

Mathematical Analysis of Hepatitis B Virus Model with Interventions in Taraba State, Nigeria

Umar S. Bashir^{a*} and Ahmed Umar^b

^aDepartment of General Studies, Federal Polytechnic, Bali, Nigeria.

^bDepartment of Mathematics, Modibbo Adama University, P.M.B 2076, Yola, Nigeria.

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ABSTRACT

A new version of a mathematical model for the dynamics of the Hepatitis B Virus (HBV) with a combination of three interventions was proposed. The primary focus of the study was to combine treatment, vaccination, and media campaigns as control strategies for reducing the spread of HBV. The study established the existence of a disease-free steady state of the model. The disease-free steady state was both locally and globally asymptotically stable. Additionally, the numerical results revealed that combining the three interventions is the best control strategy for curbing the menace of HBV. The study suggested that more effective campaign efforts should be supplemented with other major control strategies to get substantial paybacks in curbing the menace of HBV.

1. Introduction

For several decades, hepatitis has posed a global threat, motivating advocates in the fields of medicine, biology, chemistry, and mathematics to take action to stop it. It is an inflammation of the liver that is caused by a variety of infectious viruses and non-infectious agents leading to a range of health problems, some of which can be fatal (WHO, 2017). The hepatitis virus comes in five primary strains, which are A, B, C, D, and E. Particularly, types B and C are the most common causes of liver cirrhosis, liver cancer, and mortality associated with viral hepatitis. They have also led to chronic stages that have cost millions of lives. Hepatitis B or C is thought to affect 354 million people globally, and for the majority, testing and treatment are still out of reach (WHO, 2021).

Acute self-limited hepatitis, fulminant hepatitis necessitating liver transplantation, and both symptomatic and asymptomatic infections were the outcomes of HBV infection. Hepatitis B virus infection affects at least 250 million individuals, and the majority of these cases have resulted in over 820,000 fatalities (WHO, 2021). One of the most common infectious diseases on the globe, hepatitis B (HB) is most common in the Western Pacific and African regions, even though there has been a vaccination to prevent the disease for the past thirty years (WHO, 2017). The life cycle of the Hepatitis B Virus (HBV) is intricate. Once within the host liver cell, the virus travels to the liver cell's nucleus. After entering the nucleus, the viral DNA changes and becomes incorporated into the DNA of the host liver cell, thereby persistently creating a new hepatitis B virus (Canadian Centre for Occupational Health and Safety). It can survive outside the body for at least 7 days and has an incubation period of 75 days (WHO, 2017).

This infection has two possible phases: acute and chronic. Acute hepatitis B infection lasts less than six months. If the disease is acute, the immune system is usually able to clear the virus from the infected body, and the infected body should recover completely within a few months. Most people who acquire hepatitis B as adults have an acute infection. Chronic hepatitis B infection lasts six months or longer. Most infants are infected with HBV at birth and many children infected between 1 and 6 years of age become chronically infected (WHO, 2013). People with AIDS, organ transplant recipients, and children under the age of six who are carriers have a higher risk of developing into a chronic stage (WHO, 2017; Fan *et al.*, 2020). Hepatitis B spreads from person to person by vertical transmission (mother to unborn child) and horizontal transmission (blood, semen, or other body fluids, sexual contact, sharing of needles, unintentional needle sticks) (Bhattacharyya and Ghosh, 2010). Hence, hepatitis B infection does not usually require treatment because most adults clear the infection spontaneously (Hollinger and Lau, 2006).

Two billion people, or one in three people, worldwide have been infected with hepatitis B; 400 million have a chronic infection, one million deaths from the disease each year, and 10–30 million become infected (HBV Foundation, 2014). According to Kramvis and Kew (2007), Nigeria is one of the nations in Africa where HBV infection is hyper-endemic (> 8%). With an age-standardized incidence estimate of 2.6 to <5.1 cases per 100,000 person-years, approximately nine out of ten Nigerians who live with chronic HBV are unaware of their infection status due to government negligence (International Agency for Research on Cancer, 2018; World Hepatitis Alliance,

* Corresponding author. Tel.: +2347068221228

E-mail address: jikanbunu@gmail.com (U. S. Bashir).

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2018; De Maratel *et al.*, 2020; Hepatitis B Foundation, 2014 and Hepatitis Foundation, 2020).

Many control strategies, such as vaccination; media campaigns in educating the public about HBV; patient counseling regarding HBV and enormous ways to prevent infection spread; standard safety precautions in labs and hospitals; and several others, have been implemented to completely eradicate the threat or lessen its degree of spread throughout the world's population. Improving control to reduce the infection in the population is one of the main goals of this research on Hepatitis B Virus (HBV) infection. Clinical studies have been conducted so far to stop the transmission of HBV; these studies include those by Aba and Aminu (2016), Ihedigbo *et al.* (2018), Fan *et al.* (2020), Talla *et al.* (2021), and others. In a similar vein, mathematical models have been a supportive tool that allows us to effectively get control measures and maximize the use of finite resources. Several mathematical models have been built and analyzed, including those by Zhang (2018), Tilahun *et al.* (2021), Aniji *et al.* (2020), Kamyad *et al.* (2014), and among others.

Therefore, the goal of this work was to enhance an earlier model created by Kamyad *et al.* (2014) to have a more effective control method for reducing the dynamics of HBV infection transmission. Thus, adding media attention (campaign) to the current model is the study's primary goal. The combination of three interventions: vaccination, treatment, and media coverage to effectively eradicate HBV illness and stop its spread makes this study exceptional. Additionally, the study developed a new mathematical model that is very unique and closely mimics reality when compared with all other HBV studies hitherto reviewed.

This paper is devoted to the mathematical formulation of an infectious disease (transmission dynamics of HBV) and exhibiting the impact of vaccination, treatment, and media campaigns. the disease-free steady state is established. Local and global asymptotic stability of the disease-free steady state was discussed. Moreover, the numerical result of the proposed model was studied and presented in the form of plots for illustrations. Lastly, the discussion and conclusion of the study were made.

2. Methods

2.1 Model Formulation

The total population $N(t)$, at a time t , is subdivided into eight (8) mutually exclusive compartments, namely: uneducated susceptible individuals, $(S_u(t))$, educated susceptible individuals, $(S_e(t))$, Exposed individuals, $(E(t))$, Acute infected individuals unaware of their status, $(I_u(t))$, Acute infected individuals aware of their status, $(I_a(t))$, Chronic infected individuals unaware of their status, $(C_u(t))$, Chronic infected individual aware of their status, $(C_a(t))$. Recovered Individuals from HBV either spontaneous or treatment or vaccination, $(R(t))$. Educated individuals in this respect are those who have received appropriate public health information about hazardous behaviour that could lead to HBV infection. On the other hand, uneducated individuals are those who do not receive or tend to ignore public health information. Thus

$$N(t) = S_u(t) + S_e(t) + E(t) + I_u(t) + I_a(t) + C_u(t) + C_a(t) + R(t) \quad (1)$$

It is assumed that individuals are recruited into the susceptible population at a constant rate Λ . The fraction ρ of the newly recruited individuals that are educated enter S_e and the remaining $(1-\rho)\Lambda$ individuals enter S_u . Suppose, β is the effective contact rate, then the force of infection is defined as:

$$\lambda = \beta(1-\varepsilon_1)(I_u + I_a + \theta_1 C_u + \theta_2 C_a) \quad (2)$$

where, θ_1 and θ_2 measure the infection level of chronic uneducated and educated respectively. Also, ε_1 is the efficacy of media coverage/campaign on only infected individuals. The uneducated susceptible individuals, S_u become infected at a rate λ and move to E . Also, v is the birth rate and its fluctuation into the S_u is described by $v - v\rho_1(C_u + C_a) - v\rho_2 R$. Parameter ρ_1 and ρ_2 are new born from chronic and recovered compartments respectively. While, S_e acquires the infection at a reduced rate $(1-\kappa)\lambda$, $0 < \kappa \leq 1$ due to enlightenment and move to E . The S_u may be enlightened and move to S_e at a rate ω_1 . It is assumed that individuals at E , some are enlightened and some are not. Moreso, individuals at E move to I_a through little enlightenment with a proportion

$(1 - \lambda_1)$, while Individuals at E move I_u at a proportion λ_1 . Individuals at I_u move to C_u at proportion $\rho_3\lambda_2$ and the remaining proportion $(1 - \rho_3)\lambda_2$ to R . The I_u may be educated and move to I_a at the rate ω_2 . Similarly, individuals at I_a move to C_a at proportion $\rho_4\lambda_3$ and the remaining proportion $(1 - \rho_4)\lambda_3$ to R . It is assumed that treatment is initiated in chronic HBV compartments. Therefore, individuals at C_u move to R by factor of spontaneous recovery (Kar and Jana, 2013; Lai and Yuen, 2007; Zou, Zhang and Ruan, 2010) at a rate λ_4 . The C_u may be educated and move to C_a at a rate ω_3 . Also, C_u recruit new born to the compartment at $\nu\rho_1$. Individual at C_a move to R by factor of spontaneous recovery and treatment at a rate λ_5 and u_1 respectively. Also, C_a recruit new born to the compartment at $\nu\rho_1$. Remember that, recruitment of new born to either C_u or C_a compartment is assumed to be at same rate. Individuals at R move to S_u and S_e by reason of loss of immunity due to vaccine wane at a rate λ_6 and λ_7 respectively. R receives proportion of vaccinated individual both from S_u and S_e at a rate η_1 and η_2 respectively. It is assumed that the vaccine is imperfect (Keeling and Rohani, 2008). $\nu\rho_2$ represents the proportion of immune new born from R . μ denotes natural death rate for all the compartment, while δ denotes a disease-induced death rate for C_u and C_a . The transition from one compartment to other is explained in epidemiological diagram in Figure 1.

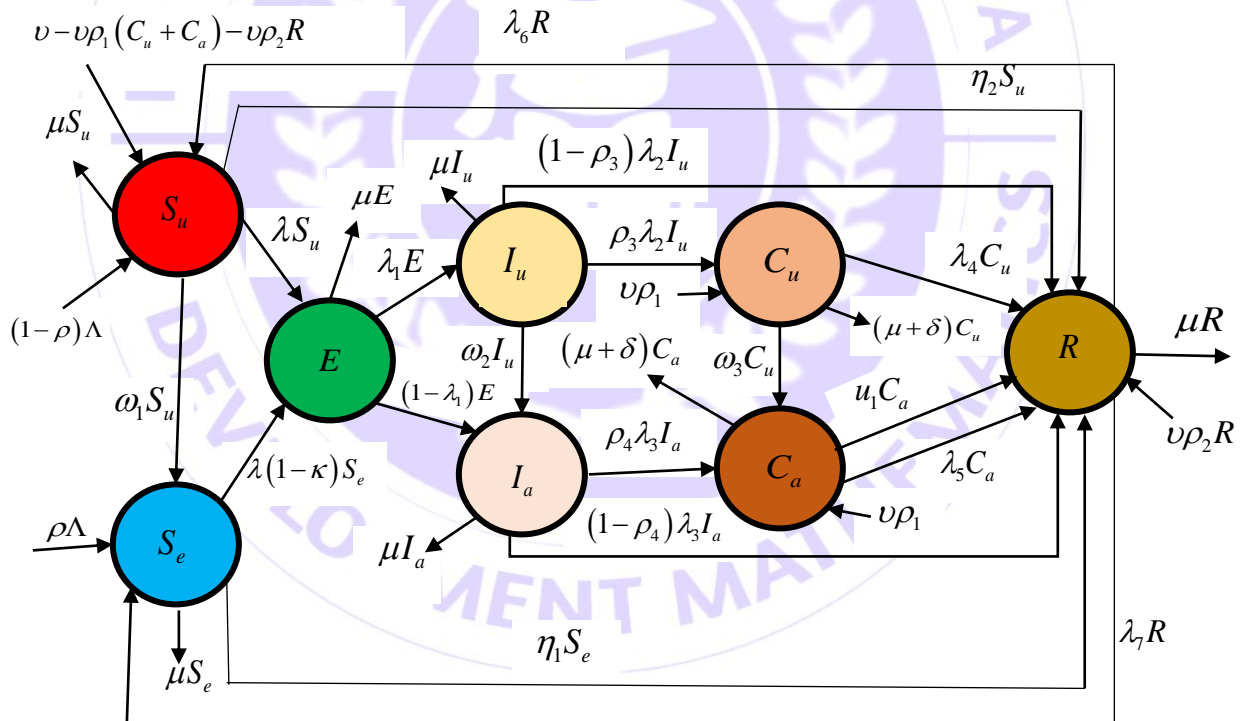


Figure 1: Epidemiological diagram of the model

2.2 Model Equation

The model consists of the following system of nonlinear ordinary differential equations:

$$\begin{aligned}
 \frac{dS_u}{dt} &= \nu + (1 - \rho)\Lambda - \nu\rho_1(C_u + C_a) - (\nu\rho_2 - \lambda_6)R - (\eta_2 + \omega_1 + \lambda + \mu)S_u \\
 \frac{dS_e}{dt} &= \rho\Lambda + \omega_1 S_u + \lambda_7 R - (\lambda(1 - \kappa) + \eta_1 + \mu)S_e \\
 \frac{dE}{dt} &= \lambda(1 - \kappa)S_e + \lambda S_u - ((1 - \lambda_1) + \lambda_1 + \mu)E \\
 \frac{dI_u}{dt} &= \lambda_1 E - (\omega_2 + \rho_3 \lambda_2 + (1 - \rho_3)\lambda_2 + \mu)I_u \\
 \frac{dI_a}{dt} &= \omega_2 I_u + (1 - \lambda_1)E - (\rho_4 \lambda_3 + (1 - \rho_4)\lambda_3 + \mu)I_a \\
 \frac{dC_u}{dt} &= \rho_3 \lambda_2 I_u + \nu\rho_1 C_u - (\lambda_4 + \omega_3 + \mu + \delta)C_u \\
 \frac{dC_a}{dt} &= \rho_4 \lambda_3 I_a + \omega_3 C_u + \nu\rho_1 C_a - (u_1 + \lambda_5 + \mu + \delta)C_a \\
 \frac{dR}{dt} &= (1 - \rho_3)\lambda_2 I_u + (1 - \rho_4)\lambda_3 I_a + \lambda_4 C_u + (u_1 + \lambda_5)C_a + \eta_1 S_e + \eta_2 S_u + \nu\rho_2 R - (\lambda_6 + \lambda_7 + \mu)R
 \end{aligned} \tag{3}$$

With initial conditions

$(S_u(0) \geq 0, S_e(0) \geq 0, E(0) \geq 0, I_u(0) \geq 0, I_a(0) \geq 0, C_u(0) \geq 0, C_a(0) \geq 0, R(0) \geq 0) \in \mathfrak{R}_+^8$, where λ is given in equation (2)

3. Results

3.1 Basic Properties of the Model

The positivity of solution and invariant region of dynamical system (3) is handy, given the fact that, the model describes human population. Hence, the study claims the following results.

3.1.1 Invariant region

Theorem 1: The closed set $\Gamma = \left\{ (S_u, S_e, E, I_u, I_a, C_u, C_a, R) \in \mathfrak{R}_+^8 : N \leq \frac{\nu + \Lambda}{\mu} \right\}$ is positively invariant and

attracting with respect to dynamical system (3)

Proof: Recall that, the total human population $N = S_u + S_e + E + I_u + I_a + C_u + C_a + R$ gives

$$\frac{dN}{dt} = \nu + \Lambda - \mu N - \delta(C_u + C_a)$$

Since all the state variables and parameters of the model remain positive for all $t > 0$, then, the following inequality holds

$$\frac{dN}{dt} \leq (\nu + \Lambda) - \mu N \tag{4}$$

Solving the linear equation in (4) and by comparison theorem of Kribs-Zaleta (1999), it gives:

$$N(t) = N_0 e^{-\mu t} + \frac{(\nu + \Lambda)}{\mu} (1 - e^{-\mu t}) \tag{5}$$

Therefore, the total human population $N(t)$ is bounded above by $\frac{(\nu + \Lambda)}{\mu}$ as $t \rightarrow \infty$. Moreover, $N(t) \leq \frac{(\nu + \Lambda)}{\mu}$ if

$N(0) \leq \frac{(\nu + \Lambda)}{\mu}$. Also, Γ is positively invariant. If $N(0) > \frac{(\nu + \Lambda)}{\mu}$, then, $\frac{dN}{dt} < 0$. Hence, every solution of the dynamical system (3), with initial condition in \mathfrak{R}_+^8 , tend towards Γ as $t \rightarrow \infty$. Thus, Γ is an attracting region.

3.1.2 Positivity of the solution

Theorem 2: Let the initial values for the dynamical system (3) be $S_u(0) \geq 0$, $S_e(0) \geq 0$, $E(0) \geq 0$, $I_u(0) \geq 0$, $I_a(0) \geq 0$, $C_u(0) \geq 0$, $C_a(0) \geq 0$ and $R(0) \geq 0$. Then the solutions $\{S_u, S_e, E, I_u, I_a, C_u, C_a, R\}$ of dynamical system (3) with positive initial values, will remain positive for all time $t > 0$.

Proof:

Let $t = \text{Sup}\{t > 0 : S_u \geq 0, S_e \geq 0, I_u \geq 0, I_a \geq 0, C_u \geq 0, C_a \geq 0, R \geq 0 \in [0, t]\}$, thus $t > 0$. Then, it follows from the first equation of dynamical system (3) considering only negative terms in S_u , it gives:

$$\frac{dS_u}{dt} \geq \nu + \lambda_6 R + (1 - \rho)\Lambda - \nu\rho_1(C_u + C_a) - \nu\rho_2 R - (\eta_2 + \omega_1 + \lambda + \mu)S_u \geq -(\eta_2 + \omega_1 + \lambda + \mu)S_u$$

Using separation of variables method, it gives:

$$\frac{dS_u}{S_u} \geq -(\eta_2 + \omega_1 + \lambda + \mu) dt$$

Integrating both sides and taking anti-log, it gives:

$$\ln S_u \geq -(\eta_2 + \omega_1 + \lambda + \mu)t + c$$

$$\text{So that, } S_u(t) \geq e^{-(\eta_2 + \omega_1 + \lambda + \mu)t + c}$$

At $t = 0$ and with initial condition $S_u(0)$

Then,

$$S_u(t) \geq S_u(0)e^{-(\eta_2 + \omega_1 + \lambda + \mu)t} > 0, \text{ since } (\eta_2 + \omega_1 + \lambda + \mu)t > 0 \quad (6)$$

Using similar argument, it can be shown that the state variables are all positive for all values of time $t (\forall t \geq 0)$. Hence, the solution of dynamical system (3) remains positive for $t > 0$.

3.2 Existence of Steady State

The dynamical system (3) has a disease-free steady state, given by

$$E_0 = (S_u^0, S_e^0, E^0, I_u^0, I_a^0, C_u^0, C_a^0, R^0) = E_0 = \left(\frac{\nu + \Lambda(1 - \rho)}{(\eta_2 + \mu + \omega_1)}, \frac{\Lambda\rho(\eta_2 + \mu) + \omega_1(\Lambda + \nu)}{(\eta_1 + \mu)(\eta_2 + \mu + \omega_1)}, 0, 0, 0, 0, 0, 0 \right)$$

3.3 Local stability of Disease-Free Steady State

Establishing the stability of disease-free steady state of the dynamical system (3) requires to examine the root of the Jacobian matrix of the model.

Theorem 3: (Wiggins, 1983). Suppose all the eigenvalues of the Jacobian matrix evaluated at disease-free steady state have negative real parts. Then, the disease-free steady state of the dynamical system (3) is Locally Asymptotically Stable (LAS), and unstable if at least one of the eigenvalues has positive real part.

Proof:

Proving **Theorem 3**, the study obtained the Jacobian matrix of the dynamical system (3) and eventually evaluate it at the disease-free steady state.

The Jacobian matrix is given by:

$$J(E_1^*) = \begin{bmatrix} -(\eta_2 + \omega_1 + \mu) & 0 & 0 & 0 & 0 & -v\rho_1 & -v\rho_1 & -v\rho_2 \\ \omega_1 & -(\eta_1 + \mu) & 0 & 0 & 0 & 0 & 0 & \lambda_6 \\ 0 & 0 & -(1 + \mu) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_1 & -(\omega_2 + \lambda_2 + \mu) & 0 & 0 & 0 & 0 \\ 0 & 0 & (1 - \lambda_4) & \omega_2 & -(\lambda_3 + \mu) & 0 & 0 & 0 \\ 0 & 0 & 0 & \rho_3 \lambda_2 & 0 & v\rho_1 - (\lambda_4 + \omega_3 + \mu + \delta) & 0 & 0 \\ 0 & 0 & 0 & 0 & \rho_4 \lambda_3 & \omega_3 & v\rho_1 - (u_1 + \lambda_5 + \mu + \delta) & 0 \\ \eta_2 & \eta_1 & 0 & (1 - \rho_3) \lambda_2 & (1 - \rho_4) \lambda_3 & \lambda_4 & u_1 + \lambda_5 & v\rho_2 - (\lambda_6 + \lambda_7 + \mu) \end{bmatrix} \quad (7)$$

By the elementary row operation, it gives matrix (7):

$$J(E_1^*) = \begin{bmatrix} -a_1 & 0 & 0 & 0 & 0 & -v\rho_1 & -v\rho_1 & -v\rho_2 \\ 0 & -a_2 & 0 & 0 & 0 & -a_8 & -a_8 & a_7 \\ 0 & 0 & -a_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -a_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -a_5 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -v\rho_1 + (\lambda_4 + \omega_3 + \mu + \delta) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -v\rho_1 + (u_1 + \lambda_5 + \mu + \delta) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a_6 \end{bmatrix} \quad (8)$$

where

$$a_1 = (\eta_2 + \omega_1 + \mu), \quad a_2 = (\eta_1 + \mu), \quad a_3 = (1 + \mu), \quad a_4 = (\omega_2 + \lambda_2 + \mu), \quad a_5 = (\lambda_3 + \mu),$$

$$a_6 = \left(\frac{\eta_2(\mu(\mu + \lambda_6 + \lambda_7) + \eta_1(\mu + 2\lambda_6 + \lambda_7)) + (\eta_1(\mu + 2\lambda_6 + \lambda_7 - 2v\rho_2) + \mu(\mu + \lambda_6 + \lambda_7 - v\rho_2))\omega_1}{(\eta_1 + \mu)(\eta_2 + \omega_1)} \right), \quad a_7 = \left(\frac{\lambda_6(\eta_2 + \omega_1) - v\omega_1\rho_2}{\eta_2 + \omega_1} \right),$$

and $a_8 = \left(\frac{v\omega_1\rho_1}{\eta_2 + \omega_1} \right).$

The characteristic equation of the row-transformed Jacobian matrix evaluated at the disease-free steady state E_0 is given by

$$|J(E_1^*) - \lambda I| = \begin{vmatrix} -a_1 - \lambda & 0 & 0 & 0 & 0 & -v\rho_1 & -v\rho_1 & -v\rho_2 \\ 0 & -a_2 - \lambda & 0 & 0 & 0 & -a_8 & -a_8 & a_7 \\ 0 & 0 & -a_3 - \lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -a_4 - \lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -a_5 - \lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -v\rho_1 + (\lambda_4 + \omega_3 + \mu + \delta) - \lambda & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -v\rho_1 + (u_1 + \lambda_5 + \mu + \delta) - \lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -a_6 - \lambda \end{vmatrix} = 0$$

$$\left[\begin{aligned} &(-a_1 - \lambda)(-a_2 - \lambda)(-a_3 - \lambda)(-a_3 - \lambda)(-a_4 - \lambda)(-a_5 - \lambda)(-v\rho_1 + (\lambda_4 + \omega_3 + \mu + \delta) - \lambda) \\ &(-v\rho_1 + (u_1 + \lambda_5 + \mu + \delta) - \lambda)(-a_6 - \lambda) \end{aligned} \right] = 0 \quad (9)$$

from equation (9) we present the eigenvalues of the row-transformed Jacobian matrix evaluated at disease-free steady state E_0 as follows:

$$\lambda_1 = -a_1 = -(\eta_2 + \omega_1 + \mu), \text{ since } \eta_2, \omega_1, \mu > 0$$

$$\lambda_2 = -a_2 = -(\eta_1 + \mu), \text{ since } \eta_1, \mu > 0$$

$$\lambda_3 = -a_3 = -(1 + \mu), \text{ since } 1, \mu > 0$$

$$\lambda_4 = -a_4 = -(\omega_2 + \lambda_2 + \mu), \text{ since } \omega_2, \lambda_2, \mu > 0$$

$$\lambda_5 = -a_5 = -(\lambda_3 + \mu), \text{ since } \lambda_3, \mu > 0$$

$$\lambda_6 = -a_6 = \left(\frac{\eta_2(\mu(\mu + \lambda_6 + \lambda_7) + \eta_1(\mu + 2\lambda_6 + \lambda_7)) + (\eta_1(\mu + 2\lambda_6 + \lambda_7 - 2\nu\rho_2) + \mu(\mu + \lambda_6 + \lambda_7 - \nu\rho_2))\omega_1}{(\eta_1 + \mu)(\eta_2 + \omega_1)} \right)$$

Since $\eta_2(\mu(\mu + \lambda_6 + \lambda_7) + \eta_1(\mu + 2\lambda_6 + \lambda_7)) + (\eta_1(\mu + 2\lambda_6 + \lambda_7 - 2\nu\rho_2) + \mu(\mu + \lambda_6 + \lambda_7 - \nu\rho_2))\omega_1 > 0$, and $(\eta_1 + \mu)(\eta_2 + \omega_1) > 0$

$$\lambda_7 < 0, \text{ if } \nu\rho_1 > (\lambda_4 + \omega_3 + \mu + \delta)$$

$$\lambda_8 < 0, \text{ if } \nu\rho_1 > (u_1 + \lambda_5 + \mu + \delta)$$

Therefore, the eigenvalues of the row-transformed Jacobian matrix evaluated at the disease-free steady state, E_0 has negative eigen values. Hence, the disease-free steady state, E_0 of the dynamical system (3) is Locally Asymptotically Stable (LAS), which establishes the theorem 3.

3.4 Global Stability of Disease-Free Steady State

Castillo-Chavez, Feng and Huang (2002) method is used to investigate the global asymptotic stability of the disease-free steady state. In this section, we highlight two conditions that if met, guarantee the global asymptotic stability of the disease-free steady state of the dynamical system. First, the dynamical system (3) must be written in the form:

$$\left. \begin{aligned} \frac{dX}{dt} &= H(X, Z) \\ \frac{dZ}{dt} &= G(X, Z), G(X, Z) = 0 \end{aligned} \right\} \quad (10)$$

where $X \in \mathfrak{R}^m$ denotes (its components) the number of uninfected individuals and $Z \in \mathfrak{R}^n$ denotes (its components) the number of infected individuals.

The conditions (H_1) and (H_2) below must be met to guarantee global asymptotic stability.

$$(H_1): \frac{dX}{dt} = H(X, 0), X^* \text{ is Globally Asymptotically Stable (G.A.S)}$$

$$(H_2): G(X, Z) = PZ - \hat{G}(X, Z), \hat{G}(X, Z) \geq 0 \text{ for } (X, Z) \in \Gamma$$

where $P = D_1G(X^*, 0)$ is an M-matrix (the off-diagonal elements of P are nonnegative) and Γ is the region where the dynamical system makes biological sense.

If the dynamical system (3) satisfies the above two conditions the following theorem holds.

Theorem 4: The fixed point $E_0 = (X_1^*, 0)$ is a globally asymptotic stable (G.A.S) of disease-free steady state of the dynamical system (3) provided that it is Locally Asymptotically Stable (L.A.S) and that (H_1) and (H_2) are satisfied.

Proof:

$$\text{Let } X = (S_u, S_e, R), Z = (E, I_u, I_a, C_u, C_a), X \in \mathfrak{R}^3, Z \in \mathfrak{R}^5, E_0 = (X^*, 0) \text{ and } X^* = (S_u^*, S_e^*, 0)$$

Thus, the uninfected compartments from the dynamical system (3), it gives:

$$H(X, Z) = \begin{bmatrix} \nu + \lambda_6 R + (1 - \rho) \Lambda - \nu \rho_1 (C_u + C_a) - \nu \rho_2 R - (\eta_2 + \omega_1 + \lambda + \mu) S_u \\ \omega_1 S_u + \rho \Lambda + \lambda_7 R - (\lambda(1 - \kappa) + \eta_1 + \mu) S_e \\ (1 - \rho_3) \lambda_2 I_u + (1 - \rho_4) \lambda_3 I_a + \lambda_4 C_u + (u_1 + \lambda_5) C_a + \eta_1 S_e + \eta_2 S_u + \nu \rho_2 R - (\lambda_6 + \lambda_7 + \mu) R \end{bmatrix} \quad (11)$$

Evaluating (11) at the disease-free steady state, it gives:

$$H(X, 0) = \begin{bmatrix} \nu + (1 - \rho) \Lambda - (\eta_2 + \omega_1 + \mu) S_u \\ \omega_1 S_u + \rho \Lambda - (\eta_1 + \mu) S_e \\ 0 \end{bmatrix} \quad (12)$$

Taking the first entry in (12), it gives:

$$\frac{dS_u}{dt} = \nu + (1 - \rho) \Lambda - (\eta_2 + \omega_1 + \mu) S_u \quad (13)$$

Solving the linear equation in (13), it gives:

$$S_u(t) = S_u^0 e^{-(\eta_2 + \omega_1 + \mu)t} + \frac{\nu + (1 - \rho) \Lambda}{(\eta_2 + \omega_1 + \mu)} (1 - e^{-(\eta_2 + \omega_1 + \mu)t}) \quad (14)$$

Where S_u^0 is an arbitrary constant, taking the limit of equation (14) as $t \rightarrow \infty$ it gives

$$S_u(t) \rightarrow \frac{\nu + (1 - \rho) \Lambda}{(\eta_2 + \omega_1 + \mu)} \quad (15)$$

Taking the second entry in (12), it gives:

$$\frac{dS_e}{dt} = \rho \Lambda - (\eta_1 + \mu) S_e \quad (16)$$

Solving the linear equation in (16), it gives:

$$S_e(t) = S_e^0 e^{-(\eta_1 + \mu)t} + \frac{\rho \Lambda}{(\eta_1 + \mu)} (1 - e^{-(\eta_1 + \mu)t}) \quad (17)$$

Where S_e^0 is an arbitrary constant, taking the limit of equation (17) as $t \rightarrow \infty$ it gives

$$S_e(t) \rightarrow \frac{\rho \Lambda}{(\eta_1 + \mu)}$$

Taking the infected compartments of the dynamical system (3), it gives:

$$G(X, Z) = \begin{bmatrix} \lambda(1 - \kappa) S_e + \lambda S_u - ((1 - \lambda_1) + \lambda_1 + \mu) E \\ \lambda_1 E - (\omega_2 + \rho_3 \lambda_2 + (1 - \rho_3) \lambda_2 + \mu) I_u \\ \omega_2 I_u + (1 - \lambda_1) E - (\rho_4 \lambda_3 + (1 - \rho_4) \lambda_3 + \mu) I_a \\ \rho_3 \lambda_2 I_u + \nu \rho_1 C_u - (\lambda_4 + \omega_3 + \mu + \delta) C_u \\ \rho_4 \lambda_3 I_a + \omega_3 C_u + \nu \rho_1 C_a - (u_1 + \lambda_5 + \mu + \delta) C_a \end{bmatrix} \quad (18)$$

Taking the partial derivatives of the state variables in an infected compartments and evaluating equation (18) at disease-free steady state, it gives:

$$P = \begin{bmatrix} (1-\lambda_1)+\lambda_1+\mu & \beta(1-\varepsilon_1)((1-\kappa)S_e^*+S_u^*) & \beta(1-\varepsilon_1)((1-\kappa)S_e^*+S_u^*) & \beta\theta_1(1-\varepsilon_1)((1-\kappa)S_e^*+S_u^*) & \beta\theta_2(1-\varepsilon_1)((1-\kappa)S_e^*+S_u^*) \\ \lambda_1 & -(\omega_2+\rho_3\lambda_2+(1-\rho_3)\lambda_2+\mu) & 0 & 0 & 0 \\ (1-\lambda_1) & \omega_2 & -(\rho_4\lambda_3+(1-\rho_4)\lambda_3+\mu) & 0 & 0 \\ 0 & \rho_3\lambda_2 & 0 & \nu\rho_1-(\lambda_4+\omega_3+\mu+\delta) & 0 \\ 0 & 0 & \rho_4\lambda & \omega_3 & \nu\rho_1-(u_1+\lambda_5+\mu+\delta) \end{bmatrix} \tag{19}$$

Multiply equation (19) with $Z = (E, I_u, I_a, C_u, C_a)^T$, it gives:

$$PZ = \begin{bmatrix} ((1-\lambda_1)+\lambda_1+\mu)E + \lambda(1-\kappa)S_e + \lambda S_u \\ \lambda_1 E - (\omega_2 + \rho_3\lambda_2 + (1-\rho_3)\lambda_2 + \mu)I_u \\ (1-\lambda_1)E + \omega_2 I_u - (\rho_4\lambda_3 + (1-\rho_4)\lambda_3 + \mu)I_a \\ \rho_3\lambda_2 I_u + (\nu\rho_1 - (\lambda_4 + \omega_3 + \mu + \delta))C_u \\ \rho_4\lambda_3 I_a + \omega_3 C_u + (\nu\rho_1 - (u_1 + \lambda_5 + \mu + \delta))C_a \end{bmatrix} \tag{20}$$

Thus, subtracting (18) from (20), it gives:

$$G(X, Z) = PZ - \hat{G}(X, Z) = \begin{bmatrix} \lambda \left[(1-\kappa)(S_e - S_e^*) + (S_u - S_u^*) \right] \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{21}$$

Thus rewriting (21) gives:

$$\hat{G}(X, Z) = \begin{bmatrix} \lambda \left[(1-\kappa)(S_e - S_e^*) + (S_u - S_u^*) \right] \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}$$

Since $0 \leq \kappa \leq 1$, $S_e > S_e^*$ and $S_u > S_u^*$, then, $\hat{G}(X, Z) \geq 0$. Hence, the global stability of E_0 of the dynamical system (3) is proven.

4. Numerical Simulations

The numerical simulation of the study considered parameter values from the published articles and some are assumed with feasible range values given in Table 1 and Table 2. The primary focus of the study was incorporated three combined control interventions on the dynamics of HBV. The impact of vaccination, treatment and media campaign were simulated either separately or as combine interventions

Table 1: State Variables

Symbol	Value	References
$S_u(t)$	8000	Assumed
$S_e(t)$	8000	Assumed
$E(t)$	2000	Assumed

$I_u(t)$	2000	Assumed
$I_a(t)$	2000	Assumed
$C_u(t)$	1000	Assumed
$C_a(t)$	1000	Assumed
$R(t)$	1000	Assumed
$N(t)$	25000	Assumed

Table 2: Parameters

Symbol	Dimension	Values	References
Λ	Year ⁻¹	5000	Assumed
ν	Year ⁻¹	0.0121	Zhang <i>et al.</i> (2012)
β	Year ⁻¹	0.8–20.49	Pang <i>et al.</i> (2010)
θ_1	Year ⁻¹	0-1	Pang <i>et al.</i> (2010)
θ_2	Year ⁻¹	0-1	Pang <i>et al.</i> (2010)
ε_1	Dimensionless	0-1	Assumed
κ	Dimensionless	0-1	Assumed
λ	Year ⁻¹	0-1	Assumed
λ_1	Year ⁻¹	6	Zou <i>et al.</i> (2010)
λ_2	Year ⁻¹	4	Zou <i>et al.</i> (2010)
λ_3	Year ⁻¹	4	Zou <i>et al.</i> (2010)
λ_4	Year ⁻¹	0.025	Zou <i>et al.</i> (2010)
λ_5	Year ⁻¹	0.025	Zou <i>et al.</i> (2010)
λ_6	Year ⁻¹	0.03–0.06	Pang <i>et al.</i> (2010)
ρ	Year ⁻¹	0.2	Estimated
$(1-\rho)$	Year ⁻¹	0.8	Estimated
ρ_1	Year ⁻¹	0.11	Bhattacharyya <i>et al.</i> (2010)
ρ_2	Year ⁻¹	0.1	Bhattacharyya <i>et al.</i> (2010)
ρ_3	Year ⁻¹	0.3	Assumed
$(1-\rho_3)$	Year ⁻¹	0.7	Assumed
ρ_4	Year ⁻¹	0.2	Assumed
$(1-\rho_4)$	Year ⁻¹	0.8	Assumed
ω_1	Year ⁻¹	0–0.75	Estimated
ω_2	Year ⁻¹	0–1	Estimated
ω_3	Year ⁻¹	0–1	Estimated

η_1	Year ⁻¹	0 - 1	Pang <i>et al.</i> (2010)
η_2	Year ⁻¹	0 - 1	Pang <i>et al.</i> (2010)
u_1	Year ⁻¹	0 - 1	Assumed
δ	Year ⁻¹	0.007	Abdulrahman <i>et al.</i> (2013a)
μ	Year ⁻¹	0.011	Abdulrahman <i>et al.</i> (2013a)



Experiment 1: Impact of media campaign only

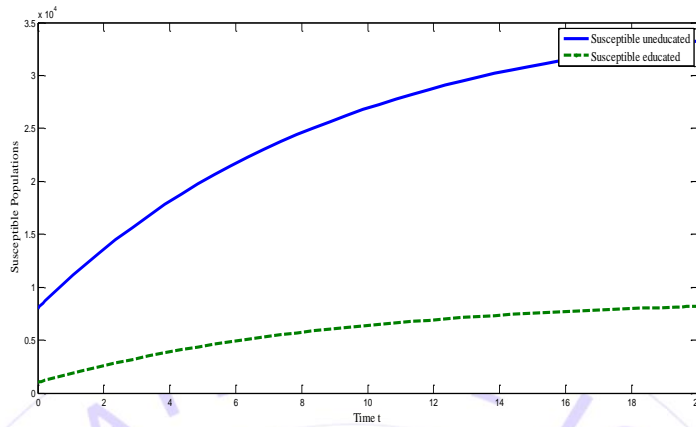


Figure 2a: Plot of Susceptible population without media campaign at rate $\epsilon_1 = 0$

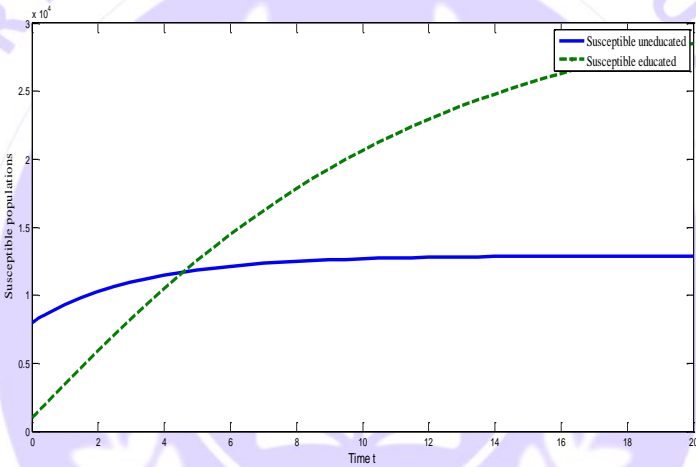


Figure 2b: Plot of Susceptible population with media campaign at rate $\epsilon_1 = 0.2$

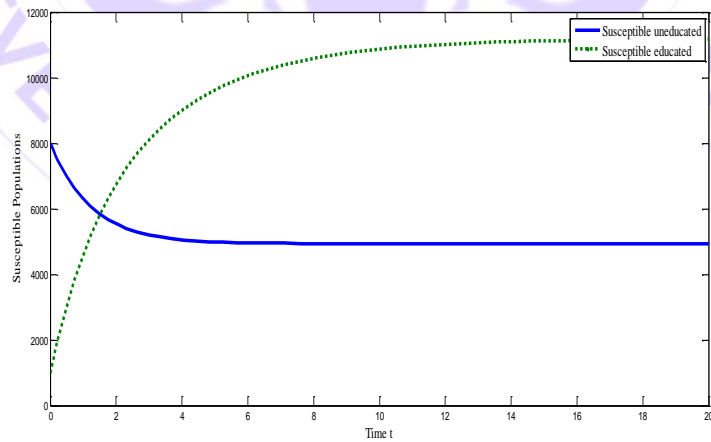


Figure 2c: Plot of Susceptible population with media campaign at rate $\epsilon_1 = 0.5$

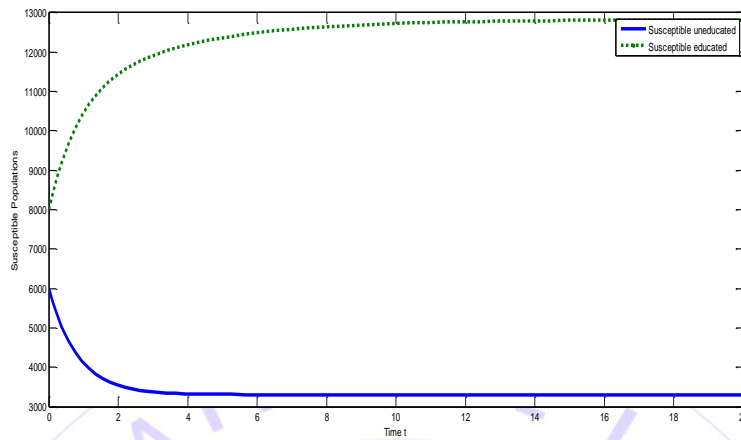


Figure 2d: Plot of Susceptible population with media campaign at $\epsilon_1 = 0.9$

Experiment 2: Impact of media campaign and vaccination

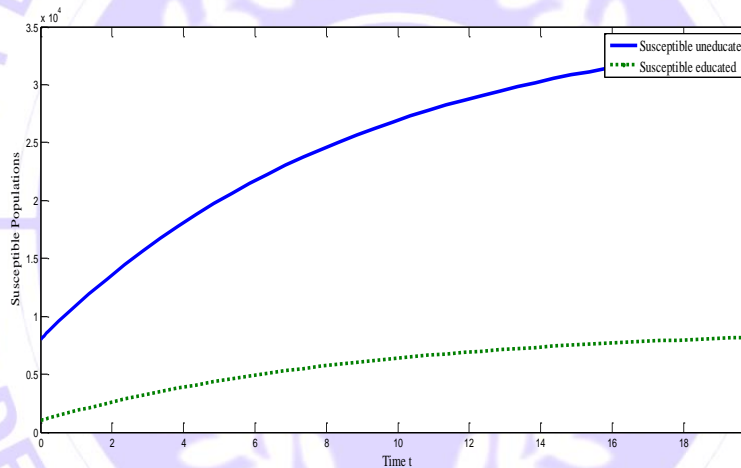


Figure 2e: Plot of Susceptible population without vaccination and media campaign at $\eta_1, \eta_2, \epsilon_1 = 0$

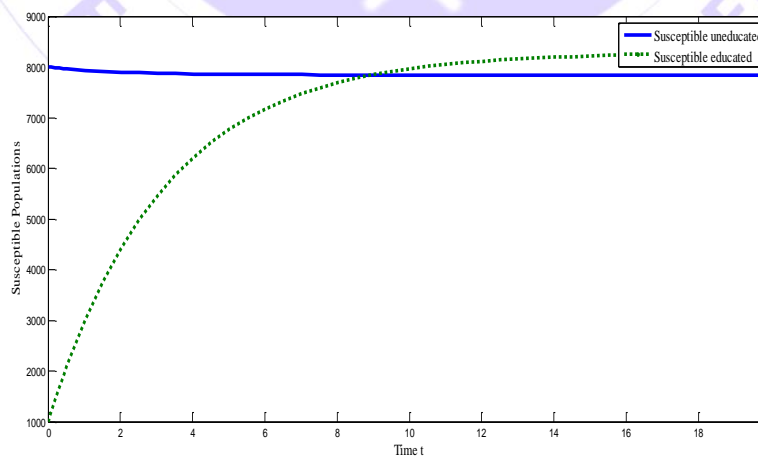


Figure 2f: Plot of Susceptible population with vaccination and media campaign at rate $\eta_1, \eta_2, \epsilon_1 = 0.2$

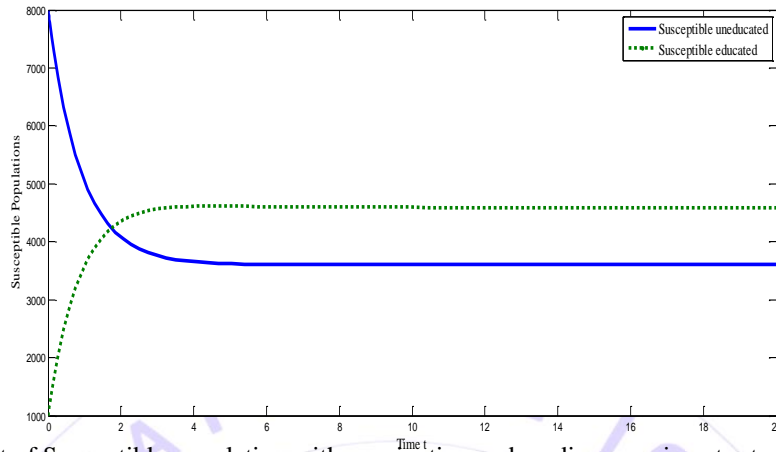


Figure 2g: Plot of Susceptible population with vaccination and media campaign at rate $\eta_1, \eta_2, \epsilon_1 = 0.5$

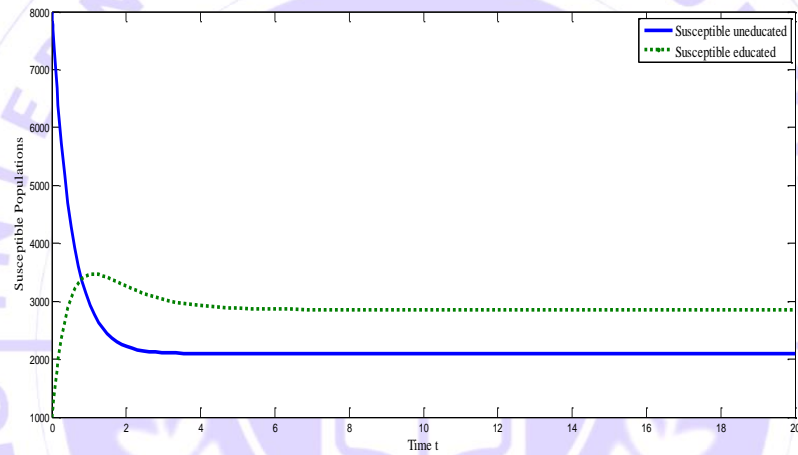


Figure 2h: Plot of Susceptible populations with vaccination and media campaign at rate $\eta_1, \eta_2, \epsilon_1 = 0.9$

Experiment 3: Impact of vaccination, treatment and media campaign

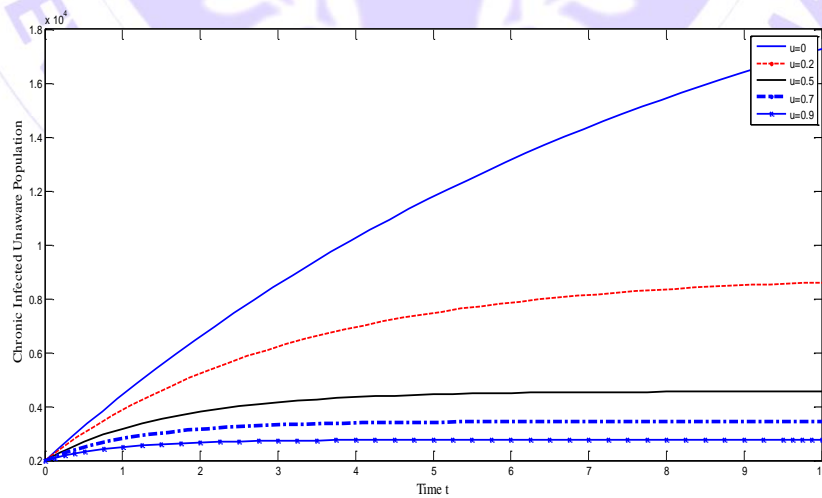


Figure 2i: Plot of Chronic Unaware Population with media campaign at various rates

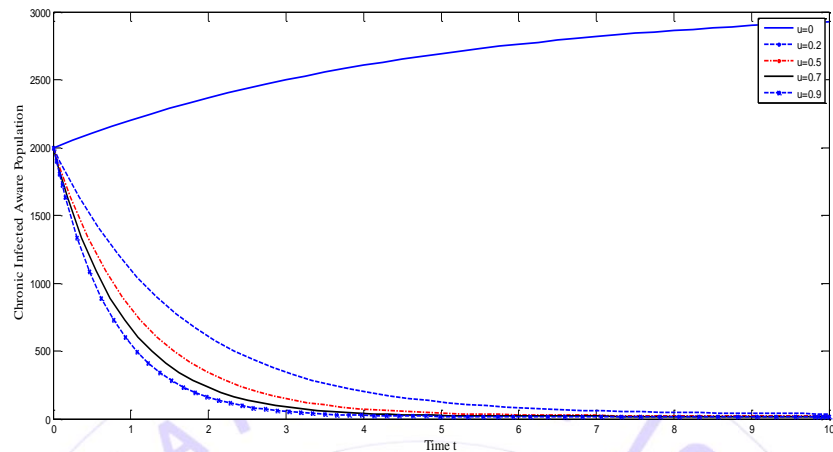


Figure 2j: Plot of Chronic Aware Population with treatment and media campaign at various rates

5. Discussion

In the study, the analytical solutions, numerical simulations and survey results were presented. In the numerical simulations, from Figures 2a, 2b, 2c and 2d; the study simulated the susceptible population of uneducated and educated with different rate of media campaign. At figure 2a, where there is absence of media campaign, the population of both susceptible uneducated and educated rise slightly to their equilibrium levels. At figure 2b, 2c and 2d; population of susceptible educated rises sharply while that of susceptible uneducated falls steadily; this happened due to increase in the rate of media campaign. The trend of reducing susceptible population by implementing the strategy of educating susceptible population was observed to have long run benefit. Hence, the benefit of media campaign is obviously portrayed in this study which is synonymous to Khan *et al.* (2018).

From Figure 2e, 2f, 2g and 2h; the study simulated the susceptible population of uneducated and educated with different rate of two combined interventions of vaccination and media campaign. At figure 2e, where there was absence of both vaccination and media campaign, the population of both susceptible uneducated and educated rise slightly to their equilibrium level. In the presence of the two combine interventions with lower effort, the population of susceptible educated at onset rises sharply and suddenly grows steadily, while the population of susceptible uneducated falls steadily as depicted in figure 2f. At figure 2g, the effort of the two combine interventions was increased; which portrayed a sharp decline in the population of susceptible uneducated and truncated the sharper grows of the population of susceptible educated. As more and more effort of the two combine interventions was increased, there is always a corresponding sharper decline in the population of susceptible uneducated than the population of susceptible educated as depicted in Figure 2h. The scenario of two combined interventions of vaccination and media campaign has a greater return than in vaccination and treatment as pointed out by Kamyad *et al.* (2014) and Emerenini and Inyama (2017)

From Figure 2i, the study simulated the population of chronic unaware with different rates of vaccination and no media campaign. The population of chronic unaware growth was slowly truncated as more and more effort of vaccination is imposed. On the contrary, From Figure 2j, the study simulated the population of chronic aware with different rates of treatment, higher rates of vaccination and media campaign. The chronic aware population grows steadily at no treatment level, but decline sharply as treatment began. The decline in the population continue as higher effort on treatment is imposed. Hence, the combined three interventions have higher impact than the two interventions or a single intervention as depicted in Figure 2i. As such, in this study, combined three interventions is the best control strategy for curbing the menace of HBV similar to Chan *et al.* (2022). On the contrary, Khan *et al.* (2018) considered educational campaign, vaccination and the media coverage as three control functions.

This study has some limitations. The mathematical model developed and studied didn't capture sanitation, variable recruitment and immigration. Also, the mathematical model developed is an integer differential equation not fractional differential equation.

6. Conclusion

A new version of compartmental model of HBV was proposed. The basic properties of the model such invariant region and positivity was established. The disease-free steady state existence was also established. The disease-free steady state is locally as well as globally asymptotically stable. The numerical experiments on the dynamics of susceptible, chronic unaware and chronic aware populations with the impact of vaccination, treatment and media campaign were performed. The numerical results established that three combined intervention strategy was much better and reduces the menace of HBV speedily and drastically. Lastly, the study suggested that more effective campaign efforts should be supplemented with other major control strategies in order to get substantial paybacks in curbing the menace of HBV.

6. Conflicts of Interest

This study was funded by Tertiary Education Trust Fund (TETFUND) in Nigeria. The conduct of the study was approved on Taraba State, Nigeria via Institutional-Based Research (IBR) scheme.

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