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# Stability Analysis of HBV Epidemic Model with Non-Monotonic Incidence Function

*A.K. Dotia[1], M.O. Ibrahim[2], K.A. Bello[2], B.M. Yisa[2], and B.M. Ahmed[2].*

[1]Department of Mathematics, Kwara State Polytechnic, Ilorin, Nigeria
[2]Department of Mathematics, University of Ilorin, Ilorin, Nigeria.

*Abstract*

*In this paper, we present an hepatitis B model with non-monotonic incidence function. The model, which is ofthe form of system of nonlinear differential equations, are constructed. This epidemic model is investigated fordifferent classes of infectious diseases that can be transmitted through an effective contact with an infectiveindividuals, who are contagious (symptomatic and asymptomatic carrier). Mathematical analysis are carriedout, that determines the equilibria solutions and the stability analysis of the equilibria of the model, usingnonlinear Lyapunov function of Goh-Volterra type. In addition, we obtained the numerical simulation to verify the model predictions. The result suggest that the endemic nature of the model is approaching equilibriumwith increase immunization program and other control measures put in place*

**Keywords:** Hepatitis B, endemic, stability, equilibrium

## 1.0 Introduction

Hepatitis B is a potentially life-threatening liver infection caused by the hepatitis B virus (HBV), Edmundet al. (1993). It is a major global health problem and the most serious type of viral hepatitis. Originally knownas "serum hepatitis", the disease has caused epidemics in parts of Asia and Africa, and it is endemic in China(Williams, 2006). About a third of the world population has been infected at one point in their lives, including350 million who are chronic carriers which causes 620,000 deaths worldwide each year (WHO, 2004). If yourbody is able to fight off the hepatitis B infection, any symptoms that you had should go away over a period
of weeks to months, this is termed acute hepatitis B. some people's bodies are not able to completely get ridof the hepatitis B infection. This is called chronic hepatitis B.
Hepatitis is an inflammation of the liver caused by viruses, bacterial infections, or continuous exposureto alcohol, drugs, or toxic chemicals, such as those found in aerosol sprays and paint thinners (Ganem, D.,Prince, A. M. 2004). Inflammation is the painful, red swelling that result when tissues of the body becomeinjured or infected. Inflammation can cause organs to not work properly. Hepatitis can also result from anautoimmune disorder, in which the body mistakenly sends disease-fighting cells to attack its own healthytissue, in this case the liver (Barker et al., 1996). The liver is located in the upper right hand side of theabdomen, mostly behind the rib cage. The liver of an adult normally weighs close to three pounds (Chang,June 2007). No matter what its cause, hepatitis reduces the liver's ability to perform life-preserving functions,
including filtering harmful infectious agents from the blood, storing blood sugar and converting it to usableenergy forms, and producing many proteins necessary for life (Edmund et al., 1993).
In many developed countries (e.g. those in Western Europe and North America), patterns of transmission are different from those in developing countries. The majority of infections in developed countries aretransmitted during young adulthood by sexual activity, tattoo or acupuncture with unclean needles and instruments, and injecting drug use (Gane, E. 2005). Hepatitis B is a major infectious occupational hazard ofhealth workers (Barker et al., 1996). The hepatitis B virus is not spread by contaminated food or water, andcannot be spread casually in the workplace (McManhon et al. 1985).

Corresponding author: A.K. Dotia, E-mail: oe.oyewande@ui.edu.ng, Tel.: +2348077697136

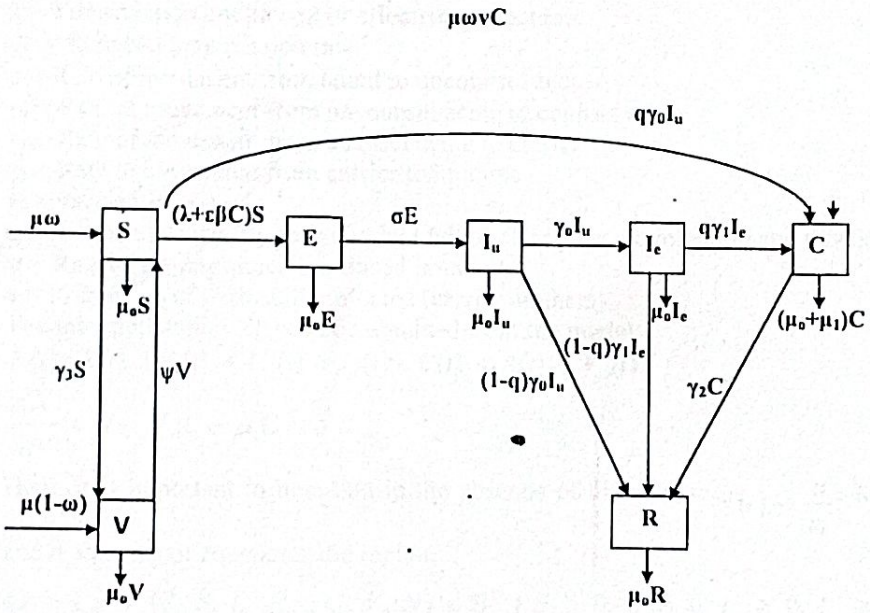
## 2.0 Mathematical Model Formulation

A detailed understanding of the transmission of the hepatitis B virus and other emerging pathogens is crucial in its containment (Goldstein, et al., 2005). Mathematical models can give insight into potential impact interventions (Hou, et al., 2005). The complex interaction of different infection control strategies and their likely impact on transmission can be predicted using mathematical models (Wang, et al., 2004).

We propose a mathematical model to understand the transmission dynamics and prevalence of HBV.The model is constructed based on the characteristics of HBV transmission and the model of L. Zou et al.(2010).Note that L. Zou et al. (2010) considered only six epidemiological groups and did not distinguish theinfective class into subgroups. In fact, the immunity after recovery is lifetime, while that following vaccinationmight wane after some time (Edmunds et al., 1996b). Therefore, we divide the host population into sevenepidemiological groups: the proportion susceptible to infection S; those latently infected E; uneducated acute infections $I_u$ ;educated acute infections $I_e$ ; carriers C; recovered R; and Vaccinated V.

We assume that the population of newborn carriers born to carriers is less than the sum of the death ofcarriers and the population moving from carrier to immune state. In this case we have $\mu\omega v < \mu_0 + \mu_1 + \gamma_2$ .

Otherwise, carriers would keep increasing rapidly as long as there is infection (Lan Zou et al. 2010); i.e., $\frac{dC}{dt} \geq 0$ for $C \neq 0$ or $I \neq 0$ *and* $t \geq 0$ : The model is given by seven ordinary differential equations:




$$\frac{dS}{dt} = \mu\omega(1-vC) + \psi V - [\mu_0 + \lambda + \varepsilon\beta C + \gamma_3]S \tag{2.1}$$

$$\frac{dE}{dt} = [\lambda + \varepsilon\beta C]S - (\mu_0 + \sigma)E \tag{2.2}$$

$$\frac{dI_u}{dt} = \sigma E - (\mu_0 + 2\gamma_0)I_u \tag{2.3}$$

$$\frac{dI_e}{dt} = \gamma_0 I_u - (\mu_0 + \gamma_1)I_e \tag{2.4}$$

$$\frac{dC}{dt} = \mu\omega vC + q(\gamma_0 I_u + \gamma_1 I_e) - (\mu_0 + \mu_1 + \gamma_2)C \tag{2.5}$$

$$\frac{dR}{dt} = \gamma_2 C + (1-q)(\gamma_0 I_u + \gamma_1 I_e) - \mu_0 R \tag{2.6}$$

$$\frac{dV}{dt} = \mu(1-\omega) + \gamma_3 S - (\mu_0 + \psi)V \tag{2.7}$$

Where

$$\lambda = \beta\left(\frac{I_u}{1+\alpha_1 I_u} + \frac{\eta I_c}{1+\alpha_2 I_c}\right)$$

In this model, the Holling-type II incidence function given by

$$g(I) = \frac{\beta I_c}{1+\omega I}$$

The choice of this incidence function is necessary due to the preventive measure (and behavioral changes)taken by the susceptible individuals in response to the severity of the disease.

## 2.1 The Parameters Used are Defined Below

$\mu$ is the birth rate, $\mu_0$ is the Natural mortality rate, $\mu_1$ is the HBV related mortality rate
$\omega$ = proportion of birth without successful vaccination
$\beta$ = Transmission coefficient or effective contact rate
$\varepsilon$ = Reduced transmission rate
$\sigma$ = Rate of movement from latent to uncounsel acute
$\gamma_0$ = Rate of movement from uncounsel acute to counsel acute
$\gamma_1$ = Rate of movement from counsel acute to carrier
$\gamma_2$ = Rate of movement from carrier to immune
$\gamma_3$ = vaccination rate
q = Average probability an individual fail to clear an acute infection and develops to carrier state
$\psi$ = Rate of waning vaccine-induced immunity
v = Proportion of perinatally infected (carrier mothers)
The total population N(t) can be obtained from the model;

$$N(t) = S(t) + E(t) + I_u(t) + I_c(t) + C(t) + R(t) + V(t)$$

$$\frac{dN}{dt} = \mu - N\mu_0 - \mu_1 C \tag{2.8}$$

Here, it is important to note that in the absence of the disease $N(t) \to \frac{\mu}{\mu_0}$. Moreover, under the dynamicsdescribed by the above systems of equations, the region.

$\Omega = \{x = (S, E, I_u, I_c, C, V, N) \in \Re_+^7 \mid S \geq 0, E \geq 0, I_u \geq 0, I_c \geq 0, C \geq 0, V \leq N \leq \frac{\mu}{\mu_0}\}$, is positively invariant. Hence the system is both mathematically and epidemiologically well-posed. Therefore,for initial starting point $x \in \Re_+^7$, the trajectory lies in $\Omega$. Thus we restrict our analysis to the region $\Omega$. (wherethe model make biological sense).

**Lemma:** The basic reproduction number for our model system (2.1) to (2.7) is

$$R_0 = \frac{\beta S_0 \sigma}{k_0 k_1} + \frac{\beta S_0 \gamma_0 \sigma}{k_0 k_1 k_2} + \frac{\varepsilon \beta S_0 q \gamma_0 \sigma (k_2 + \gamma_1)}{k_0 k_1 k_2 k_3}$$

where

$$R_0 = \frac{\beta\mu(\mu_0\omega + \psi)\sigma\left((\mu_0+\gamma_1)q\varepsilon\gamma_0 + q\varepsilon\gamma_0\gamma_1 + (\mu_0+\gamma_1)(-\mu\omega v + \mu_0 + \mu_1 + \gamma_2) + (-\mu\omega v + \mu_0 + \mu_1 + \gamma_2)\gamma_0\right)}{\mu_0(\mu_0+\gamma_3+\psi)(\mu_0+\gamma_1)(\mu_0+2\gamma_0)(\mu_0+\sigma)(-\mu\omega v + \mu_0 + \mu_1 + \gamma_2)} \tag{2.9}$$

The simulations were done using MAPLE program. We use some parameters from literature forour model in equation (2.1) - (2.7) as follows; $\mu = 0.0121$, $\mu_0 = 0.00693$, $\mu_1 = 0.2$ percent, $\beta$ = from 0.95 to 20.49, $\varepsilon = 0.16$, $\sigma = 6$ per year, $\gamma_0 = 4$ per year, $\gamma_1 = 2$ per year, $\gamma_2 = 0.025$ per year, $\gamma_3 = 0$ - 100 percent, $\omega = 0$- 100 percent, q = 0.885, $\psi = 0.1$, v = 0.11 . We calculate the basic reproduction number as $R_0 = 3.961482318$.

## 3.0 Stability Analysis

In our attempt on the steady state analysis, we consider the reduced following system, because R appearsonly in the sixth equation:

$$\frac{dS}{dt} = \mu\omega(1-\nu C) + \psi V - [\mu_0 + \lambda + \varepsilon\beta C + \gamma_3]S \tag{3.1}$$

$$\frac{dE}{dt} = [\lambda + \varepsilon\beta C]S - (\mu_0 + \sigma)E \tag{3.2}$$

$$\frac{dI_u}{dt} = \sigma E - (\mu_0 + 2\gamma_0)I_u \tag{3.3}$$

$$\frac{dI_c}{dt} = \gamma_0 I_u - (\mu_0 + \gamma_1)I_c \tag{3.4}$$

$$\frac{dC}{dt} = \mu\omega\nu C + q(\gamma_0 I_u + \gamma_1 I_c) - (\mu_0 + \mu_1 + \gamma_2)C \tag{3.5}$$

$$\frac{dV}{dt} = \mu(1-\omega) + \gamma_3 S - (\mu_0 + \psi)V \tag{3.6}$$

## 3.1 Equilibrium Solutions

The system equation (3.1 - 3.6) has two equilibrium solutions:

*(1) a disease free equilibrium solution*

$$E_0^{dfe} = (S_0,\ 0,\ 0,\ 0,\ 0,\ V_0),$$

where

$$S_0 = \frac{\mu(\mu_0\omega + \psi)}{\mu_0(\mu_0 + \gamma_3 + \psi)}, \qquad V_0 = \frac{\mu(\mu_0 + \gamma_3 - \mu_0\omega)}{\mu_0(\mu_0 + \gamma_3 + \psi)} \tag{3.1.1}$$

*(2) an endemic equilibrium solution*

if $R_0 > 1$ . where

$$E_*^{ee} = (S^{**},\ E^{**},\ I_u^{**},\ I_c^{**},\ C^{**},\ V^{**}),$$

$$S^{**} = \frac{(\mu_0+\sigma)(\mu_0+2\gamma_0)(\mu_0+\gamma_1)}{\beta\gamma_0\sigma}\left(\frac{(\mu_0+\gamma_1)}{(\mu_0+\gamma_1)\alpha_1 I_c^{**} + \gamma_0} + \frac{\eta}{\alpha_2 I_c^{**} + 1} + \frac{\varepsilon q(\mu_0 + 2\gamma_1)}{-\mu\omega\nu + \mu_0 + \mu_1 + \gamma_2}\right)^{-1} \tag{3.1.2}$$

$$E^{**} = \frac{(\mu_0 + 2\gamma_0)(\mu_0 + \gamma_1)I_c^{**}}{\gamma_0\sigma} \tag{3.13}$$

$$I_c^{**} = \frac{\gamma_0 I_u^{**}}{(\mu_0 + \gamma_1)} \tag{3.1.4}$$

$$I_u^{**} = \frac{(\mu_0 + \gamma_1)I_c^{**}}{\gamma_0} \tag{3.1.5}$$

$$C^{**} = \frac{qI_u^{**}(\mu_0 + 2\gamma_1)}{-\mu\omega\nu + \mu_0 + \mu_1 + \gamma_2} \tag{3.1.6}$$

$$V^{**} = \frac{-\mu(1-\omega) - \gamma_3 S^{**}}{-\mu_0 - \psi} \tag{3.1.7}$$

### 3.2 Local Stability of the Equilibria

**Theorem 1** The disease free equilibrium is locally asymptotically stable if $R_0<1$.

**Proof**

The Jacobian matrix of the system of equations is given in the form;

(3.2.1)

Evaluating the above at disease free, the Variational (Jacobian) matrix becomes:

$$J_0=\begin{pmatrix} -\mu_0-\gamma_3 & 0 & -\beta S & -\eta\beta S & -\varepsilon\beta S-\mu\omega\nu & \psi \\ 0 & -\mu-\sigma & \beta S & \eta\beta S & \varepsilon\beta S & 0 \\ 0 & \sigma & -\mu_0-2\gamma_0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_0 & -\mu_0-\gamma_1 & 0 & 0 \\ 0 & 0 & q\gamma_0 & q\gamma_1 & -(-\mu\omega\nu+\mu_0+\mu_1+\gamma_2) & 0 \\ \gamma_3 & 0 & 0 & 0 & 0 & -\mu_0-\psi \end{pmatrix}$$

$$J^*=\begin{pmatrix} -\mu_0-\beta\left(\frac{I_u}{\alpha_1 I_u+1}+\frac{\eta I_c}{\alpha_2 I_c+1}\right)-\varepsilon\beta C-\gamma_3 & 0 & \frac{-\beta S}{(\alpha_1 I_u+1)^2} & \frac{-\eta\beta S}{(\alpha_1 I_u+1)^2} & -\varepsilon\beta S-\mu\omega\nu & \psi \\ \beta\left(\frac{I_u}{\alpha_1 I_u+1}+\frac{\eta I_c}{\alpha_2 I_c+1}\right)-\varepsilon\beta C & -\mu-\sigma & \frac{\beta S}{(\alpha_1 I_u+1)^2} & \frac{\eta\beta S}{(\alpha_1 I_u+1)^2} & \varepsilon\beta S & 0 \\ 0 & \sigma & -\mu_0-2\gamma_0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_0 & -\mu_0-\gamma_1 & 0 & 0 \\ 0 & 0 & q\gamma_0 & q\gamma_1 & -(-\mu\omega\nu+\mu_0+\mu_1+\gamma_2) & 0 \\ \gamma_3 & 0 & 0 & 0 & 0 & -\mu_0-\psi \end{pmatrix}$$

The eigen-values of the matrix $J_0$ are;

(3.2.2)

$$\lambda_1=-k_5,\quad \lambda_2=-\frac{k_4k_5-\psi\gamma_3}{k_5},\quad \lambda_3=-k_0,\quad \lambda_4=\frac{T\sigma-k_0k_1}{k_0},$$

for $\lambda_4$ to be negative,

$$\lambda_5=-\frac{T\eta\sigma k_1+T\sigma k_2-k_0k_1k_3}{T\sigma-k_0k_1},\quad \lambda_6=-\frac{T\sigma\varepsilon k_1q\gamma_1+T\sigma\varepsilon k_2q\gamma_0-T\eta\sigma k_1k_3-T\eta\sigma k_2k_3-k_0k_1k_2k_3}{T\eta\sigma k_1+T\sigma k_2-k_0k_1k_2}$$

$$\lambda_4<0\Rightarrow T\sigma-k_0k_1<0\Rightarrow -k_0k_1<-T\sigma\Rightarrow k_0k_1>T\sigma \qquad (\mu_0+2\sigma)(\mu_0+\gamma_0+\alpha_1)>\beta S\sigma$$

If all the eigenvalues are negative, then $R_0<1$, and the disease-free equilibrium is locally asymptotically stable.

**Theorem 2**

The endemic equilibrium is locally asymptotically stable if $R_0>1$.

**Proof**

The Jacobian matrix of the system of equations is given in the form;

the above can be rewritten as;

(3.2.3)

$$\begin{pmatrix} -\mu_0-\beta\left(\frac{I_u}{\alpha_1 I_u+1}+\frac{\eta I_c}{\alpha_2 I_c+1}\right)-\varepsilon\beta C-\gamma_3 & 0 & \frac{-\beta S}{(\alpha_1 I_u+1)^2} & \frac{-\eta\beta S}{(\alpha_1 I_u+1)^2} & -\varepsilon\beta S-\mu\omega\nu & \psi \\ \beta\left(\frac{I_u}{\alpha_1 I_u+1}+\frac{\eta I_c}{\alpha_2 I_c+1}\right)-\varepsilon\beta C & -\mu-\sigma & \frac{\beta S}{(\alpha_1 I_u+1)^2} & \frac{\eta\beta S}{(\alpha_1 I_u+1)^2} & \varepsilon\beta S & 0 \\ 0 & \sigma & -\mu_0-2\gamma_0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_0 & -\mu_0-\gamma_1 & 0 & 0 \\ 0 & 0 & q\gamma_0 & q\gamma_1 & -(-\mu\omega\nu+\mu_0+\mu_1+\gamma_2) & 0 \\ \gamma_3 & 0 & 0 & 0 & 0 & -\mu_0-\psi \end{pmatrix}, \quad (3.2.4)$$

Reducing the rows using an elementary row-transformation into upper echelon's form

$$J^* = \begin{pmatrix} -k_5 & 0 & -T_1 & -\eta T_2 & -k_6 & \psi \\ k_7 & -k_0 & T_1 & \eta T_2 & \varepsilon T_3 & 0 \\ 0 & \sigma & -k_1 & 0 & 0 & 0 \\ 0 & 0 & \gamma_0 & -k_2 & 0 & 0 \\ 0 & 0 & q\gamma_0 & q\gamma_1 & -k_3 & 0 \\ \gamma_3 & 0 & 0 & 0 & 0 & -k_4 \end{pmatrix} \quad J^* = \begin{pmatrix} -k_5 & 0 & -T_1 & -\eta T_2 & -k_6 & \psi \\ 0 & -k_0 & M_{23} & M_{24} & M_{25} & M_{26} \\ 0 & 0 & M_{33} & M_{34} & M_{35} & M_{36} \\ 0 & 0 & 0 & -M_{44} & -M_{45} & -M_{46} \\ 0 & 0 & 0 & 0 & -M_{55} & -M_{56} \\ 0 & 0 & 0 & 0 & 0 & -M_{66} \end{pmatrix} \quad (3.2.5)$$

Where

$$M_{23}=\frac{T_1(k_5-k_7)}{k_5},\quad M_{24}=\frac{T_2(k_5-k_7)}{k_5},\quad M_{25}=\frac{\varepsilon T_3 k_5-k_7k_6}{k_5},\quad M_{26}=\frac{k_7\psi}{k_5}$$

$$M_{33}=\frac{\sigma T_1k_5-\sigma Tk_7-k_1k_0k_5}{k_0k_5},\quad M_{34}=\frac{\sigma\eta T_2(k_5-k_7)}{k_0k_5},\quad M_{35}=\frac{\sigma(\varepsilon T_3k_5-k_7k_6)}{k_0k_5},\quad M_{36}=\frac{\sigma k_7\psi}{k_0k_5},$$

$$M_{44}=\frac{\gamma_0\sigma\eta T_2k_5-\gamma_0\sigma\eta T_2k_7+T_1k_2k_5\sigma-T_1k_2k_7\sigma-k_1k_0k_2k_5}{T_1k_5\sigma-T_1k_7\sigma-k_1k_0k_5},\quad M_{45}=\frac{\gamma_0\sigma(\varepsilon T_3k_5-k_7k_6)}{\sigma T_1k_5-\sigma T_1k_7-k_0k_1k_5},$$

$$M_{46}=\frac{\gamma_0\sigma k_7\psi}{T_1k_5\sigma-T_1k_7\sigma-k_1k_0k_5},$$

$$M_{55}=\frac{\left\{\begin{matrix}T_1k_2\sigma k_3k_5-T_1k_2\sigma k_3k_7+T_3k_2k_5q\sigma\varepsilon\gamma_0+T_3k_5q\sigma\varepsilon\gamma_0\gamma_1+T_2\eta k_3k_5\sigma\gamma_0\\-k_1k_0k_2k_3k_5-T_2\eta k_3k_7\sigma\gamma_0-k_2k_6k_7q\sigma\gamma_0-k_6k_7q\sigma\gamma_0\gamma_1\end{matrix}\right\}}{\eta\gamma_0T_2k_5\sigma-\eta\gamma_0T_2k_7\sigma-T_1k_2k_7\sigma-k_1k_0k_2k_5},$$

$$M_{56}=\frac{(k_2+\gamma_1)q\gamma_0\sigma k_7\psi}{\eta\gamma_0T_2k_5\sigma-\eta\gamma_0T_2k_7\sigma-T_1k_2k_7\sigma-k_1k_0k_2k_5},$$

$$M_{65}=\frac{\left\{\begin{matrix}T_1k_2\sigma k_3k_4k_5-T_1k_2\sigma k_3k_4k_7-T_1k_2\sigma k_3\psi\gamma_3+T_3k_2k_4k_5q\sigma\varepsilon\gamma_0+T_3k_2\psi q\sigma\varepsilon\gamma_0\gamma_3\\-k_2k_4k_6k_7q\sigma\gamma_0-k_1k_0k_2k_3k_4k_5+k_1k_0k_2k_3\psi\gamma_3+T_3k_4k_5q\sigma\varepsilon\gamma_0\gamma_1-T_2\eta k_3k_4k_5\sigma\gamma_0\\-T_3\psi q\sigma\varepsilon\gamma_0\gamma_3-T_2\eta k_3k_4k_7\sigma\gamma_0-T_2\eta k_3\psi\sigma\gamma_0\gamma_3-k_4k_6k_7q\sigma\gamma_0\gamma_1\end{matrix}\right\}}{\left\{\begin{matrix}T_1k_2\sigma k_3k_5-T_1k_2\sigma k_3k_7+T_3k_2k_5q\sigma\varepsilon\gamma_0+T_3k_5q\sigma\varepsilon\gamma_0\gamma_1+T_2\eta k_3k_5\sigma\gamma_0\\-T_2\eta k_3k_7\sigma\gamma_0-k_2k_6k_7q\sigma\gamma_0-k_1k_0k_2k_3k_5-k_6k_7q\sigma\gamma_0\gamma_1\end{matrix}\right\}}.$$

The eigenvalues are; $\lambda_1 = -k_5$, $\lambda_2 = -k_0$, $\lambda_3 = M_{33}$, $\lambda_4 = -M_{44}$, $\lambda_5 = -M_{55}$, $\lambda_6 = -M_{66}$ for $\lambda_3$ to be negative, $\lambda_3 < 0 \Rightarrow \sigma T_1' k_5 - \sigma T k_7 - k_1 k_0 k_5 < 0 \Rightarrow \sigma T_1' k_5 < \sigma T k_7 + k_1 k_0 k_5$ i.e. $\sigma T k_7 + k_1 k_0 k_5 > \sigma T_1' k_5$

If all the eigenvalues are negative, then $R_0 > 1$, and the endemic equilibrium is locally asymptotically stable.

## 3.3 Global Stability

**Theorem 3** The disease free equilibrium DFE of the model (3.1-3.6), is globally asymptotically stable GAS in Ω, whenever $R_0 \leq 1$.

**Proof**

Consider the following Lyapunov function:

$$F = \left[\frac{\sigma k_2 k_3 + \sigma\eta k_3\gamma_0 + \varepsilon q\sigma\gamma_0(k_2+\gamma_1)}{\varepsilon k_0 k_1 k_2}\right]E + \left[\frac{k_2 k_3 + \eta k_3\gamma_0 + \varepsilon q\gamma_0(k_2+\gamma_1)}{\varepsilon k_1 k_2}\right]I_u + \left[\frac{k_0 k_1 + (\eta k_3 + \varepsilon q\gamma_1)}{\varepsilon k_0 k_1 k_2}\right]I_c + C \tag{3.3.1}$$

the dot represents differentiation with respect to time, given by

$$\dot{F} = \left[\frac{\sigma k_2 k_3 + \sigma\eta k_3\gamma_0 + \varepsilon q\sigma\gamma_0(k_2+\gamma_1)}{\varepsilon k_0 k_1 k_2}\right]\dot{E} + \left[\frac{k_2 k_3 + \eta k_3\gamma_0 + \varepsilon q\gamma_0(k_2+\gamma_1)}{\varepsilon k_1 k_2}\right]\dot{I}_u + \left[\frac{k_0 k_1 + (\eta k_3 + \varepsilon q\gamma_1)}{\varepsilon k_0 k_1 k_2}\right]\dot{I}_c + \dot{C}$$

$$\left[\frac{\sigma k_2 k_3 + \sigma\eta k_3\gamma_0 + \varepsilon q\sigma\gamma_0(k_2+\gamma_1)}{\varepsilon k_0 k_1 k_2}\right]\left[\beta S_0\left(\frac{I_u}{1+\alpha_1 I_u} + \frac{\eta I_c}{1+\alpha_2 I_c}\right) + \varepsilon\beta C S_0 - k_0 E\right]$$

$$+\left[\frac{k_2 k_3 + \eta k_3\gamma_0 + \varepsilon q\gamma_0(k_2+\gamma_1)}{\varepsilon k_1 k_2}\right](\sigma E - k_1 I_u) + \left[\frac{k_0 k_1 + (\eta k_3 + \varepsilon q\gamma_1)}{\varepsilon k_0 k_1 k_2}\right](\gamma_0 I_u - k_2 I_c) + q(\gamma_0 I_u + \gamma_1 I_c) - k_3 C \tag{3.3.3}$$

$$\leq \left[\frac{k_3 R_0}{\varepsilon}\right](I_u + \eta I_c + \varepsilon C) - \frac{k_3 I_u}{\varepsilon} - \frac{\eta k_3\gamma_0 I_u}{\varepsilon k_2} - \frac{\varepsilon q\gamma_0(k_2+\gamma_1)I_u}{\varepsilon k_2}$$

$$+\frac{(\eta k_3 + \varepsilon q\gamma_1)}{k_2}(\gamma_0 I_u - k_2 I_c) + q(\gamma_0 I_u + \gamma_1 I_c) - k_3 C \tag{3.3.4}$$

$$= \frac{k_3 R_0}{\varepsilon}(I_u + \eta I_c + \varepsilon C) - \frac{k_3}{\varepsilon}(I_u + \eta I_c + \varepsilon C) \tag{3.3.5}$$

$$= \frac{k_3}{\varepsilon}(R_0 - 1)(I_u + \eta I_c + \varepsilon C) \tag{3.3.6}$$

since all the parameters and variables of the model are non-negative,it follows that $\dot{F} \leq 0$ for $R_0 \leq 1$ with $\dot{F} = 0$ if and only if $E = I_u = I_c = C = 0$. Hence, F is a Lyapunov function on Ω.

**Theorem 4** The endemic equilibrium of the reduced model,given by (3.1-3.6), is globally asymptotically stable (GAS) in Ω, if $R_0 > 1$.

**Proof** Consider the reduced model in constructing the following nonlinear Lyapunov function:

$$F = S - S^{**} - S^{**}\ln\left(\frac{S}{S^{**}}\right) + E - E^{**} - E^{**}\ln\left(\frac{E}{E^{**}}\right) + \frac{k_0}{\sigma}\left[I_u - I_u^{**} - I_u^{**}\ln\left(\frac{I_u}{I_u^{**}}\right)\right]$$

$$+\frac{k_1}{q\gamma_0}\left[C - C^{**} - C^{**}\ln\left(\frac{C}{C^{**}}\right)\right] \tag{3.3.7}$$

Lyapunov derivatives gives;

$$\dot{F} = \dot{S} - \frac{S^{**}}{S}\dot{S} + \dot{E} - \frac{E^{**}}{E}\dot{E} + \frac{k_0}{\sigma}\left[\dot{I}_u - \frac{I_u^{**}}{I_u}\dot{I}_u\right] + \frac{k_1}{q\gamma_0}\left[\dot{C} - \frac{C^{**}}{C}\dot{C}\right] \tag{3.3.8}$$

Substituting the reduced model,

$$\dot{F} = \mu\omega(1-\nu C) + \psi V - \left[\begin{matrix}\mu_0 + \beta\left(\frac{I_u}{1+\alpha_1 I_u} + \frac{\eta I_e}{1+\alpha_2 I_e}\right) \\ + \varepsilon\beta C + \gamma_3\end{matrix}\right] S - \frac{S^*}{S}\left[\begin{matrix}\mu\omega(1-\nu C) + \psi V \\ -\left[\begin{matrix}\mu_0 + \beta\left(\frac{I_u}{1+\alpha_1 I_u} + \frac{\eta I_e}{1+\alpha_2 I_e}\right) \\ + \varepsilon\beta C + \gamma_3\end{matrix}\right] S\end{matrix}\right]$$
$$+ \left[\beta\left(\frac{I_u}{1+\alpha_1 I_u} + \frac{\eta I_e}{1+\alpha_2 I_e}\right) + \varepsilon\beta C\right] S - k_0 E - \frac{E^*}{E}\left[\left(\beta\left(\frac{I_u}{1+\alpha_1 I_u} + \frac{\eta I_e}{1+\alpha_2 I_e}\right) + \varepsilon\beta C\right) S - k_0 E\right] \quad (3.3.9)$$
$$+ \frac{k_0}{\sigma}\left[\sigma E - k_1 I_u - \frac{I_u^*}{I_u}(\sigma E - k_1 I_u)\right] + \frac{k_1}{q\gamma_0}\left[\begin{matrix}\mu\omega\nu C + q(\gamma_0 I_u + \gamma_1 I_e) - k_3 C \\ -\frac{C^*}{C}\left[\mu\omega\nu C + q(\gamma_0 I_u + \gamma_1 I_e) - k_3 C\right]\end{matrix}\right]$$

expanding the above, and also considering special case when $\eta = 0$ then we have $f(I_u)$;

$$\dot{F} = A - (\mu_0 + \gamma_3) S - (\beta f(I_u) + \varepsilon\beta C) S - A\frac{S^{**}}{S} + (\mu_0 + \gamma_3) S^{**} + (\beta f(I_u) + \varepsilon\beta C) S^{**}$$
$$+ (\beta f(I_u) + \varepsilon\beta C) S - k_0 E - (\beta f(I_u) + \varepsilon\beta C)\frac{SE^{**}}{E} + k_0 E^{**} + k_1 I_u + \frac{k_1\gamma_1 I_e}{\gamma_0} - \frac{k_1 k_3 C}{q\gamma_0} \quad (3.3.10)$$
$$+ \frac{k_1 I_u C^{**}}{C} - \frac{k_1\gamma_1 I_e C^{**}}{\gamma_0 C} + \frac{k_1 k_3 C^{**}}{q\gamma_0} + k_0 E - \frac{k_0 k_1 I_u}{\sigma} - \frac{k_0 E I_u^{**}}{I_u} + \frac{k_0 k_1 I_u^{**}}{\sigma}$$

simplifying and factorizing the common factors,

$$\dot{F} = \left(A - (\mu_0 + \gamma_3) S\right)\left(1 - \frac{S^{**}}{S}\right) + \left(\beta f(I_u) + \varepsilon\beta C\right) S^{**}\left(1 - \frac{E^{**} S}{E S^{**}}\right) + k_0 E\left(\frac{E^{**}}{E} - 1\right)$$
$$+ \left(k_1 I_u + \frac{k_1\gamma_1 I_e}{\gamma_0} - \frac{k_1 k_3 C}{q\gamma_0}\right)\left(1 - \frac{C^{**}}{C}\right) + \left(k_0 E - \frac{k_0 k_1 I_u}{\sigma}\right)\left(1 - \frac{I_u^{**}}{I_u}\right) \quad (3.3.11)$$

considering the following extract from the above;

$$\left(1 - \frac{S^{**}}{S}\right) \le 0, \quad \left(1 - \frac{E^{**} S}{E S^{**}}\right) \le 0, \quad \left(\frac{E^{**}}{E} - 1\right) \le 0, \quad \left(1 - \frac{C^{**}}{C}\right) \le 0, \quad \left(1 - \frac{I_u^{**}}{I_u}\right) \le 0. \quad (3.3.12)$$

Since all the parameters of the model are nonnegative, it follows that $\dot{F} \le 0$ for all $R_0 > 1$ and with $\dot{F} = 0$ if $S = S^{**}$, $E = E^{**}$, $I_u = I_u^{**}$, *and* $C = C^{**}$ Hence F is a Lyapunov function on Ω.

## 4.0 Numerical Simulation

We makeuse of Maple software to simulate numerically and determine the effect and behaviorof the parameters of the model.

**Table 4a**

| Parameters | | | | | Model ($R_0$) | |
|---|---|---|---|---|---|---|
| $\beta$ | $\gamma_3$ | $\gamma_2$ | $\omega$ | q | DFE | Remark |
| 1.0 | 0.05 | 0.015 | 1.0 | 0.885 | 14.61905689 | unstable |
| 0.9 | 0.15 | 0.025 | 0.9 | 0.885 | 8.408021461 | unstable |
| 0.8 | 0.25 | 0.035 | 0.8 | 0.885 | 6.703960764 | unstable |
| 0.7 | 0.35 | 0.045 | 0.7 | 0.885 | 3.509192401 | unstable |
| 0.6 | 0.45 | 0.055 | 0.6 | 0.885 | 2.376014317 | unstable |
| 0.5 | 0.55 | 0.065 | 0.5 | 0.885 | 1.614546696 | unstable |
| 0.4 | 0.65 | 0.075 | 0.4 | 0.885 | 1.079135561 | unstable |
| 0.3 | 0.75 | 0.085 | 0.3 | 0.885 | 0.6893268001 | stable |
| 0.2 | 0.85 | 0.095 | 0.2 | 0.885 | 0.3975633876 | stable |
| 0.1 | 0.95 | 0.105 | 0.1 | 0.885 | 0.1742101439 | stable |

### 4.1 Discussion of Results

Simulations illustrate the asymptotic stability of DFE studied in section 3. The model described by equations(2.1 - 2.7) exhibit a rich dynamic. We observed that the biological basic reproduction number $R_0$ is fundamental for the study of the basic dynamical properties. Applied to hepatitis B, the model suggests that infectionrates play a great role in the description of the disease. Simulations conducted confirm our results and suggestpossible way to curb the outbreak of the disease epidemic. To control HBV infection, we would like to see whatparameters can reduce the basic reproduction number. From table 4(a), we see that as $\beta$ (effective contact rate)decreases, $R_0$decreases. Consequently, $R_0$ decreases as $1-\omega$, $\gamma_2$ and $\gamma_3$ increase in the model. Therefore, weobserved that the immunization of both newborn carrier and susceptible individuals is an efficient intervention.

## 5.0 Reference


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