

Journal of Science and Technology Research

Journal homepage: www.nipesjournals.org.ng



Optimal Test Strategies for HBV-HIV Co-Infection Model

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ARTICLE INFORMATION

Article history: Received 26 February 2019 Revised 9 March 2019 Accepted 15 March 2019 Available online 25 March 2019

Keywords: Vaccination Treatment Optimal Control Co-interaction

ABSTRACT

In this paper, we present a deterministic model for the co-interaction of HBV and HIV in a population. Mathematical analyses are carried out, which determines the positivity of solution and optimal control. This study examined the effectiveness of the efforts put in place in eliminating the birth of prenatally infected chronic carrier mother, considering the efficacy of prophylactic vaccine against the incidence of new cases. The scheme shows that the disease can be resisted with vaccination(s) and treatment.

1. Introduction

Hepatitis B virus infection is fifty to one hundred times more transmissible than HIV [1]. The management of HIV-HBV co-infection is difficult by the use of drugs with action against both viruses, the risk of flares and hepatic decompensation with immune reconstitution, and the increasing incidence of antiviral resistance. Due to shared modes of transmission, co-infection is general and an estimated 4 million people worldwide are co-infected with HBV-HIV. Some drugs used to treat HIV are poisonous to the liver, which may amount to more damage from the hepatitis B infection [7]. Individuals co-infected with both hepatitis B and HIV are 14 to 17 times more likely to die than those with hepatitis B alone [6].

Hepatitis B is a dynamic disease and it is imperative to understand its virology and natural history in other to reduce complications and limit the progression of disease. The use of drugs with activity against both viruses (HIV-HBV co-infection), the risk of flares and hepatic decompensation with immune reconstitution, and the increasing prevalence of antiviral resistance complicate the management of HIV - HBV co - infection. Forty million people around the world are infected with HIV. To make things worse, certain medicines used to treat HIV are toxic to the liver that may already be damaged by the infection with hepatitis B. In this study, a realistic mathematical model for HBV - HIV co - infection is formulated and analyzed, incorporating the key epidemiological and biological characteristics of each of the two diseases. This study's main contribution is to conduct an optimal test of the resulting model.

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2. Mathematical Model Formulation

The model is constructed based on the HBV/HIV transmission framework and the model proposed in [6]. The population is divided into thirteen classes based on epidemiological status; a coinfections models of susceptible individuals of both diseases (S (t)). Infected individuals in the asymptomatic stage of HIV infection (H_1 (t)). HIV-infected individuals that exhibit clinical symptoms of AIDS (H_2 (t)). Dually-infected individuals with HBV acute infection, in the asymptomatic acute stage of HIV infection (H_{1I} (t)). Dually-infected individuals with HBV acute infection, displaying symptoms (symptomatic) of AIDS (H_{2I} (t)). Dually-infected individuals with HBV chronic infection, in the asymptomatic stage of HIV infection (H_{1C} (t)). Dually-infected individuals with HBV chronic infection, displaying symptoms of AIDS (H_{2C} (t)). HBV recovered individuals with protective immunity in the asymptomatic stage of HIV infection (H_{1R} (t)). HBV recovered individuals (V (t)). Individuals with HBV acute infection (H_{1R} (t)). HBV vaccinated individuals (V (t)). Individuals with HBV acute infection (H_{1R} (t)). HBV chronic carriers (H_{1R} (t)) and HBV recovered with protective immunity (H_{1R} (t)).

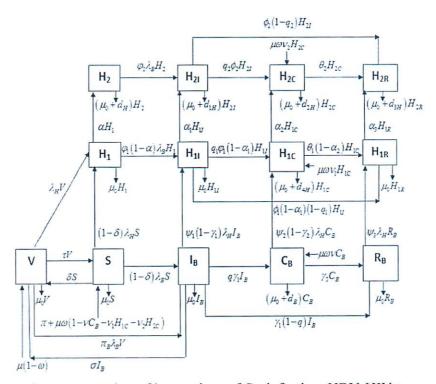


Figure 1: Schematic representation of interactions of Co-infection, HBV-HIV transmission

$$\frac{dS}{dt} = \pi + \mu\omega \left(1 - \nu C_B - \nu_1 H_{1C} - \nu_2 H_{2C}\right) + \tau V - \left(1 - \delta\right) \left(\lambda_H + \lambda_B\right) S - \left(\mu_0 + \delta\right) S \tag{1}$$

$$\frac{dV}{dt} = \mu (1 - \omega) + \delta S + \sigma I_B - (\lambda_H + \pi_B \lambda_B) V - (\mu_0 + \tau) V \tag{2}$$

$$\frac{dH_1}{dt} = \left(V + (1 - \delta)S\right)\lambda_H - \varphi_1(1 - \alpha)\lambda_B H_1 - (\mu_0 + \alpha)H_1, \tag{3}$$

$$\frac{dH_2}{dt} = \alpha H_1 - \varphi_2 \lambda_B H_2 - (\mu_0 + d_H) H_2 \quad , \tag{4}$$

$$\frac{dH_{1I}}{dt} = \varphi_1 (1 - \alpha) \lambda_B H_1 + \psi_1 (1 - \gamma_1) \lambda_H I_B - (\mu_0 + \phi_1 (1 - \alpha_1)) H_{1I},
\frac{dH_{2,I}}{dt} = \varphi_2 \lambda_B H_2 + \alpha_1 H_{1,I} - (\mu_0 + d_{1H} + \phi_2) H_{2,I},$$
(5)

$$\frac{-dt}{dt} = \psi_2 \lambda_B I_2 + u_1 I_{1,I} - (\mu_0 + u_{1H} + \psi_2) I_{2,I},$$
(6)

$$\frac{dH_{1,C}}{dt} = \mu\omega v_1 H_{1,C} + q_1 \phi_1 (1 - \alpha_1) H_{1,I} + \psi_2 (1 - \gamma_2) \lambda_H C_B - (\mu_0 + d_{4H} + \alpha_2 + \theta_1 (1 - \alpha_2)) H_{1C}$$
(7)

$$\frac{dH_{2,C}}{dt} = \mu\omega v_2 H_{2,C} + q_2 \phi_2 H_{2,I} + \alpha_2 H_{1,C} - (\mu_0 + d_{2H} + \theta_2) H_{2C} ,$$
(8)

$$\frac{dH_{1,R}}{dt} = \phi_1 (1 - \alpha_1) (1 - q_1) H_{1,I} + \theta_1 (1 - \alpha_2) H_{1,C} + \psi_3 \lambda_H R_B - (\mu_0 + \alpha_3) H_{1R} , \qquad (9)$$

$$\frac{dH_{2R}}{dt} = \phi_2 (1 - q_2) H_{2I} + \theta_2 H_{2C} + \alpha_3 H_{1R} - (\mu_0 + d_{3H}) H_{2R} , \qquad (10)$$

$$\frac{dI_B}{dt} = \left(\pi_B V + (1 - \delta)S\right) \lambda_B - \psi(1 - \gamma_1) \lambda_H I_B - \left(\mu_0 + \sigma + \gamma_1\right) I_B \tag{11}$$

$$\frac{dC_B}{dt} = \mu\omega v C_B + q\gamma_1 I_B - \psi_2 (1 - \gamma_2) \lambda_H C_B - (\mu_0 + d_B + \gamma_2) C_B$$
(12)

$$\frac{dR_B}{dt} = (1 - q)\gamma_1 I_B + \gamma_2 C_B - \psi_3 \lambda_H R_B - \mu_0 R_B \tag{13}$$

Where,

$$\lambda_{B} = \frac{\beta_{B} \left(I_{B} + \eta_{1} \left(H_{1I} + \eta_{1h} H_{2I} \right) + \eta \left(C_{B} + \eta_{1c} H_{1C} + \eta_{2c} H_{2C} \right) \right)}{N}$$

$$\lambda_{H} = \frac{\beta_{H} \left(H_{1} + \varepsilon H_{2} + \varepsilon_{1} \left(H_{1I} + \varepsilon_{1h} H_{2I} \right) + \varepsilon_{2} \left(H_{1C} + \varepsilon_{2h} H_{2C} \right) + \varepsilon_{3} \left(H_{1R} + \varepsilon_{3h} H_{2R} \right) \right)}{N}$$

Table 1: The parameters used

Parameter	Interpretation	Value	Reference
μ	Birth rate in the population	0.0121	[5]
μ_0	Natural mortality rate	0.00693	[5]
μ_{l}	HBV related mortality rate	0.007	[4]
ω	Proportion of births without successful vaccination	0 - 100 percent	[2]
σ	Vaccination rate of acute HBV infectious class I _B	Hypothetical	
$\beta_{\!\scriptscriptstyle B}$	Effective contact rate for HBV	0.4	[5]
$\beta_{\!\scriptscriptstyle H}$	Effective contact rate for HIV	0.03	[5]
π	The recruitment rate of susceptible individuals in the population	500	[5]
$\pi_{_B}$	Rate of waning of vaccine	0.1	[5]
τ	The rate of relapse of vaccine induced immunity	Hypothetical	
φ_1 and φ_2	Probabilities of acquiring HBV infection of individuals in H ₁ andH ₂	0.3, 0.5	[5]

ψ_1, ψ_2 and ψ_3	Probabilities of acquiring HIV infection of individuals in I_B , C_B and R_B	0.4, 0.5, 0.1	[5]
v, v_1 and v_2	The proportion of perinatal infected born by carrier mothers in the C_B , H_{1C} and H_{2C} classes	0.11, 0.12, 0.13	[5]
γ_1, ϕ_2 and ϕ_3	Rate of moving from I_B , H_{11} and H_{21} classes to C_B , H_{1C} and H_{2C} classes	4, 2, 3	[5]
q, q_2 and q_3	The average probability of moving from I_B , H_{11} and H_{21} classes fail to clear an acute state and develops a carrier state C_B , H_{1C} and H_{2C} classes	0.885, 0.9, 0.95	[5]
γ_2, θ_1 and θ_2	Rate of moving from C_B , H_{1C} and H_{2C} Classes for R_B , H_{1R} and H_{2R} classes	0.025, 0.05, 0.06	[5]
$d_{\scriptscriptstyle B}$	HBV induced death rate in C _B class	0.002	[5]
$d_{H}, d_{1H},$ d_{2H}, d_{3H}	HIV induced death rate of individuals in H ₂ , H _{1I} , H _{2I} , H _{1C} , H _{2C} and H _{2R} classes	0.01, 0.03, 0.05, 0.02, 0.06	[5]
and $d_{_{4H}}$ δ	The vaccination rate	0.75	[5]
$\eta_1, \eta_{1h}, \eta,$ η_{1C} and η_{2C}	modification parameters of the relative infectiousness of individual in the $C_B,H_{11},H_{21},H_{1C},H_{2C}$	1.1, 1.2, 0.16, 1.3, 1.4	[5]
$\varepsilon, \varepsilon_1, \varepsilon_{1h}, \\ \varepsilon_2, \varepsilon_{2h}, \varepsilon_3,$	modification parameters of the relative infectiousness of individual in the H_{11} , H_{21} , H_{2} , H_{1C} , H_{2C} , H_{1R} and, H_{2R}	1.2, 1.25, 1.1, 1.5, 1.55, 1.3, 1.35	[5]
\mathcal{E}_{2h} , \mathcal{E}_{3h}			
$\alpha, \alpha_1, \alpha_2,$ and α_3	Rate of moving from H_1 , H_{1I} , H_{1C} and H_{1R} classes to H_2 , H_{2I} , H_{2C} and H_{2R} classes	0.0303, 0.04, 0.16, 0.05, 0.06	[5]

The total population N (t) can be obtained from

$$N(t) = S(t) + V(t) + H_1(t) + H_2(t) + H_{1I}(t) + H_{2I}(t) + H_{1C}(t) + H_{2C}(t) + H_{1R}(t) + H_{2R}(t)$$
$$+ I_B(t) + C_B(t) + R_B(t)$$

(14)

Here, it is important to note that in the absence of the disease $N(t) \to \frac{\pi}{\mu_0}$ Moreover, under the

dynamics

described by the above systems of equations, the region

$$\Omega = \begin{cases} x = \left(S(t), V(t), H_1(t), H_2(t), H_{1I}(t), H_{2I}(t), H_{1C}(t), H_{2C}(t), H_{1R}(t), H_{2R}(t), I_B(t), C_B(t), N\right) \in \Re_+^{13} \mid \\ S \ge 0, V \ge 0, H_1 \ge 0, H_2 \ge 0, H_{1I} \ge 0, H_{2I} \ge 0, H_{1C} \ge 0, H_{2C} \ge 0, H_{1R} \ge 0, H_{2R} \ge 0, I_B \ge 0, C_B \ge 0, N \le \frac{\mu + \pi}{\mu_0} \end{cases}$$

(15)

Is positively invariant. Hence the system is both mathematically and epidemiologically well-posed. Therefore, for initial starting point $x \in \mathfrak{R}^{13}_+$, the trajectory lies in Ω . Thus we restrict our analysis to the region Ω . (Where the models make biological sense)

Lemma 1 All the solution of the Equations 2.1 -2.13 are positive for all time $t \ge 0$ provided the initial condition are positive.

Proof: Let

$$(S(0), V(0), H_1(0), H_2(0), H_{1I}(0), H_{2I}(0), H_{1C}(0), H_{2C}(0), H_{1R}(0), H_{2R}(0), I_B(0), C_B(0), N) \in \mathfrak{R}^{13}_+$$

$$\frac{dS}{dt} = \pi + \mu\omega \left(1 - \nu C_B - \nu_1 H_{1C} - \nu_2 H_{2C}\right) + \tau V - \left(1 - \delta\right) \left(\lambda_H + \lambda_B\right) S - \left(\mu_0 + \delta\right) S \tag{16}$$

$$\geq -(1-\delta)(\lambda_H + \lambda_R)S - (\mu_0 + \delta)S \tag{17}$$

This implies,

$$S'(t) \ge -(1-\delta)(\lambda_{\mathcal{H}} + \lambda_{\mathcal{B}})S - (\mu_0 + \delta)S \tag{18}$$

integrating we have

similarly it can be shown that

$$S \geq 0, V \geq 0, \ H_1 \geq 0, H_2 \geq 0, H_{1I} \geq 0, H_{2I} \geq 0, H_{1C} \geq 0, H_{2C} \geq 0, H_{1R} \geq 0, H_{2R} \geq 0, I_B \geq 0, C_B \geq 0,$$

for all time t > 0. Hence all solutions of the HBV model remain positive for all non-negative initial conditions.

3. Optimal Control Modeling

$$\frac{dS}{dt} = \pi + \mu\omega \left(1 - \nu(1 - u_0)C_B - \nu_1(1 - u_1)H_{1C} - \nu_2(1 - u_2)H_{2C}\right) + \tau V - (1 - \delta)(\lambda_H + \lambda_B)S - (\mu_0 + \delta)S(19)$$

$$\frac{dV}{dt} = \mu (1 - \omega) + \delta S + \sigma I_B - (\lambda_H + \pi_B \lambda_B) V - (\mu_0 + \tau) V \tag{20}$$

$$\frac{dH_1}{dt} = \left(V + (1 - \delta)S\right)\lambda_H - \varphi_1(1 - \alpha)(1 - u_3)\lambda_B H_1 - (\mu_0 + \alpha)H_1, \tag{21}$$

$$\frac{dH_2}{dt} = \alpha H_1 - \varphi_2 \lambda_B H_2 - (\mu_0 + d_H) H_2 \quad , \tag{22}$$

$$\frac{dH_{1I}}{dt} = \varphi_1 (1 - \alpha) (1 - u_3) \lambda_B H_1 + \psi_1 (1 - \gamma_1) \lambda_H I_B - (\mu_0 + \phi_1 (1 - \alpha_1)) H_{1I}, \qquad (23)$$

$$\frac{dH_{2,I}}{dt} = \varphi_2 \lambda_B H_2 + \alpha_1 H_{1,I} - (\mu_0 + d_{1H} + \phi_2) H_{2,I} , \qquad (24)$$

$$\frac{dH_{1,C}}{dt} = \mu\omega V_1 (1 - u_1) H_{1,C} + q_1 \phi_1 (1 - \alpha_1) H_{1,I} + \psi_2 \rho_2 \lambda_H C_B - (\mu_0 + d_{4H} + \alpha_2 + \theta_1 (1 - \alpha_2)) H_{1C}, \qquad (25)$$

$$\frac{dH_{2C}}{dt} = \mu\omega v_2 (1 - u_2) H_{2C} + q_2 \phi_2 H_{2I} + \alpha_2 H_{1C} - (\mu_0 + d_{2H} + \theta_2) H_{2C} , \qquad (26)$$

$$\frac{dH_{1,R}}{dt} = \phi_1 (1 - \alpha_1) (1 - q_1) H_{1,I} + \theta_1 (1 - \alpha_2) H_{1,C} + \psi_3 \lambda_H R_B - (\mu_0 + \alpha_3) H_{1R} , \qquad (27)$$

$$\frac{dH_{2R}}{dt} = \phi_2 (1 - q_2) H_{2I} + \theta_2 H_{2C} + \alpha_3 H_{1R} - (\mu_0 + d_{3H}) H_{2R} , \qquad (28)$$

$$\frac{dI_B}{dt} = \left(\pi_B V + (1 - \delta)S\right) \lambda_B - \psi(1 - \gamma_1) \lambda_H I_B - \left(\mu_0 + \sigma + \gamma_1\right) I_B \tag{29}$$

$$\frac{dC_{B}}{dt} = \mu\omega\nu(1 - u_{0})C_{B} + q\gamma_{1}I_{B} - \psi_{2}(1 - \gamma_{2})\lambda_{H}C_{B} - (\mu_{0} + d_{B} + \gamma_{2})C_{B}$$
(30)

$$\frac{dR_B}{dt} = (1 - q)\gamma_1 I_B + \gamma_2 C_B - \psi_3 \lambda_H R_B - \mu_0 R_B \tag{31}$$

Here, the control parameters employed in the above model; U_0 , U_1 , U_2 and U_3 are the effective rate of professional effort that the medical expatriate put in place in prevention or eradicate of mother to child transmission in the HBV carrier class, dually infected (HBV-HIV) asymptomatic carrier class, dually infected (HBV-HIV) symptomatic carrier class respectively while U_3 is the treatment rate of $H_{1,1}$ dually infected (HBV/HIV) asymptomatic acute class. Where

$$z_{0} = \mu_{0} + \delta, z_{1} = \mu_{0} + \tau, z_{2} = \mu_{0} + \alpha, z_{3} = \mu_{0} + d_{H}, z_{4} = \mu_{0} + \phi_{1}(1 - \alpha_{1}), z_{5} = \mu_{0} + d_{1H} + \phi_{2},$$

$$z_{6} = q_{1}\phi_{1}(1 - \alpha_{1}), z_{7} = \psi_{2}(1 - \gamma_{2}), z_{8} = \mu_{0} + d_{4H} + \alpha_{2} + \theta_{1}(1 - \alpha_{2}), z_{8} = \mu_{0} + d_{2H} + \theta_{2},$$

$$z_{10} = \phi_{1}(1 - \alpha_{1})(1 - q_{1}), z_{11} = \theta_{1}(1 - \alpha_{2}), z_{12} = \mu_{0} + \alpha_{3}, z_{13} = \phi_{2}(1 - q_{2}), z_{14} = \mu_{0} + d_{3H},$$

$$z_{15} = \psi(1 - \gamma_{1}), z_{16} = \mu_{0} + \sigma + \gamma_{1}, z_{17} = \psi_{2}(1 - \gamma_{2}), z_{18} = \mu_{0} + d_{B} + \gamma_{2}.$$
The objective function;

$$J(u_0, u_1, u_2, u_3) = \int_{t_0}^{t_1} A_1 H_{1C}(t) + A_2 H_{2C}(t) + A_3 C_B(t) + A_4 H_{1I}(t) + \frac{B_1}{2} u_0^2(t) + \frac{B_2}{2} u_1^2(t) + \frac{B_3}{2} u_2^2(t) + \frac{B_4}{2} u_3^2(t) dt$$

$$(32)$$

Using similar control variables and objective function (as in above) we find that the Hamiltonian takes the form;

$$H = A_{1}H_{1C}(t) + A_{2}H_{2C}(t) + A_{3}C_{B}(t) + A_{4}H_{1I}(t) + \frac{B_{1}}{2}u_{0}^{2}(t) + \frac{B_{2}}{2}u_{1}^{2}(t) + \frac{B_{3}}{2}u_{2}^{2}(t) + \frac{B_{4}}{2}u_{3}^{2}(t) + \sum_{i=1}^{10}\lambda_{i}f_{i}$$

$$(33)$$

$$H = A_{1}H_{1C}(t) + A_{2}H_{2C}(t) + A_{3}C_{B}(t) + A_{4}H_{1I}(t) + \frac{B_{1}}{2}u_{0}^{2}(t) + \frac{B_{2}}{2}u_{1}^{2}(t) + \frac{B_{3}}{2}u_{2}^{2}(t) + \frac{B_{4}}{2}u_{3}^{2}(t)$$

$$+\lambda_{4} \left[\alpha H_{1} - \varphi_{2} \lambda_{B} H_{2} - (\mu_{0} + d_{H}) H_{2}\right] + \lambda_{5} \left[\varphi_{1} (1 - \alpha) (1 - u_{3}) \lambda_{B} H_{1} + \psi_{1} (1 - \gamma_{1}) \lambda_{H} I_{B} - (\mu_{0} + \phi_{1} (1 - \alpha_{1})) H_{1I}\right]$$

$$- \left[\mu \omega V_{1} (1 - u_{1}) H_{1C} + q_{1} \phi_{1} (1 - \alpha_{1}) H_{1I} + \psi_{2} \rho_{2} \lambda_{H} C_{B}\right]$$

$$+ \lambda_{6} \left[\varphi_{2} \lambda_{B} H_{2} + \alpha_{1} H_{1,I} - \left(\mu_{0} + d_{1H} + \phi_{2} \right) H_{2,I} \right] + \lambda_{7} \left[\mu \omega \nu_{1} \left(1 - u_{1} \right) H_{1,C} + q_{1} \phi_{1} \left(1 - \alpha_{1} \right) H_{1,I} + \psi_{2} \rho_{2} \lambda_{H} C_{B} \right] - \left(\mu_{0} + d_{4H} + \alpha_{2} + \theta_{1} \left(1 - \alpha_{2} \right) \right) H_{1C}$$

$$+\lambda_{8} \left[\mu\omega V_{2}(1-u_{2})H_{2C}+q_{2}\phi_{2}H_{2I}+\alpha_{2}H_{1C}-(\mu_{0}+d_{2H}+\theta_{2})H_{2C}\right]+\lambda_{9} \left[\frac{(\pi_{B}V+(1-\delta)S)\lambda_{B}-\psi(1-\gamma_{1})\lambda_{H}I_{B}}{-(\mu_{0}+\sigma+\gamma_{1})I_{B}}\right]$$

$$+\lambda_{10} \Big[\mu \omega \nu (1 - u_0) C_B + q \gamma_1 I_B - \psi_2 (1 - \gamma_2) \lambda_H C_B - (\mu_0 + d_B + \gamma_2) C_B \Big]$$
(34)

The adjoint equations;

$$\lambda_{1}' = \left[(1 - \delta)(\lambda_{H} + \lambda_{B}) + Z_{0} \right] \lambda_{1} - \delta \lambda_{2} - (1 - \delta)\lambda_{H}\lambda_{3} - (1 - \delta)\lambda_{B}\lambda_{11}$$

$$(35)$$

$$\lambda_{2}' = -\tau \lambda_{1} + \left[\left(\lambda_{H} + \pi_{B} \lambda_{B} \right) + Z_{1} \right] \lambda_{2} - \lambda_{H} \lambda_{3} - \pi_{B} \lambda_{B} \lambda_{11}$$

$$\tag{36}$$

$$\lambda_{3}' = \left[\varphi_{1} (1 - \alpha) (1 - u_{3}) (\lambda_{3} - \lambda_{5}) \lambda_{B} \right] \lambda_{3} + Z_{2} \lambda_{3} - \alpha \lambda_{4} - \varphi_{1} (1 - \alpha) \lambda_{B} \lambda_{5}$$

$$(37)$$

$$\lambda_{a}' = \left[\varphi_{2}\lambda_{B} + Z_{3}\right]\lambda_{4} - \varphi_{2}\lambda_{B}\lambda_{6} \tag{38}$$

$$\lambda_s' = -A_1 + Z_1 \lambda_s - \alpha_1 \lambda_h + Z_6 \lambda_7 + Z_{10} \lambda_9 \tag{39}$$

$$\lambda_{\kappa}' = Z_{5}\lambda_{\kappa} - q_{2}\phi_{2}\lambda_{\kappa} - Z_{13}\lambda_{10} \tag{40}$$

$$\lambda_{1}' = -A_{1} + \mu\omega v_{1}(1 - u_{1})(\lambda_{1} - \lambda_{2}) + Z_{8}\lambda_{2} - \alpha_{2}\lambda_{8} - Z_{11}\lambda_{9} \tag{41}$$

$$\lambda_{s}' = -A_{2} + \mu\omega v_{2} (1 - u_{2})(\lambda_{1} - \lambda_{s}) + Z_{0}\lambda_{s} - \theta_{2}\lambda_{10} \tag{42}$$

$$\lambda_{0}' = -\sigma \lambda_{2} - \psi_{1} (1 - \gamma_{1}) \lambda_{11} \lambda_{2} + (Z_{15} + Z_{16}) \lambda_{11} - q \gamma_{1} \lambda_{12}$$

$$\tag{43}$$

$$\lambda_{10}' = -A_3 + \mu\omega\nu(1 - u_0)(\lambda_1 - \lambda_{12}) + Z_7\lambda_H\lambda_7 + Z_{17}\lambda_H\lambda_{12} - Z_{18}\lambda_{12}$$
(44)

With transversality conditions $\lambda_i(t_f) = 0$ (i = 1, ..., 10, t_f is the end time). Using the

Hamiltonian, we obtain the optimality conditions;

$$u_0 = \frac{\left(\lambda_1 - \lambda_{12}\right)\mu\omega\nu C_B}{B_1} \tag{45}$$

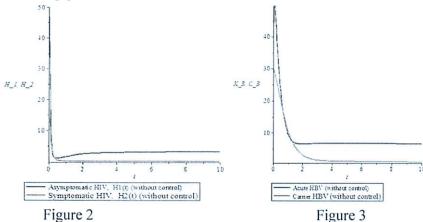
$$u_{\rm l} = \frac{\left(\lambda_{\rm l} - \lambda_{\rm l}\right)\mu\omega\nu_{\rm l}H_{\rm lC}}{B_2} \tag{46}$$

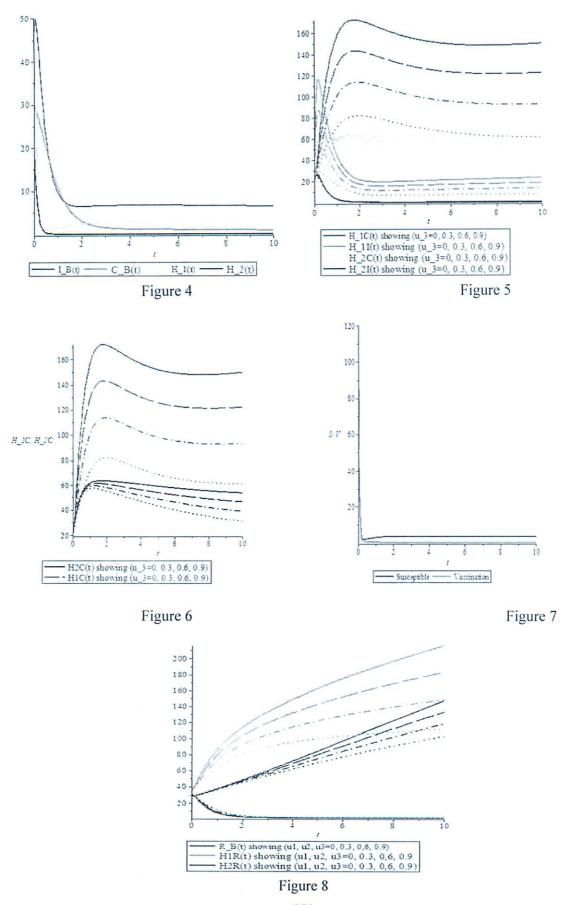
$$u_2 = \frac{\left(\lambda_1 - \lambda_8\right)\mu\omega\nu_2 H_{2C}}{B_3} \tag{47}$$

$$u_3 = \frac{\left(\lambda_3 - \lambda_5\right)\varphi_1\left(1 - \alpha\right)\lambda_B H_1}{B_4} \tag{48}$$

4. Results and Discussion

To obtain further insight into the influence of the control parameters on the behavior of the systems under consideration, thus, we consider the following Figures plotted with the aid of Maple software [7].





In the co-infection model above, the control parameters employed are; U_0 , U_1 , U_2 and U_3 are the effective rate of professional effort that the medical expatriate put in place in prevention or eradicate of mother to child transmission in the HBV carrier class, dually infected (HBV-HIV) asymptomatic carrier class, dually infected (HBV-HIV) symptomatic carrier class respectively. While U_3 is the treatment rate of HIV asymptomatic acute class (H_1) without considering any optimal control measure except the therapeutic vaccination. We considered the state of asymptomatic HIV (H_1) and symptomatic HIV (H_2) (without control) in Figure 2. Similarly, in Figure 3, relationship between acute infection, HBV (I_B) and Carrier HBV (I_B) were considered. Figures 4, harbored Figures 2 and 3. Increasing the controls (Figures 5) from 0, 30, 60 and 90 percents, reduces the dual infection in H_{1C} , H_{11} and H_{2C} respectively.

Thus, H_{1C} show major significant difference in response to the control adopted, while H_{2I} made no difference and die-out. Figures 6 shows the response (decrease) of H_{1C} and H_{2C} with the increase in control measure. Also, H_{1C} registered a major significant difference when compared with H_{2C} . Meanwhile, Figure 7 displays the relationship between the Susceptible and Vaccinated class, which decreases and moves at a constant rate. In addition, the sensitivities of the recovery classes are obtained in Figure 8, where H_{1R} recorded a more pronounced significant difference, also in H_{2R} , however, little or none is noticed with R_B . A similar pattern of some of the results was obtained in [6], however, when comparing with other results in literature we are able to record a significant changes in the behavioral dynamics of the model owing to the sensitive control measure put in place.

5. Conclusion

This scheme has shown that the disease can be resisted with vaccination(s) and treatment. As the results mentioned earlier reveal, with a two-year robust vaccination campaign, the epidemic will be avoided, but the rate drops only to move at a constant rate. However, we must be cognizant of the fact that a two-year campaign does not only eliminate future epidemics, but the routine must be repeated to avoid future occurrence. So the more active our campaign strategies (vaccination(s) and treatments) are, the sooner the future epidemic will be arrested.

6. Conflict of Interest

There is no conflict of interest associated with this work.

References

- [1] Adeoye, G. (2010) Twenty million Nigerians at Risk from Hepatitis B. Retrieved from http://www.plurpol.org/joom/index.php/regional-news/64-africa/6245.
- [2] WHO, Hepatitis B. http://www.who.int/mediacentre/factsheets/fs204/en/ index.htmlS. », Revised August 2008.
- [3] Reza Danaei, Aliasghar Jodayree Akbarfam, Habib Molaei (2015) ODE modeling and Dynamical Stability of HBV/HIVco-infections, IJISET International Journal of Innovative Science, Engineering Technology, 2 (5), 312-315.
- [4] MOHC, (2009). Ministry of Health of the People Republic of China. /http://www.moh.gov.cn/publicfiles//business/htmlfiles/zwgkzt/pyq/list.htmS.
- [5] MMWR, (2007). Progress in hepatitis B prevention through universal infant vaccination China. Morbidity Mortality Weekly Rep. 56 (18), 441-445.
- [6] Lan Zou, Weinian Zhang Shigui Ruan, (2010). Modeling the transmission dynamics and control of hepatitis B virus in China, Journal of Theoretical Biology 262 (2010) 330-338 control measures. J. Viral. Hepat. 11, 97-107
- [7] Maple 18 Software. (2014) Waterloo Maple Inc. Ontario, Canada.