics whereas numerical simulation are given in Section 6. Brief conclusion is given in Section 7.

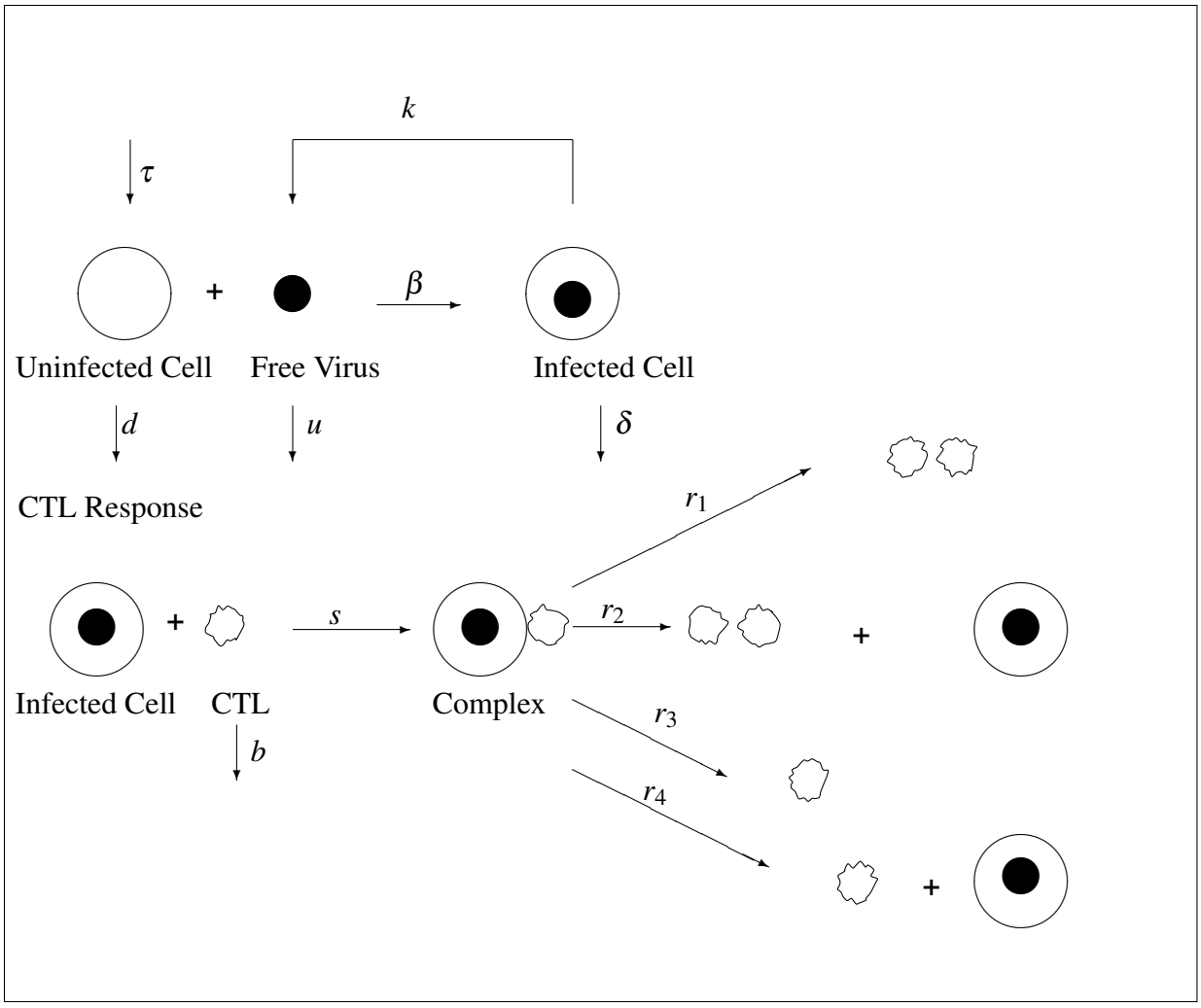

FIGURE 1. Flow chart of a model for virus replication

2. EXISTENCE OF EQUILIBRIUM STATES AND BASIC REPRODUCTION NUMBER

We will explore equilibrium states of a discrete HBV model (6) and basic reproduction number in this section.

Lemma 2.1. For HBV model's equilibrium states, following statements hold:

(i) $\forall\ \tau,d,\beta,\delta,k,u,h$, discrete HBV model (6) has disease-free equilibrium state (DFES) $\Phi_1 = \left(\frac{\tau}{d},0,0\right)$;

(ii) If $\beta > \frac{\delta ud}{\tau k}$ then discrete HBV model (6) has epidemic equilibrium state (EES) $\Phi_2 = \left(\frac{\delta u}{\beta k}, \frac{\tau\beta k-\delta ud}{\delta k\beta}, \frac{\tau\beta k-\delta ud}{\delta u\beta}\right)$.

Proof. If HBV model (6) has equilibrium state $\Phi = (s, I, v)$ then

$$\begin{cases} s = h\tau + (1-hd)s - \beta hsv, \\ I = (1-\delta h)I + h\beta sv, \\ v = (1-hu)v + hkI. \end{cases} \tag{7}$$

It is noted that algebraic system (7) satisfied obviously if $\Phi = (s, I, v) = \left(\frac{\tau}{d}, 0, 0\right)$, and so HBV model (6) has DFES $\Phi_1 = \left(\frac{\tau}{d}, 0, 0\right)$ for all model parameters τ, d, β, δ, k, u and h. Now in order to find EES, we will solve the following system simultaneously:

$$\tau - ds - \beta sv = 0,\ \beta sv - \delta I = 0,\ kI - uv = 0. \tag{8}$$

From last equation of (8), we get

$$I = \frac{uv}{k}. \tag{9}$$

On utilizing (9) into second equation of system (8), we get

$$s = \frac{\delta u}{k\beta}. \tag{10}$$

From first equation of system (8) and (10), we get

$$v = \frac{\tau\beta k - \delta ud}{\delta u\beta}. \tag{11}$$

Finally, from (9) and (11), we get

$$I = \frac{\tau\beta k - \delta ud}{\delta k\beta}. \tag{12}$$

Equations (10), (11) and (12) imply that HBV model (6) has EES $\Phi_2 = \left(\frac{\delta u}{\beta k}, \frac{\tau\beta k-\delta ud}{\delta k\beta}, \frac{\tau\beta k-\delta ud}{\delta u\beta}\right)$ if $\beta > \frac{\delta ud}{\tau k}$. More importantly, it is noted here that if $\beta > \frac{\delta ud}{\tau k}$, that is, $\frac{\beta\tau k}{\delta ud} > 1$ then HBV model (6) has EES $\Phi_2 = \left(\frac{\delta u}{\beta k}, \frac{\tau\beta k-\delta ud}{\delta k\beta}, \frac{\tau\beta k-\delta ud}{\delta u\beta}\right)$, and so we define $\mathfrak{R}_0 := \frac{\beta\tau k}{\delta ud} > 1$ as a BRN. On the other hand, we can say that HBV model (6) has EES $\Phi_2 = \left(\frac{\delta u}{\beta k}, \frac{\tau\beta k-\delta ud}{\delta k\beta}, \frac{\tau\beta k-\delta ud}{\delta u\beta}\right)$ if $\mathfrak{R}_0 > 1$. □

Now linearized form for HBV model (6) at equilibrium state Φ is formulated. So, under the map $(f,g,h) \mapsto (s_{t+1}, I_{t+1}, v_{t+1})$, one has the following variational matrix $J|_\Phi$, which is evaluated at equilibrium state Φ:

$$J|_\Phi := \begin{pmatrix} 1-h(d+\beta v) & 0 & -h\beta s \\ h\beta v & 1-\delta h & h\beta s \\ 0 & kh & 1-hu \end{pmatrix}, \tag{13}$$

where

$$\begin{cases} f = s + h(\tau - ds - \beta sv), \\ g = I + h(\beta sv - \delta I), \\ h = v + h(kI - uv). \end{cases} \tag{14}$$

3. Local Stability Analysis at Equilibrium States

In this section, we will study local behavior of HBV model (6) at equilibrium states by existing theory [12, 13, 14, 15, 16, 17]. For DFES, (13) becomes

$$J|_{\text{DFES}} := \begin{pmatrix} 1-hd & 0 & -\frac{h\beta\tau}{d} \\ 0 & 1-\delta h & \frac{h\beta\tau}{d} \\ 0 & kh & 1-hu \end{pmatrix}, \tag{15}$$

with

$$\lambda_1 = 1-dh,\ \lambda_{2,3} = \frac{2-\delta h - hu \pm h\sqrt{(u-\delta)^2 + \frac{4k\beta\tau}{d}}}{2}. \tag{16}$$

Now at DFES, we will give the local behavior as follows.

Theorem 3.1. DFES of HBV model (6) is

(i) a locally asymptotically stable if

$$0 < d < \frac{2}{h} \text{ and } \frac{4-2\delta h - 2hu + \delta uh^2}{\delta uh^2} < \Re_0 < 1; \tag{17}$$

(ii) an unstable if

$$d > \frac{2}{h} \text{ and } 1 < \Re_0 < \frac{4-2\delta h - 2hu + \delta uh^2}{\delta uh^2}; \tag{18}$$

(iii) a saddle if

$$0 < d < \frac{2}{h} \text{ and } 1 < \mathfrak{R}_0 < \frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2}, \tag{19}$$

or

$$d > \frac{2}{h} \text{ and } \frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2} < \mathfrak{R}_0 < 1; \tag{20}$$

(iv) non-hyperbolic if

$$d = \frac{2}{h}, \tag{21}$$

or

$$\mathfrak{R}_0 = 1, \tag{22}$$

or

$$\mathfrak{R}_0 = \frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2}. \tag{23}$$

Proof. DFES of HBV model (6) is a locally asymptotically stable if $|\lambda_1| = |1-dh| < 1$ and $|\lambda_{2,3}| = \left|\frac{2-\delta h-hu\pm h\sqrt{(u-\delta)^2+\frac{4k\beta\tau}{d}}}{2}\right| < 1$, that is, $0 < d < \frac{2}{h}$ and $\frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2} < \mathfrak{R}_0 < 1$. Therefore, DFES is a stable if $0 < d < \frac{2}{h}$ and $\frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2} < \mathfrak{R}_0 < 1$. Similar calculation shows that DFES of HBV model (6) is an unstable if $d > \frac{2}{h}$ and $1 < \mathfrak{R}_0 < \frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2}$, saddle if $0 < d < \frac{2}{h}$ and $1 < \mathfrak{R}_0 < \frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2}$ or $d > \frac{2}{h}$ and $\frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2} < \mathfrak{R}_0 < 1$, and non-hyperbolic if $d = \frac{2}{h}$ or $\mathfrak{R}_0 = 1$ or $\mathfrak{R}_0 = \frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2}$. □

Finally, we will study local behavior at EES of HBV model (6). At EES, (13) gives

$$J|_{\text{EES}} := \begin{pmatrix} \frac{\delta u-h\tau\beta k}{\delta u} & 0 & -\frac{h\delta u}{k} \\ \frac{h\tau\beta k-\delta uhd}{\delta u} & 1-\delta h & \frac{h\delta u}{k} \\ 0 & kh & 1-hu \end{pmatrix}, \tag{24}$$

with

$$P(\lambda) = \lambda^3 + \mathscr{S}_1\lambda^2 + \mathscr{S}_2\lambda + \mathscr{S}_3 = 0, \tag{25}$$

where

$$\begin{cases} \mathscr{S}_1 = & -3+\delta h+hu+\frac{h\beta\tau k}{\delta u}, \\ \mathscr{S}_2 = & -2hu-2h\delta-\frac{2h\tau\beta k}{\delta u}+\frac{h^2\tau\beta k}{\delta}+\frac{h^2\tau\beta k}{u}+3, \\ \mathscr{S}_3 = & -1+h^3\tau\beta k-h^3\delta ud+\delta h+hu-\frac{h^2\tau\beta k}{u}+\frac{h\tau\beta k}{\delta u}-\frac{h^2\tau\beta k}{\delta}. \end{cases} \tag{26}$$

By Theorem 1.4 of [13], one has the following result

Theorem 3.2. If

$$\begin{cases} |\mathscr{S}_1+\mathscr{S}_3|<1+\mathscr{S}_2, \\ |\mathscr{S}_1-3\mathscr{S}_3|<3-\mathscr{S}_2, \\ \mathscr{S}_3^2+\mathscr{S}_2-\mathscr{S}_3\mathscr{S}_1<1, \end{cases} \tag{27}$$

then EES of HBV model (6) is a sink where $\mathscr{S}_i(1=1,2,3)$ are depicted in (26).

4. Convergence Rate of HBV Model (6)

We study convergence rate of HBV model (6) in this section as follows.

Theorem 4.1. If $\{(s_t,I_t,v_t)\}_{t=0}^{\infty}$ is a positive solution of HBV model (6) such that $\lim_{t\to\infty}\{(s_t,I_t,v_t)\}=\Phi$ then

$$\varpi_t=\begin{pmatrix} \varpi_t^1 \\ \varpi_t^2 \\ \varpi_t^3 \end{pmatrix}, \tag{28}$$

satisfying

$$\begin{cases} \lim_{t\to\infty}\sqrt[t]{||\varpi_t||}= & \left|\lambda_{1,2,3}J|_{\Phi}\right|, \\ \lim_{t\to\infty}\frac{||\varpi_{t+1}||}{||\varpi_t||}= & \left|\lambda_{1,2,3}J|_{\Phi}\right|. \end{cases} \tag{29}$$

Proof. If model (6) has a positive solution $\{(s_t,I_t,v_t)\}_{t=0}^{\infty}$ such that $\lim_{t\to\infty}\{(s_t,I_t,v_t)\}=\Phi$ then

$$\begin{cases} s_{t+1}-s=(1-hd-h\beta v_t)(s_t-s)-h\beta s(v_t-v), \\ I_{t+1}-I=h\beta v_t(s_t-s)+(1-\delta h)(I_t-I)+h\beta s(v_t-v), \\ v_{t+1}-v=kh(I_t-I)+(1-hu)(v_t-v). \end{cases} \tag{30}$$

On setting

$$\begin{cases} \varpi_t^1 = s_t - s, \\ \varpi_t^2 = I_t - I, \\ \varpi_t^3 = v_t - v. \end{cases} \tag{31}$$

From (30) and (31), one has

$$\begin{cases} \varpi_{t+1}^1 = \pi_{11}\varpi_t^1 + \pi_{13}\varpi_t^3, \\ \varpi_{t+1}^2 = \pi_{21}\varpi_t^1 + \pi_{22}\varpi_t^2 + \pi_{23}\varpi_t^3, \\ \varpi_{n+1}^3 = \pi_{32}\varpi_t^2 + \pi_{33}\varpi_t^3, \end{cases} \tag{32}$$

where

$$\begin{cases} \pi_{11} = 1 - hd - h\beta v_t, \\ \pi_{13} = -h\beta s, \\ \pi_{21} = h\beta v_t, \\ \pi_{22} = 1 - \delta h, \\ \pi_{23} = h\beta s, \\ \pi_{32} = kh, \\ \pi_{33} = 1 - hu. \end{cases} \tag{33}$$

From (33), one has

$$\begin{cases} \lim\limits_{t\to\infty} \pi_{11} = 1 - hd - h\beta v, \\ \lim\limits_{t\to\infty} \pi_{13} = -h\beta s, \\ \lim\limits_{t\to\infty} \pi_{21} = h\beta v, \\ \lim\limits_{t\to\infty} \pi_{22} = 1 - \delta h, \\ \lim\limits_{t\to\infty} \pi_{23} = h\beta s, \\ \lim\limits_{t\to\infty} \pi_{32} = kh, \\ \lim\limits_{t\to\infty} \pi_{33} = 1 - hu, \end{cases} \tag{34}$$

that is

$$
\begin{aligned}
\pi_{11} &= 1 - hd - h\beta v + \eta_{11},\\
\pi_{13} &= -h\beta s + \eta_{13},\\
\pi_{21} &= h\beta v + \eta_{21},\\
\pi_{22} &= 1 - \delta h + \eta_{22},\\
\pi_{23} &= h\beta s + \eta_{23},\\
\pi_{32} &= kh + \eta_{32},\\
\pi_{33} &= 1 - hu + \eta_{33},
\end{aligned} \tag{35}
$$

where $\eta_{11}, \eta_{13}, \eta_{21}, \eta_{22}, \eta_{23}, \eta_{32}, \eta_{33} \to 0$ as $t \to \infty$. From existing literature (see [18]), we have error system

$$\varpi_{t+1} = (A + B_t)\varpi_t, \tag{36}$$

where $A = J|_{\Phi}$ and $B_n = \begin{pmatrix} \eta_{11} & 0 & \eta_{13} \\ \eta_{21} & \eta_{22} & \eta_{23} \\ 0 & \eta_{32} & \eta_{33} \end{pmatrix}$. So, the error system becomes

$$\begin{pmatrix} \varpi^1_{t+1} \\ \varpi^2_{t+1} \\ \varpi^3_{t+1} \end{pmatrix} = \begin{pmatrix} 1 - h(d + \beta v) & 0 & -h\beta s \\ h\beta v & 1 - \delta h & h\beta s \\ 0 & kh & 1 - hu \end{pmatrix} \begin{pmatrix} \varpi^1_t \\ \varpi^2_t \\ \varpi^3_t \end{pmatrix}, \tag{37}$$

which is same as linearized system of discrete HBV model (6) at Φ. In particular, (37) gives

$$\begin{pmatrix} \varpi^1_{t+1} \\ \varpi^2_{t+1} \\ \varpi^3_{t+1} \end{pmatrix} = \begin{pmatrix} 1 - hd & 0 & -\frac{h\beta\tau}{d} \\ 0 & 1 - \delta h & \frac{h\beta\tau}{d} \\ 0 & kh & 1 - hu \end{pmatrix} \begin{pmatrix} \varpi^1_t \\ \varpi^2_t \\ \varpi^3_t \end{pmatrix}, \tag{38}$$

and

$$\begin{pmatrix} \varpi^1_{t+1} \\ \varpi^2_{t+1} \\ \varpi^3_{t+1} \end{pmatrix} = \begin{pmatrix} \frac{\delta u - h\tau\beta k}{\delta u} & 0 & -\frac{h\delta u}{k} \\ \frac{h\tau\beta k - \delta uhd}{\delta u} & 1 - \delta h & \frac{h\delta u}{k} \\ 0 & kh & 1 - hu \end{pmatrix} \begin{pmatrix} \varpi^1_t \\ \varpi^2_t \\ \varpi^3_t \end{pmatrix}, \tag{39}$$

which are similar to the linearized system of discrete HBV model (6) obtained at DFES and EES, respectively. □

5. GLOBAL DYNAMICS

In this section, global dynamics at DFES is studied.

Theorem 5.1. DFES of discrete HBV model (6) is a globally stable if

$$0 < d < \frac{1}{h}, \tag{40}$$

$$\delta > \frac{1}{h}, \tag{41}$$

$$u > \frac{1}{h}, \tag{42}$$

and

$$\frac{h^2\beta\tau k}{d} < 1. \tag{43}$$

Proof. Recall that if (40) holds then from first equation of HBV model (6), we have

$$s_{t+1} \leq h\tau + (1-hd)s_t, \tag{44}$$

whose solution is

$$s_{t+1} \leq (1-hd)^t \left(C_1 - \frac{h\tau}{1-(1-hd)} \right) + \frac{h\tau}{1-(1-hd)}, \tag{45}$$

with common ratio $1-hd$, and if $1-hd < 1$, which is true obviously, and so from (45) one gets

$$\lim_{t\to\infty} s_t = \frac{\tau}{d}. \tag{46}$$

Now if (41) and (42) hold then from second and third equations of HBV model (6), we have

$$I_{t+1} \leq h\beta s_t v_t, \tag{47}$$

and

$$v_{t+1} \leq hkI_t. \tag{48}$$

Again recall that if the common ratio $1-hd < 1$ then from (45) and (47), one gets

$$I_{t+1} \leq \frac{h\beta\tau}{d} v_t. \tag{49}$$

From (49), one gets

$$I_t \leq \frac{h\beta\tau}{d} v_{t-1}. \tag{50}$$

From (48) and (50), one gets

$$v_{t+1} \leq \frac{h^2\beta\tau k}{d} v_{t-1}, \tag{51}$$

with

$$v_{t+1} \leq C_2 \left(\sqrt{\frac{h^2\beta\tau k}{d}}\right)^t + C_3 \left(-\sqrt{\frac{h^2\beta\tau k}{d}}\right). \tag{52}$$

From (48) and (49), one gets

$$I_{t+1} \leq \frac{h^2\beta\tau k}{d} I_{t-1}, \tag{53}$$

with

$$I_{t+1} \leq C_4 \left(\sqrt{\frac{h^2\beta\tau k}{d}}\right)^t + C_5 \left(-\sqrt{\frac{h^2\beta\tau k}{d}}\right)^t. \tag{54}$$

Now if (43) holds then from (52) and (54), one gets

$$\lim_{t\to\infty} v_t = \lim_{t\to\infty} I_t = 0. \tag{55}$$

Finally, from (46) and (55), one gets the following required conclusion

$$\lim_{t\to\infty} (s_t, I_t, v_t) = \left(\frac{\tau}{d}, 0, 0\right) = DFES. \tag{56}$$

□

6. Numerical Simulations

Example 6.1. *If* $\tau = 1.0$, $\beta = 26.0$, $\delta = 9.5$, $k = 5.0$, $u = 9.5$, $d = 1.5$, $h = 0.1067$ *then from (17) one gets* $0 < d = 1.5 < \frac{2}{h} = 18.74414245548266$ *and* $\frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2} = 0.9468606065070032 < \Re_0 = 0.9602954755309326 < 1$, *which implies that* DFES $= (0.6666666666666666, 0, 0)$ *of HBV model (6) is a sink (See Figure 2a-2c). Furthermore, if* $\tau = 1.0$, $\beta = 26.0$, $\delta = 9.5$, $k = 5.0$, $u = 9.5$, $d = 1.5$, $h = 0.1067$ *then from (40), (41), (42) and (43) one gets* $0 < d = 1.5 < \frac{1}{h} = 9.37207122774133$, $\delta = 9.5 > \frac{1}{h} = 9.37207122774133$,

$u = 9.5 > \frac{1}{h} = 9.37207122774133$ *and* $\frac{h^2\beta\tau k}{d} = 0.9866904666666666 < 1$ *hold, and therefore,* $\text{DFES} = (0.6666666666666666, 0, 0)$ *of HBV model (6) is a global attractor (See Figure 2d).*

Example 6.2. *If* $\tau = 0.9$, $\beta = 0.9$, $\delta = 0.5$, $k = 3.2$, $u = 0.5$, $d = 3.9$, $h = 0.9$ *then from (18) one gets* $d = 3.9 > \frac{2}{h} = 2.2222222222222223$ *and* $1 < \Re_0 = 2.6584615384615393 < \frac{4-2\delta h-2hu+\delta uh^2}{\delta uh^2} = 11.864197530864196$, *which gives* $\text{DFES} = (0.2307692307692308, 0, 0)$ *of HBV model (6) is an unstable (See Figure 3).*

Example 6.3. *If* $\tau = 3.0$, $\beta = 5.0$, $\delta = 0.1$, $k = 0.7004$, $u = 1.25$, $d = 3.2004$ $h = 0.0125$ *then from (26), we get*

$$\begin{cases} \mathscr{S}_1 = & -1.932525, \\ \mathscr{S}_2 = & 0.8827788750000001, \\ \mathscr{S}_3 = & 0.04976586318359366. \end{cases} \tag{57}$$

Using (57) in to (27), one gets

$$\begin{cases} |\mathscr{S}_1 + \mathscr{S}_3| = 1.8827591368164063 < 1 + \mathscr{S}_2 = 1.882778875, \\ |\mathscr{S}_1 - 3\mathscr{S}_3| = 2.081822589550781 < 3 - \mathscr{S}_2 = 2.117221125, \\ \mathscr{S}_3^2 + \mathscr{S}_2 - \mathscr{S}_3\mathscr{S}_1 = 0.9814292908872826 < 1, \end{cases} \tag{58}$$

which implies that $\text{EES} = (0.0356938892061679, 28.8576527698458, 16.169520000000002)$ *of HBV model (6) is a sink (See Figure 4).*

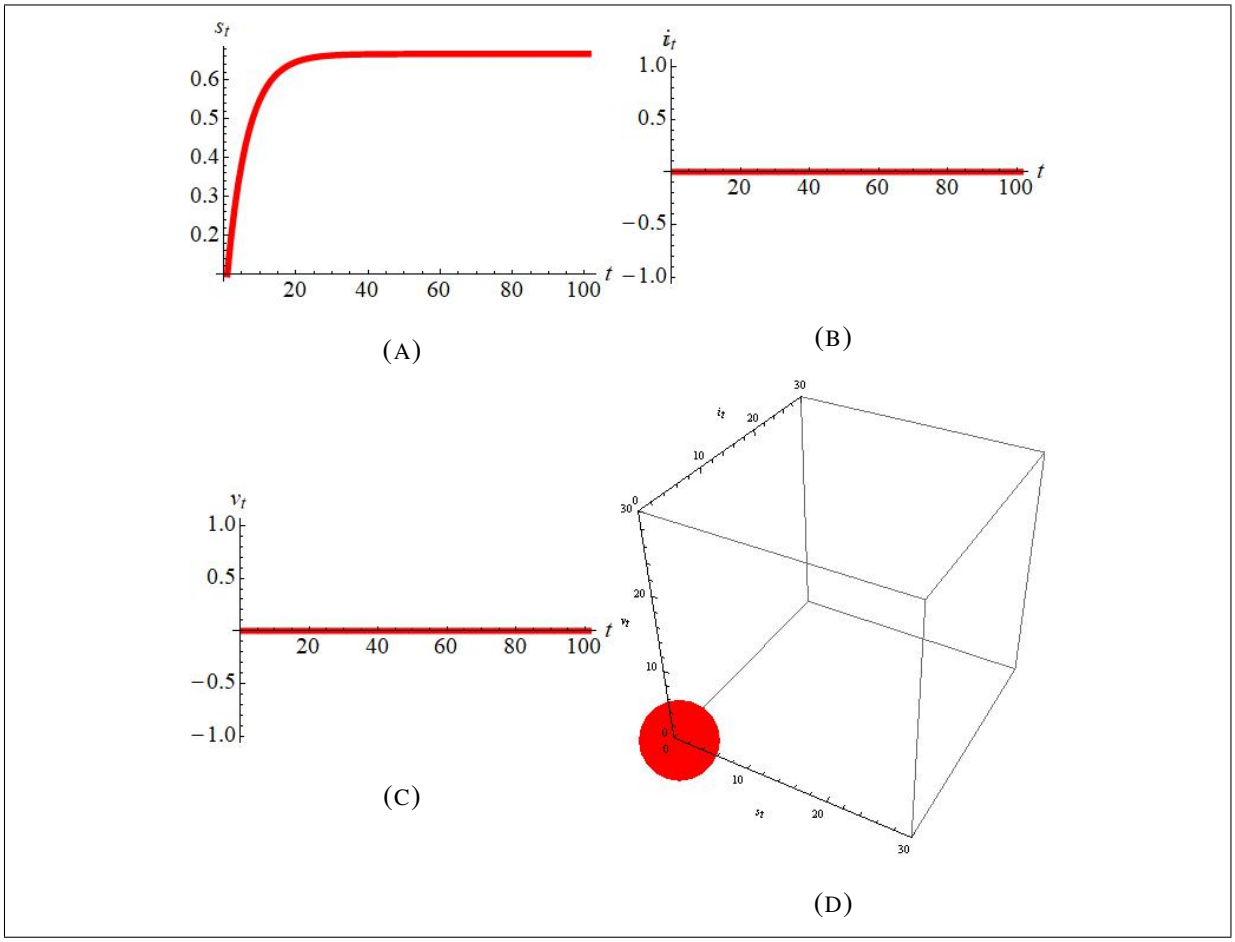

FIGURE 2. Dynamics at DFES $= (0.6666666666666666, 0, 0)$ of discrete HBV model (6) with $(0.1, 0, 0)$. (2a) plot of t vs s_t. (2b) plot of t vs I_t. (2c) plot of t vs v_t. (2d) Global attractor at DFES $= (0.6666666666666666, 0, 0)$.

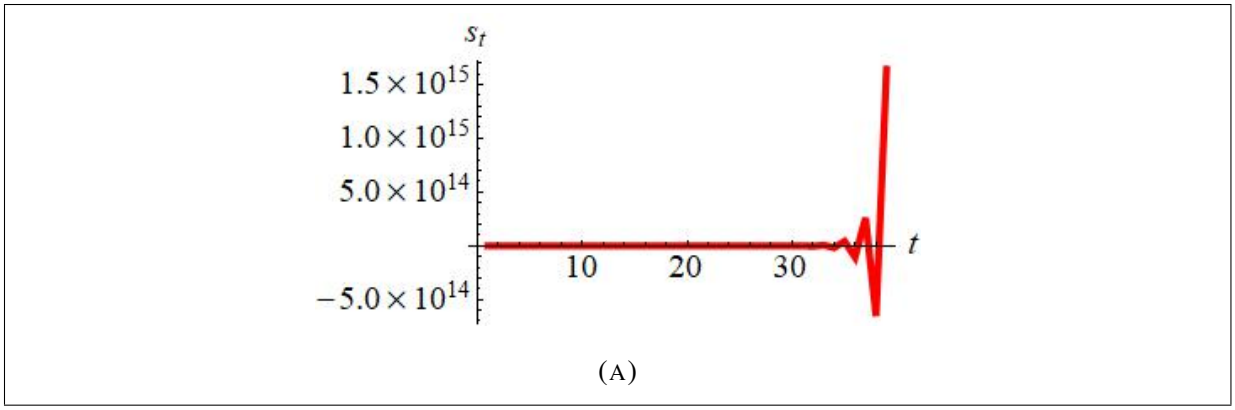

FIGURE 3. Dynamics at DFES $= (0.2307692307692308, 0, 0)$ of discrete HBV model (6) with $(0.4, 0, 0)$.

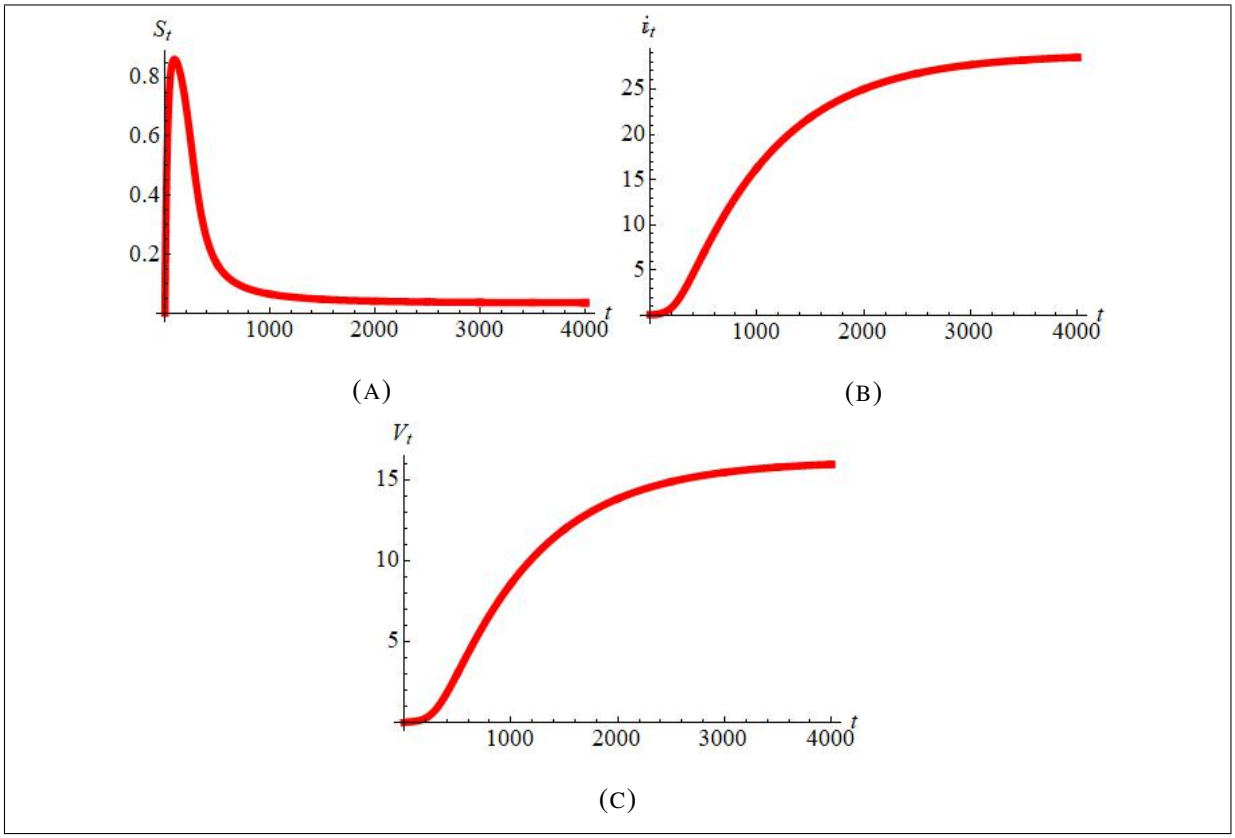

FIGURE 4. Dynamics at EES $= (0.0356938892061679, 28.8576527698458, 16.169520000$ $000002)$ of discrete HBV model (6) with $(0.0006, 0.09, 0.0007)$.

7. CONCLUSION

The work is about dynamics at equilibrium states, examine the basic reproduction number, rate of convergence and global dynamics of a discrete HBV model (6). We have examined that for all model's parameters $\tau, d, \beta, \delta, k, u, h$, discrete HBV model (6) has DFES $\Phi_1 = \left(\frac{\tau}{d}, 0, 0\right)$, and EES $\Phi_2 = \left(\frac{\delta u}{\beta k}, \frac{\tau\beta k - \delta u d}{\delta k \beta}, \frac{\tau\beta k - \delta u d}{\delta u \beta}\right)$ if $\beta > \frac{\delta u d}{\tau k}$. We have also derived the basic reproduction number $\mathfrak{R}_0 := \frac{\beta \tau k}{\delta u d}$ and alternatively, it is examined that HBV model (6) has EES if $\mathfrak{R}_0 > 1$. Next local dynamical properties at DFES of HBV model (6) are investigated, and proved that DFES is a sink if $0 < d < \frac{2}{h}$ and $\frac{4 - 2\delta h - 2hu + \delta u h^2}{\delta u h^2} < \mathfrak{R}_0 < 1$, unstable if $d > \frac{2}{h}$ and $1 < \mathfrak{R}_0 < \frac{4 - 2\delta h - 2hu + \delta u h^2}{\delta u h^2}$, saddle if $0 < d < \frac{2}{h}$ and $1 < \mathfrak{R}_0 < \frac{4 - 2\delta h - 2hu + \delta u h^2}{\delta u h^2}$ or $d > \frac{2}{h}$ and $\frac{4 - 2\delta h - 2hu + \delta u h^2}{\delta u h^2} < \mathfrak{R}_0 < 1$, and finally, non-hyperbolic if $d = \frac{2}{h}$ or $\mathfrak{R}_0 = 1$ or $\mathfrak{R}_0 = \frac{4 - 2\delta h - 2hu + \delta u h^2}{\delta u h^2}$; and moreover, EES is a sink if $|\mathscr{S}_1 + \mathscr{S}_3| < 1 + \mathscr{S}_2$, $|\mathscr{S}_1 - 3\mathscr{S}_3| < 3 - \mathscr{S}_2$ and $\mathscr{S}_3^2 + \mathscr{S}_2 - \mathscr{S}_3\mathscr{S}_1 < 1$ where $\mathscr{S}_i (1 = 1, 2, 3)$ are depicted in (26). Furthermore, rate of

convergence that converges to DFES and EES are also examined. We also proved that DFES of discrete HBV model (6) is a globally stable if $0 < d < \frac{1}{h}$, $\delta > \frac{1}{h}$, $u > \frac{1}{h}$ and $\frac{h^2\beta\tau k}{d} < 1$. At the end, main findings are illustrated numerically.

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

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