*Journal of the Nigerian Association of Mathematical Physics*
***Volume34,(March, 2016), pp 497 – 510***


# Modelling the Transmission Dynamics of Hepatitis B Virus (HBV) Infection in the Presence of Vaccination and Behavioural Change

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***Abstract***

***In this paper we proposed a Mathematical model for the transmission dynamics of Hepatitis B Virus (HBV) infection and studied the long term effects of vaccination and behavioural change in a hypothetical population. We computed the basic reproduction number $R_0$ which served as a threshold for measuring the spread of HBV infection in the assumed population. It was shown that the Disease - Free Equilibrium (DFE), $\varphi^0$ is locally asymptotically stable (LAS) if the basic reproduction number $R_0 < 1$; thus, HBV infection can be eradicated from the population, and unstable if $R_0 > 1$(leading to persistence of HBV infection within the population). The computational results revealed the effects of vaccination and behavioural change on the transmission dynamics of HBV. Furthermore, application of vaccination and behavioural change independently provide better intervention strategy for controlling HBV transmission, and the combination proffers an optimal control strategy.***

**Key words:**Vaccination; Behavioural Change; Hepatitis B Virus; Basic Reproduction Number; Stability
2010 AMS subject classification: primary: 92D40, 92D25; secondary: 34D20

## 1.0 Introduction

Hepatitis is inflammation of the liver, most commonly caused by viral infection. There are five main hepatitis viruses, referred to as types A, B, C, D and E. These five types are of greatest concern because of the illnesses and deaths which they cause and their potential for outbreaks and epidemic spread. In particular, types B and C cause chronic disease in hundreds of millions of people, and together are the most common cause of liver cirrhosis and cancer. In some cases, hepatitis B or C could destroy the liver to the extent that the patient would need liver transplant to survive, an option that is not always available or successful [1, 2].

Hepatitis B is a potentially life-threatening liver infection caused by the hepatitis B virus. It is a major global health problem. (HBV) could survive outside the body for at least seven days. During which time, the virus would still cause infection if it enters the body of unprotected person [2]. Worldwide, 2 billion people (1 out of 3 people) have been infected with **Hepatitis B;** 400 million people are chronically infected. An estimated 1 million people die each year from hepatitis B and its complications and 10-30 million are infected each year [3]. According to Hoofnagle*et al*. [4] 10% of people infected with HIV (approximately four million people world-wide) are co-infected with HBV.

Despite the fact that hepatitis B is the new epidemic ravaging the health of Nigerians, it has nonetheless, attracted very little attention from the government and people of Nigeria. Recent statistics indicate that not less than 23 million Nigerians are estimated to be infected with the Hepatitis B virus (HBV), making Nigeria one of the countries with the highest incidence of HBV infection in the world [5].

This infection has two possible phases: Acute and Chronic. Acute HBV infection lasts less than six months and is characterized by the presence of Hepatitis B surface Antigen (HBsAg) and Immunoglobulin M (IgM) antibody to the core antigen, (HBcAg). Chronic HBV infection is characterized by the persistence of HBsAg for six months or longer [2, 6].

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Hepatitis B is transmitted through infected**blood or bodily fluids in the same way as Human Immunodeficiency Virus (HIV), although HBV is** 50 to 100 times more infectious than HIV. This can occur through:**unprotected sex, direct blood-to-blood contact, shared or reuse of** unsterilized needles and syringes **(Horizontal transmission) and from an infected woman to her newbornduring the delivery process (vertical transmission)** [2, 3]. Hepatitis B Virus infection in Nigeria can be prevented or drastically reduced through health education of the general population on the various mode of transmission of HBV and preventive Measures [7].

One of the main reasons for studying Hepatitis B Virus (HBV) infection is to improve its control and if possible to eradicate the infection from the population. A number of mathematical models have been put forward in order to enhance the control of HBV infection in the world [8-25].
In this paper we improve the model due to Pang *et al*. [18] by incorporating behavioural change as additional preventive measure, HBV induced death rate due to acute and chronic infections and by splitting the immunity compartment into temporary protective immunity compartment and permanent protective immunity compartments. Behavioural change in this context is the modification of human behaviour through public health education, sensitization and creating awareness on the risk and possible ways of contracting HBV infection.

## 2.0 Materials and Methods

In this section, we present the mathematical formulation of compartmental model of Hepatitis B Virus (HBV) infection. The total population is subdivided into seven compartments as shown in Figure 1 while the model variables and parameters are presented in Table1 and Table 2 respectively.

## 2.1 Model Description

The uneducated susceptible population $x_u(t)$ is generated by coming in of the unsuccessfully immunize new-borns given by $\mu\omega(n-\varepsilon c)$, and individuals who loss immunity from temporary protective immunity compartment $v(t)$ (at the rate $\delta$ ). The uneducated susceptible individuals may acquire infection, following effective contact with acute infection individuals or chronic HBV carrier (at the rate $B(t)$), where $B(t)=\dfrac{\beta\left(y(t)+\alpha c(t)\right)}{n(t)}$. This population further decrease by natural death (at the rate $\mu_1$), vaccination (at the rate $p$ ) and education (behavioural change) (at the rate $\gamma_1$).

The population of educated susceptible individuals $x_e(t)$ is generated by educating the susceptible individuals (at the rate $\gamma_1$ ). This population is decreased by those who moved to temporary protective immunity compartment $v(t)$ (at the rate $\gamma_2$) and by natural death (at the rate $\mu_1$).

The population of exposed $e(t)$ is generated by infection of uneducated susceptible individuals $x_u(t)$ (at the rate $B(t)$) and by babies who are infected in prenatal infection and access to exposed class $e(t)$ and represented by $\mu\omega\varepsilon c(t)$. This population is reduced when the exposed become infectious (at the rate $\sigma$) and by natural death (at the rate $\mu_1$).
The population of acute infections $y(t)$ is generated by the exposed individuals who become infectious (at the rate $\sigma$). This population is decreased by those who progress to carrier $c(t)$ (at the rate $qr_1$), and by those who clear HBV infection and developed lifelong immunity and move to permanent protective immunity compartment $r(t)$ (at the rate $(1-q)r_1$ ). However, this population is further reduced by HBV induced death due acute infection (at the rate $\mu_2$) and natural death (at the rate $\mu_1$).

The population of carriers is generated by acute infections who progress to carrier (at the rate $qr_1$). The population is reduced by recovery of carriers (at the rate $r_2$ ), HBV induced death due HBV carrier (at the rate $\mu_3$) and natural death (at the rate $\mu_1$ ).

The population of temporary protective immunity compartment $v(t)$ is generated by the recovery of the HBV carriers (at the rate $r_2$ ), vaccination of uneducated susceptible individuals (at the rate $p$ ), successfully immunized newborn given by $\mu n(t)(1-\omega)$, and further increased by coming in of educated susceptible individuals (at the rate $\gamma_2$). This population is reduced by natural death (at the rate $\mu_1$) and loss of immunity (at the rate $\delta$ ). Finally, the population of permanent protective immunity compartment $r(t)$ is generated by recovery of acute infection individuals who acquire lifelong immunity at the rate $(1-q)r_1$ and decreased by natural death (at the rate $\mu_1$).

**Table 1:** Model Variables and their Description

| Parameter | parameter description |
|---|---|
| $x_u(t)$ | Uneducated susceptible population at time t |
| $x_e(t)$ | Educated susceptible population at time t |
| $e(t)$ | Exposed population at time t |
| $y(t)$ | Acute infection population at time t |
| $c(t)$ | HBV carriers population at time t |
| $v(t)$ | Population of temporary protective immunity compartment at time |
| $r(t)$ | Population permanent protective immunity compartment or recovered at time t |

**Table 2:** Model Parameters and their Description

| Parameter | parameter description |
|---|---|
| $\mu$ | Birth rate |
| $\mu_1$ | Natural death rate |
| $\mu_2$ | HBV induced death rate due to acute HBV infection |
| $\mu_3$ | HBV induced death rate due to HBV Carriers |
| $\beta$ | Transmission coefficient |
| $\gamma_1$ | Rate at which susceptible individuals get educated (behavioural change) |
| $\gamma_2$ | Rate at which educated susceptible individuals move to temporary protective immunity compartment |
| $r_1$ | Rate at which individuals leave the acute infection compartment |
| $r_2$ | Recovery rate of carriers |
| $\alpha$ | Infectiousness of carriers relative to acute infections |
| $q$ | Proportion of acute infection individuals who become carriers and another clear HBV |
| $\sigma$ | Rate of latent individuals becoming infections |
| $\omega$ | Proportion of failure immunization |
| $\varepsilon$ | Proportion of unimmunized children born to carrier mothers that have been infected |
| $p$ | Vaccination rate of susceptible and uneducated susceptible individuals |
| $\delta$ | Loss of immunity rate |

## 2.2 Model Assumption

The following assumptions are made in the construction of the model.

i. Behavioural change was introduced as additional preventive measure by targeting the susceptible individuals. Hence, we split the susceptible compartment into educated and uneducated compartments.
ii. Susceptible individuals who acquire proper education on HBV infection are capable of avoiding all the possible ways of contracting the infection through positive behavioural change.
iii. HBV induced death rates due to acute and chronic infections were considered.
iv. The immunity compartment was separated into temporary protective immunity compartment and permanent protective immunity compartment.

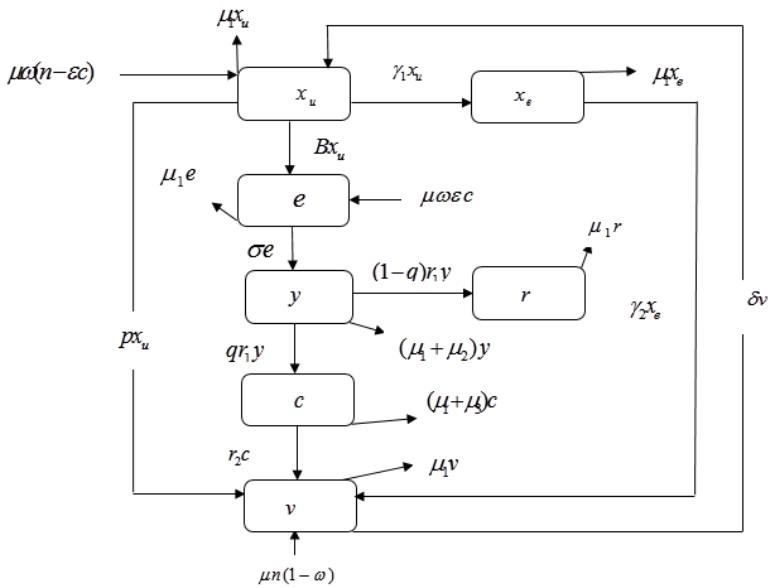


**Figure 1:** Epidemiological Flow Diagram for the Model

## 2.3 The Model Equations

Based on the assumptions and the epidemiological flow diagram in Figure 1, we derived the following system of non-linear first order ordinary differential equations that govern the model.

$$\dot{x}_u(t)=\mu\omega\big(n(t)-\varepsilon c(t)\big)+\delta v(t)-\big(B+\gamma_1+\mu_1+p\big)x_u(t) \quad (1)$$

$$\dot{x}_e(t)=\gamma_1 x_u(t)-\big(\mu_1+\gamma_2\big)x_e(t) \quad (2)$$

$$\dot{e}(t)=Bx_u(t)+\mu\omega\varepsilon c(t)-\big(\mu_1+\sigma\big)e(t) \quad (3)$$

$$\dot{y}(t)=\sigma e(t)-\big(r_1+\mu_1+\mu_2\big)y(t) \quad (4)$$

$$\dot{c}(t)=qr_1 y(t)-\big(r_2+\mu_1+\mu_3\big)c(t) \quad (5)$$

$$\dot{v}(t)=r_2 c(t)+px_u(t)+\gamma_2 x_e(t)+\mu n(t)\big(1-\omega\big)-\big(\mu_1+\delta\big)v(t) \quad (6)$$

$$\dot{r}(t)=\big(1-q\big)r_1 y(t)-\mu_1 r(t) \quad (7)$$

$$B=\frac{\beta\big(y(t)+\alpha c(t)\big)}{n(t)} \quad (8)$$

$$n(t)=x_u(t)+x_e(t)+e(t)+y(t)+c(t)+v(t)+r(t) \quad (9)$$

$$x_u(0)=x_u^0, x_e(0)=x_e^0, e(0)=e^0, y(0)=y^0, c(0)=c^0, v(0)=v^0, r(0)=r^0 \quad (10)$$

We transformed the model equations into proportions to enable us study the steady states. This is based on the assumption [26], that it is more likely for the population in proportions to attain the steady states than an individual population class which perhaps may only happen when the carrying capacity is reached. We adopted the method used in [17, 18, 23, 26, and 27] in transforming the model equations into proportions.

We set the proportion as follows:

$$X_u(t)=\frac{x_u(t)}{n(t)}, X_e(t)=\frac{x_e(t)}{n(t)}, E(t)=\frac{e(t)}{n(t)}, Y(t)=\frac{y(t)}{n(t)}, C(t)=\frac{c(t)}{n(t)}, V(t)=\frac{v(t)}{n(t)}, R(t)=\frac{r(t)}{n(t)}$$

$$X_u(t)+X_e(t)+E(t)+Y(t)+C(t)+V(t)+R(t)=1 \qquad (11)$$

From equation (11) we have $V(t)=1-\left(X_u+X_e+E+Y+C+R\right)$ and using equation (11) in (8) we obtain $B=\beta\left(Y(t)+\alpha C(t)\right)$. Thus, without loss of generality the model equations in proportions can be written as;

$$\dot{X}_u(t)=\mu\omega\left(1-\varepsilon C(t)\right)+\delta\left(1-(X_u(t)+X_e(t)+E(t)+Y(t)+C(t)+R(t))\right)$$
$$-\left(\beta\left(Y(t)+\alpha C(t)\right)+k_1\right)X_u(t) \qquad (12)$$

$$\dot{X}_e(t)=\gamma_1 X_u(t)-k_2 X_e(t) \qquad (13)$$

$$\dot{E}(t)=\beta\left(Y(t)+\alpha C(t)\right)X_u(t)+\mu\omega\varepsilon C(t)-k_3 E(t) \qquad (14)$$

$$\dot{Y}(t)=\sigma E(t)-k_4 Y(t) \qquad (15)$$

$$\dot{C}(t)=qr_1 Y(t)-k_5 C(t) \qquad (16)$$

$$\dot{R}(t)=\left(1-q\right)r_1 Y(t)-k_6 R(t) \qquad (17)$$

$$X_u(0)=X_u^0, X_e(0)=X_e^0, E(0)=E^0, Y(0)=Y^0, C(0)=C^0, R(0)=R^0 \qquad (18)$$

Where

$$k_1=\gamma_1+\mu_1+p, k_2=\mu_1+\gamma_2, k_3=\mu_1+\sigma, k_4=r_1+\mu_1+\mu_2, k_5=r_2+\mu_1+\mu_3. k_6=\mu_1$$

## 3.0 Results

In this section we present the analytical and numerical results obtained in this work.

### 3.1 Equilibrium States of the Model

The Disease – Free Equilibrium (DFE) of the model was obtained by setting the right-hand side of equations (12) – (17) to zero and is given by; $\varphi^0=\left(X_u^0, X_e^0, E^0, Y^0, C^0, R^0\right)=\left(\frac{k_2(\mu\omega+\delta)}{\left(k_2(\delta+k_1)+\gamma_1\delta\right)}, \frac{\gamma_1(\mu\omega+\delta)}{\left(k_2(\delta+k_1)+\gamma_1\delta\right)}, 0,0,0,0\right)$

*To obtain the Endemic Equilibrium (EE) of the model, we set the right hand side of equations (12) – (17) to zero and setting* $X_u=X_u^*, X_e=X_e^*, E=E^*, Y=Y^*, C=C^*, R=R^*$ *in same equations we obtained*

$\varphi^*=(X_u^*, X_e^*, E^*, Y^*, C^*, R^*)$ where

$$X_u^*=\frac{k_3E^*-\mu\omega\varepsilon C^*}{\beta\left(Y^*+\alpha C^*\right)}, X_u^*>0 \text{ iff } E^*>\frac{\mu\omega\varepsilon C^*}{k_3}, X_e^*=\frac{\gamma_1 X_u^*}{k_2}, E^*=\frac{k_4 Y^*}{\sigma}, Y^*=\frac{k_6 R^*}{\left(1-q\right)r_1 Y^*}, Y^*>0 \text{ iff } q<1, C^*=\frac{qr_1 Y^*}{k_5}$$

$$R^*=\frac{\mu\omega-\mu\omega\varepsilon C^*+\delta\left(1-(X_u^*+X_e^*+E^*+Y^*+C^*)\right)-\left(\beta\left(Y^*+\alpha C^*\right)+k_1\right)X_u^*}{\delta}$$

$$R^*>0 \text{ iff } \mu\omega+\delta>\mu\omega\varepsilon C^*+\delta(X_u^*+X_e^*+E^*+Y^*+C^*)+(\beta\left(Y^*+\alpha C^*\right)+k_1)X_u^*$$

### 3.2 Basic Reproduction Number

The basic reproduction number $R_0$ for the model gives an average number of secondary infection when an infection is introduced in a purely susceptible population. According Diekmann*et al.* [28] the basic reproduction number $R_0$ is among the quantities must urgently estimate for emerging infectious diseases outbreak situations, and its value provides insight when designing control interventions for established infections. We now evaluate the basic reproduction number using next-generation matrix. This method is given by [29, 30].

Let $F_i$ be the rate of appearance of new infection in compartment $i$ and $V_i$ represent the rate of transfer of individuals out of compartment $i$ by any other means and $\varphi^0$ is the Disease- Free Equilibrium (DFE), then $R_0$ is the spectral radius or the

largest eigenvalue of

$$\left[\frac{\partial F_i(\varphi^0)}{\partial x_j}\right]\left[\frac{\partial V_i(\varphi^0)}{\partial x_j}\right]^{-1} = FV^{-1}$$

Where, $F = \begin{bmatrix} \beta(Y+\alpha C)X_u + \mu\omega\varepsilon C \\ 0 \\ 0 \end{bmatrix}, V = \begin{bmatrix} k_3 & 0 & 0 \\ -\sigma & k_4 & 0 \\ 0 & -qr_1 & k_5 \end{bmatrix}$ and $V^{-1} = \begin{bmatrix} \frac{1}{k_3} & 0 & 0 \\ \frac{\sigma}{k_3k_4} & \frac{1}{k_4} & 0 \\ \frac{\sigma qr_1}{k_3k_4k_5} & \frac{qr_1}{k_4k_5} & \frac{1}{k_5} \end{bmatrix}$

Thus, the basic reproduction number is then given as:

$$R_0 = \frac{\sigma\beta}{k_3k_4}X_u^0 + \frac{\alpha\beta\sigma qr}{k_3k_4k_5}X_u^0 + \frac{\mu\omega\varepsilon\sigma qr_1}{k_3k_4k_5} \tag{19}$$

Define $R_{01} = \frac{\sigma\beta X_u^0}{k_3k_4}$, $R_{02} = \frac{\alpha\beta\sigma qr_1X_u^0}{k_3k_4k_5}$ and $R_{03} = \frac{\mu\omega\varepsilon\sigma qr_1}{k_3k_4k_5}$ so that equation (19) become

$$R_0 = R_{01} + R_{02} + R_{03} \tag{20}$$

## 3.3 Local Stability of Disease-Free Equilibrium (DFE)

In this section, we established the local stability of Disease-Free Equilibrium (DFE), $\varphi^0$ using linearization approach. The stability result for the model is presented in the following theorem.

**Theorem 1:** The Disease Free Equilibrium (DFE), $\varphi^0$ of the model equations in proportions given by (12) – (17) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

**Proof:**

Linearization of (12)-(17) at disease-free equilibrium gives the Jacobian matrix

$$J(\varphi^0) = \begin{pmatrix} -(\delta+k_1) & -\delta & -\delta & -(\delta+\beta X_u^0) & -(\delta+\mu\omega\varepsilon+\alpha\beta X_u^0) & -\delta \\ \gamma_1 & -k_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_3 & \beta X_u^0 & \alpha\beta X_u^0+\mu\omega\varepsilon & 0 \\ 0 & 0 & \sigma & -k_4 & 0 & 0 \\ 0 & 0 & 0 & qr_1 & -k_5 & 0 \\ 0 & 0 & 0 & (1-q)r_1 & 0 & -k_6 \end{pmatrix}$$

Using elementary row-transformation, we have

$$J(\varphi^0) = \begin{pmatrix} -M_0 & -\delta & -\delta & -M_1 & -M_2 & -\delta \\ 0 & -M_5 & -\gamma_1\delta & -\gamma_1M_1 & -\gamma_1M_2 & -\gamma_1\delta \\ 0 & 0 & -k_3 & \beta X_u^0 & M_3 & 0 \\ 0 & 0 & 0 & M_6 & \sigma M_3 & 0 \\ 0 & 0 & 0 & 0 & M_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & M_8 \end{pmatrix}$$

where

$$M_0 = \delta + k_1, M_1 = \delta + \beta X_u^0, M_2 = \delta + \mu\omega\varepsilon + \alpha\beta X_u^0, M_3 = \alpha\beta X_u^0 + \mu\omega\varepsilon, M_4 = (1-q)r_1, M_5 = \gamma_1\delta + k_2(\delta + k_1)$$

$$M_6 = \sigma\beta X_u^0 - K_3K_4, M_7 = qr_1\sigma(\alpha\beta X_u^0 + \mu\omega\varepsilon) + k_5(\sigma\beta X_u^0 - K_3K_4)$$

$$M_8 = \left(\sigma\beta X_u^0 - K_3K_4\right)\left(qr_1\sigma(\alpha\beta X_u^0 + \mu\omega\varepsilon) + k_5(\sigma\beta X_u^0 - K_3K_4)\right)\left(k_6\right)$$

Thus, the eigenvalues of row-transformed Jacobian matrix are given by

$$\lambda_1 = -M_0, \qquad \lambda_2 = -M_5, \qquad \lambda_3 = -K_3, \qquad \lambda_4 = M_6, \qquad \lambda_5 = M_7, \qquad \lambda_6 = M_8$$

Clearly $\lambda_1, \lambda_2, \lambda_3$ are all negative, for $\lambda_4 < 0$ we should have $M_6 < 0$ or $\frac{\sigma\beta X_u^0}{K_3 K_4} < 1$ i.e. $R_{01} < 1$. It follows that, $\lambda_4$ satisfies the negativity requirement for stability if $R_{01} < 1$. Now, when $R_0 < 1$ we have $0 < R_{01} < 1, 0 < R_{02} < 1$ and $0 < R_{03} < 1$. Thus $R_{01} < 1$ implies $R_0 < 1$. Similar to the proof of $\lambda_4$, we obtain that $\lambda_5, \lambda_6 < 0$ if and only if $R_0 < 1$. Therefore, the eigenvalues of the row – transformed Jacobian matrix evaluated at the (DFE), $\varphi^0$ has negative real part if $R_0 < 1$. Hence, the Disease - Free Equilibrium, (DFE), $\varphi^0$ of the model equations given by (12) - (17) is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$, which proves the theorem.

The epidemiological implication of this theorem is that HBV infection can be eradicated within the given population if the initial sizes of the state variables are within the vicinity of $\varphi^0$. This can be achieved when the magnitude of $R_0$ is below 1. So, any control measure that reduces the magnitude of $R_0$ to be less than unity should be the effective measure in fighting the menace of HBV infection.

## 3.4 Numerical Simulation

In this section, we present the numerical simulation of the model by using Runge-Kutta order four scheme. We used the baseline values for the variables and parameters as in Table 3 for the numerical simulations. We compare the performances of the existing model i.e. the model due to Pang *et al.* [18] with the modified model using the preventive measures at different rates of coverage. However, we also maintain 90% vaccination of infant i.e. ω = 0. 1 and the proportions of carriers were used for all the simulations as used by Pang *et al.* [18]. The computational results are presented in figure 2 to figure 11.

**Table 3:** The Baseline Values for Variables and Parameters of the Models.

| Parameter | parameter description | Value | Source |
|---|---|---|---|
| $\mu$ | Birth rate | 0.014 | [16] |
| $\mu_1$ | Natural death rate | 0.011 | [22] |
| $\mu_2$ | HBV induced death rate due to acute HBV infection | 0.007 | [22] |
| $\mu_3$ | HBV induced death rate due to HBV Carriers | 0.001 | [22] |
| $\beta$ | Transmission coefficient | 0.85 | [10] |
| $\gamma_1$ | Rate at which susceptible individuals get educated (behavioural change) | 0 – 0.75 | Assumed |
| $\gamma_2$ | Rate at which educated susceptible individuals move to temporary protective immunity compartment | 0 – 0.1 | Assumed |
| $r_1$ | Rate at which individuals leave the acute infection class | 4 | [14] |
| $r_2$ | Recovery rate of carriers | 0.025 | [14] |
| $\alpha$ | Infectiousness of carriers relative to acute infections | 0.5 | [18] |
| $q$ | Proportion of acute infection individuals who become carriers and another clear HBV | 0.1 | [32] |
| $\sigma$ | Rate of latent individuals becoming infections | 6 | [14] |
| $\omega$ | Proportion of failure immunization | 0, 0.1, 1 | [32] |
| $\varepsilon$ | Proportion of unimmunized children born to carrier mothers that have been infected | 0.8 | [31] |
| $p$ | Vaccination rate of susceptible and uneducated susceptible individuals | 0 – 1 | [18] |
| $\delta$ | Loss of immunity rate | 0.045 | [21] |
| x(0) | Initials proportion of susceptible individuals | 0.6 | Assumed |
| e(0)/E(0) | Initials proportion of exposed individuals | 0.2/0.1 | Assumed |
| y(0)/Y(0) | Initials proportion of acute infection individuals | 0.13 | Assumed |
| c(0)/C(0) | Initials proportion of carriers | 0.07 | [18] |
| $X_u(0)$ | Initials proportion of uneducated susceptible individuals | 0.4 | Assumed |
| $X_e(0)$ | Initials proportion of educated susceptible individuals | 0.2 | Assumed |
| R(0) | Initial proportion of recovered individuals | 0.1 | Assumed |

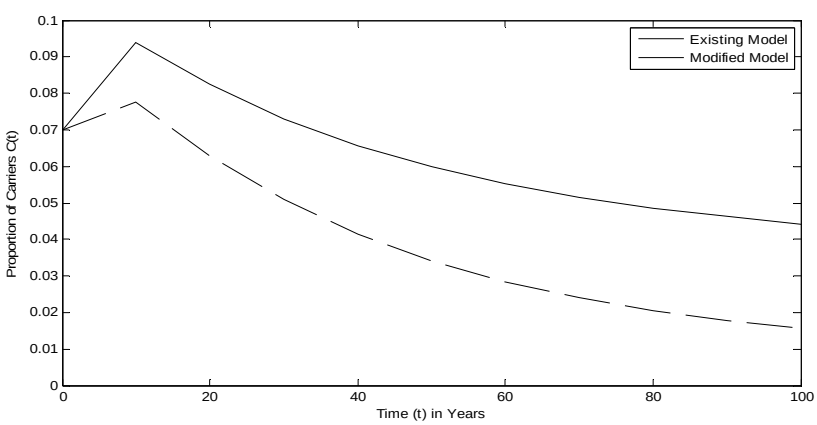


**Figure 2:** Graph Comparing the Performances of Models in the absence of Preventive Measures (Parameter values used are as in Table 1 with $\omega = 1, p = 0, \gamma_1 = 0$).

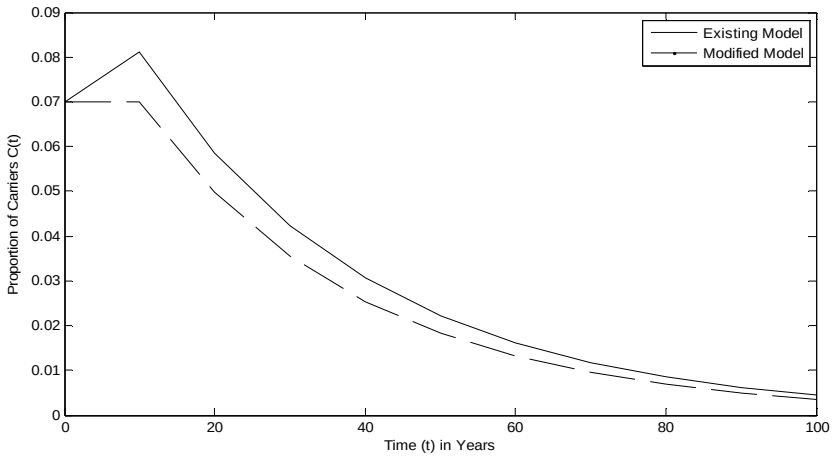


**Figure 3:** Graph Comparing the Performances of the Models in the Presence of Vaccination as the only Preventive Measure (Parameter values used are as in Table 1 with p = 25%).

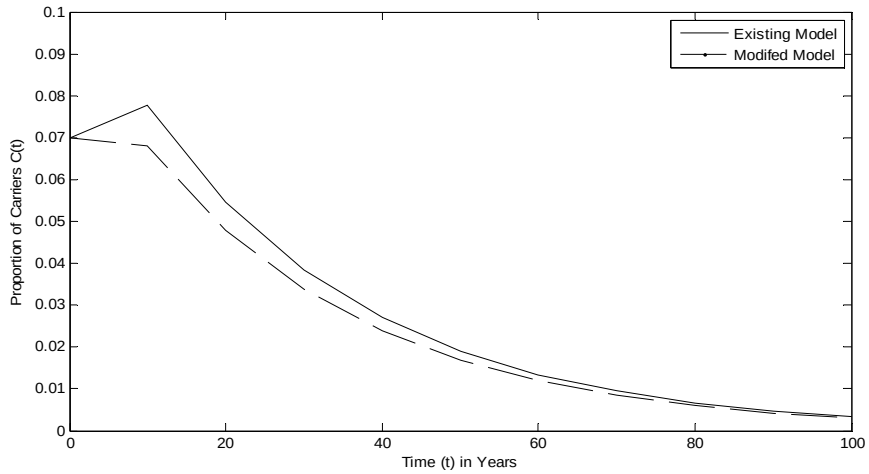


Figure 4:Graph Comparing the Performances of the Models in the Presence of Vaccination as the only Preventive Measure (Parameter values used are as in Table 1 with p = 50%).

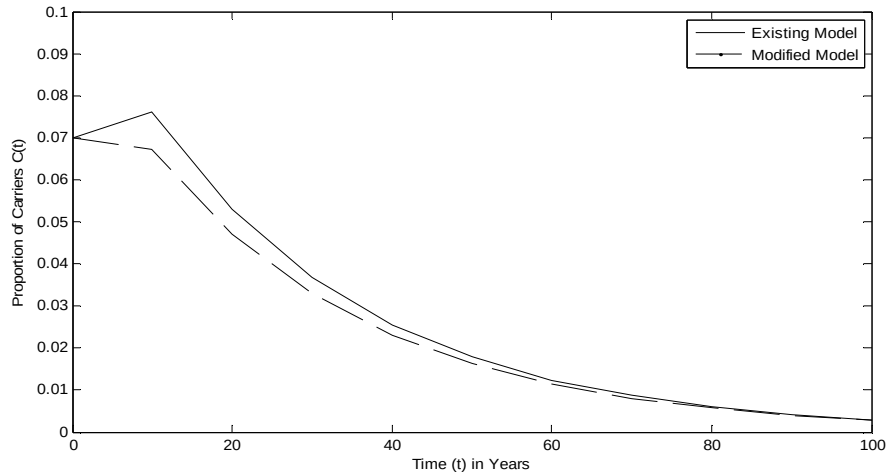


**Figure 5:** Graph Comparing the Performances of the Models in the Presence of Vaccination as the only Preventive Measure (Parameter values used are as in Table 1 with p = 75%).

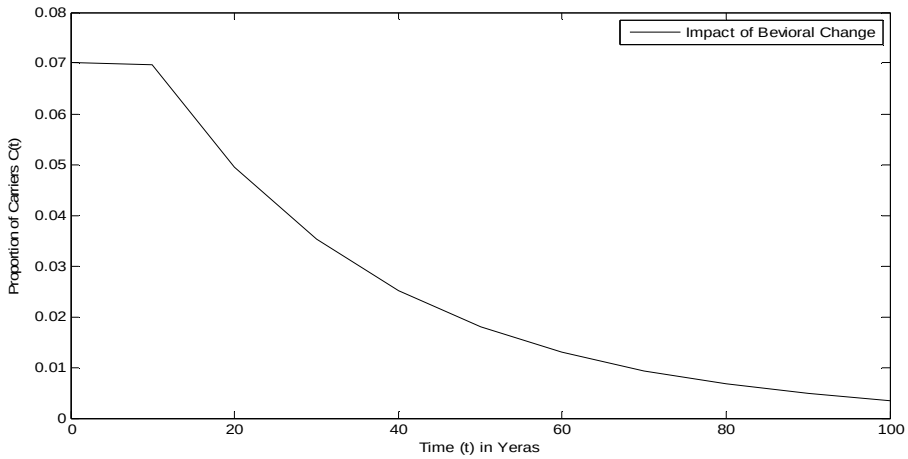


**Figure 6:** Graph showing the Impacts of Behavioural Change as the only Preventive Measure (Parameter Values used as in Table 1 with $\gamma_1 = 25\%$ using the Modified Model).

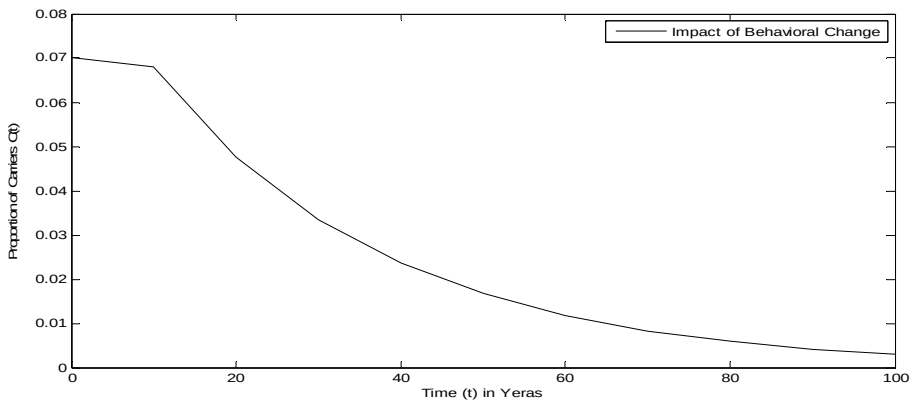


**Figure 7:** Graph Showing the Impacts of Behavioural Change as the only Preventive Measure (Parameter Values used as in Table 1 with $\gamma_1 = 50\%$ using the Modified Model).

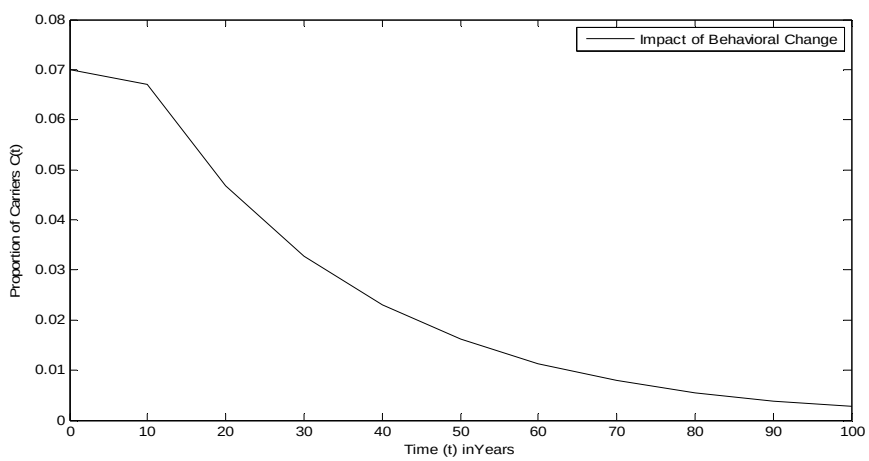


**Figure 8:** Graph Showing the Impacts of Behavioural Change as the only Preventive Measure (Parameter Values used as in Table 1 with $\gamma_1 = 75\%$ using the Modified Model).

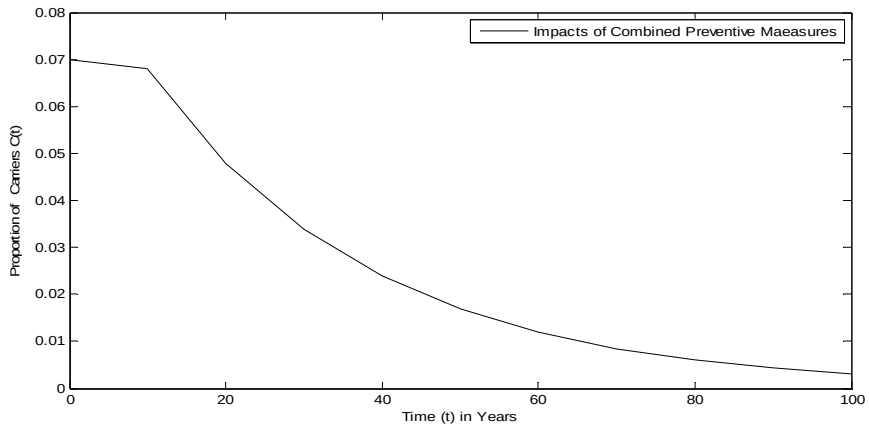


**Figure 9:** Graph Showing the Impact of Combined Preventive Measures (Parameter Values used as in Table 1 with $\gamma_1 = 25\%, p = 25\%$).

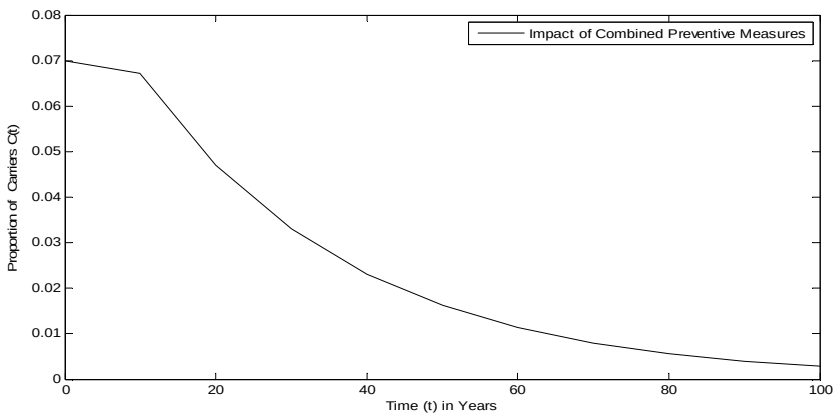


**Figure 10:** Graph Showing the Impact of Combined Preventive Measures (Parameter Values used as in Table1 with $\gamma_1 = 50\%, p = 25\%$ ).

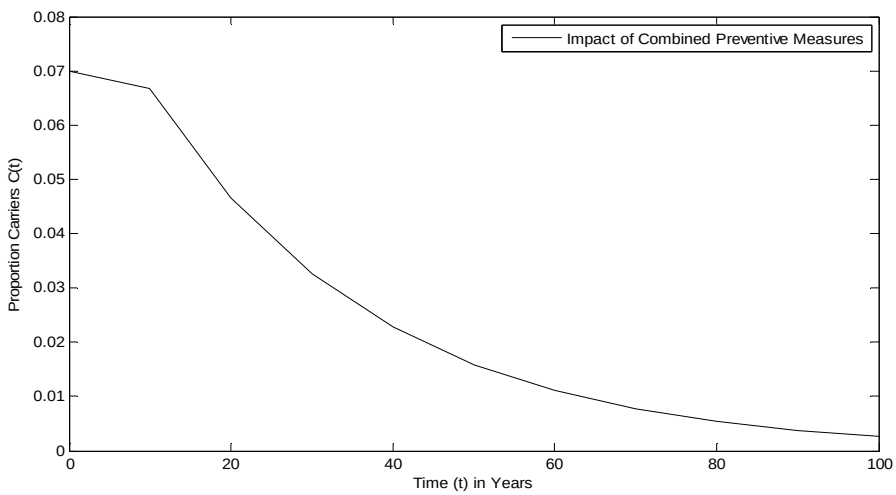


**Figure 11:** Graph Showing the Impact of Combined Preventive Measures (Parameter Values used as in Table 1 with $\gamma_1 = 75\%, p = 25\%$).

## 4.0 Discussion

In the first set of simulation, we compare the performances of the model due to Pang *et al.* [18] and the modified model in the absence of preventive measures using the proportion of carriers. The plot in Figure 2 shows that, in both the models HBV infection will persist in the population. However, the persistency is higher in the existing model as in 100 years the proportion of carriers is about 4.42% compare with 1.6% in the modified model. In the second set of simulations, we compare the long-term effectiveness of vaccination of susceptible individuals as the only preventive measure using the model due to Pang *et al.* [18] and the modified model. We vary the vaccination rate p, from 25%, 50%, and 75% in the uneducated susceptible individuals; we see in Figures 3, 4, and 5 that the application of vaccination as the only preventive measure gives better results than the absence of preventive measures in both the model due to Pang *et al.* [18] and the modified model. However, as we increase the vaccination rate p from 25% to 50 % and from 50% to 75%, the plots in Figures 3, 4, and 5 shows that in the modified model there is fast reduction in the proportions of carriers as compared with the model due to Pang *et al.* [18] This indicates that the modified model competes favourably in respect to the control of HBV infection. In the third set of simulations, we apply behavioural change as the only preventive measure in the susceptible individuals. We fixed vaccination rate p = 0, and vary $\gamma_1$ the rate at which susceptible individuals get educated on HBV (behavioural change) to see the impact of increasing its rates on the proportion of carriers. If we set $\gamma_1 = 25\%$, we notice in Figure 6 that the proportion of carriers reduces, if we increase $\gamma_1$ from 25% to 50% we observed a moderate decrease in the proportion of carriers see Figure 7. We further increase $\gamma_1$ from 50% to 75% the plot in Figure 8, shows significant reduction in proportion of carriers as in 100 years proportion of carriers reduces to 0.28%. Indeed, this result is remarkable because it shows that increase in behavioural change coverage will lead to decrease in the proportion of carriers. This result shows that behavioural change could serve as an alternative control strategy in fighting the menace of HBV infection especially in the poorest countries where the cost of the vaccine is unaffordable. Finally in this set of simulations, we examine the impact of combined preventive measures on the proportion of carriers. We fixed the vaccination rate of susceptible individual, p = 25% and vary behavioural change coverage at 25%, 50%, and 75% respectively. The plot in Figure 9 shows that, when both preventive measures are applied at the same rates i.e. 25%. We observed a reduction in the proportion of carries, as in 10 years it reduces to 6.81% and in 50 years it further reduces to 1.69%. If we increase $\gamma_1$ from 25% to 50% Figure 10 present a moderate decline in the proportion of carries. The plot in Figure 11 shows a rapid decrease in the proportion of carriers as a result of increment of $\gamma_1$ from 50% to 75%. These results revealed that application of both vaccination and behavioural change provides optimal control strategy, since the proportion of carrier in 50 years reduces to 2%. Thus, using combined preventive measures at these rates reduces the length of time taken to achieve Eradication of HBV infection in the population.

## 5.0 Conclusion

A modified version of mathematical model of HBV infection due to Pang *et al.* [18] was proposed by incorporating behavioural change as additional preventive measure targeting the susceptible individuals, HBV induced death rates due to acute and chronic infections were considered and the immunity compartment was separated into temporary protective immunity and permanent protective immunity compartments. The modified model equations were transformed into proportions in which two non – negative equilibriums namely; Disease – Free Equilibrium (DFE) and Endemic Equilibriums (EE) were established.

We evaluated the basic reproduction number of the modified model using technique of [29, 30]. Moreover, the local stability analysis of the Disease – Free Equilibrium (DFE) of the modified model was established using linearization approach, which revealed that, the model is locally asymptotically stable provided that $R_0 < 1$ and unstable if $R_0 > 1$.

Based on the computational results obtained, the modified version of HBV model in the presence of vaccination competes favourably with the existing model i.e. model due to Pang *et al.* [18] as it reduces the proportions of carriers faster. Similarly, the output of the modified model established that behavioural change could be an alternative control strategy since, increase in the rate of behavioural change reduces the proportion of carriers significantly. Indeed, the computational result of the modified model with combined preventive measures drastically reduces the proportion of carriers. Thus, application of vaccination and behavioural change independently provide better intervention strategy for controlling HBV transmission, and the combination proffers the optimal control strategy.

## 6.0 Recommendation

Based on the findings in this research we wish to recommend the model for epidemiologists to better predict and control HBV infection in a given population as it gives better insight into the transmission dynamics of HBV infection. We also recommend behavioural change as an alternative preventive measure especially for the poorest countries. However, we strongly recommend the use of combined preventive measures is as it offers optimal control strategy. The use of theoretical data is a limitation in this work, therefore we recommend the use of real data to test the model.

## 7.0 Acknowledgement

We wish to acknowledge the management of ModibboAdama University of Technology, Yola, Nigeria and the entire lecturers of Mathematics department for their immense supports and contributions for the successful completion of this research.

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