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# A DETERMINISTIC MATHEMATICAL MODEL AND ANALYSIS OF THE TRANSMISSION DYNAMICS OF HEPATITIS B VIRUS IN NIGERIA

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## Abstract

HBV is a serious liver infection caused by hepatitis B virus that can easily be preventable by a vaccine. Nigeria is one of the countries with the highest incidence of HBV infection in the world with less than 23 million Nigerians are estimated to be infected with the Hepatitis B virus (HBV) from a population of two hundred million. The aim of this research work is to modeled HBV by incorporating vaccination, on the sport treatment, sanitarium and immigration into to already exiting SEIR model; the objectives are: to obtain the equilibria state of the model. Analyze local and global stability of the equilibria state and also carry out numerical simulations of the model. We used a deterministic mathematical model of HBV transmission dynamics to demonstrate the dynamics of the disease. We partitioned the population into 7 compartments namely: Immunized M(t), Susceptible S(t), Latent L(t), Infectious I(t), Senatorium $S_1(t)$, Vaccinated V(t) and Recovered R(t). The dynamics among the compartments are

described using differential equations. Epidemiologically this means that HBV disease will continue to persist if immigration HBV infective is not controlled. The local stability of disease-free equilibrium of the dynamical system in the absent of HBV infective immigrant has a reproductive number less than one, readily observed from the coefficient of the polynomial characteristics satisfied the condition Routh-Hurwitz criterion, thus, it is locally asymptotically stable. The global stability of the disease-free equilibrium of the model was obtained and it revealed that, the entries of the matrix for infected compartment G(X, Z) are stickily positive. Hence, the global stability of the disease free is stable. In order to ascertain the impact of using vaccination or sanatorium or combination of both control strategies on fighting HBV, we established a baseline values for the parameters and implored the use of MATLAB Codes in computing numerical values, the results shows that the infected population is significantly higher than the other population densities even when vaccination is administered in relatively small amount. When the vaccination rate is increased to $\omega = 0.9$, the infected population drastically declined and vice versa.

**Keywords:** Mathematical model**,** Hepatitis B Virus (HBV), Equilibria state, Stability Analysis, Reproduction Number

## 1. Introduction

Hepatitis is a liver illness brought on by medications, alcohol, or certain medical disorders. The most prevalent causes are hepatitis A, B, and C, that are caused by a virus known as viral hepatitis. The Hepatitis B Virus causes a potentially fatal liver illness called hepatitis B (HBV). Hepatitis B is a tiny DNA virus that belongs to the "Hepadnaviridae" family of viruses. Wood chucks, ground squirrels, tree squirrels, Peking ducks, and herons all carry viruses from this family (CDC, 2008; WHO, 2013). HBV infection can cause asymptomatic or subclinical infection, acute self-limited hepatitis, or fulminant hepatitis that necessitates liver transplantation. There's an outer envelope and an inner core to the hepatitis B virus. The hepatitis B surface antigen, or "HBsAg," is found on the virus's outer membrane. A simple blood test may identify HBsAg, and a positive result suggests hepatitis B virus infection. The hepatitis B core antigen, or "HBcAg," is a protein shell that houses the virus's DNA and enzymes for reproduction. More than 250 million individuals worldwide are infected with the hepatitis B virus, which has resulted in over 800,000 fatalities (WHO, 2017). Hepatitis B (HBV) is one of the most common infectious illnesses

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known to man, with the highest incidence in the Western Pacific and African regions. Despite the fact that an HB vaccination has been available since 1982, there is still a surge in HB transmission and distribution (WHO, 2017).

The life cycle of the hepatitis B virus (HBV) is complicated. The virus penetrates the host liver cell and travels to the nucleus. The viral DNA is converted into covalently closed circular DNA (cccDNA) once within the nucleus, which acts as a template for viral replication (creation of new hepatitis B virus). New HBV virus is packed and exits the liver cell, leaving the stable viral cccDNA in the nucleus to integrate into the host liver cell's DNA and continue to produce new hepatitis B virus. The HBV virus may persist outside the body for at least 7 days, and if it enters the body of someone who has not been vaccinated, it can still cause illness. HBV has a 75-day incubation period. Within 30-60 days of infection, the virus can be diagnosed, and it can persist and evolve into chronic HB. During the acute infection phase, the majority of HB carriers have no symptoms; nevertheless, some persons develop acute sickness with symptoms that can linger for several days or even weeks. The time it takes for HB to move from latent infection to active illness varies substantially. People with AIDS or who have had an organ transplant, for example, are more prone to acquire chronic HB following infection. According to WHO (2017) the age at which a person becomes infected can also influence the chance of illness becoming chronic. Children under the age of six who become infected with HBV have the highest risk of developing a chronic infection.

The hepatitis B virus causes hepatitis B infection (HBV). The virus is transmitted from one person to another by blood, sperm, or other bodily fluids. It is not transferred by coughing or sneezing. Sexual contact, needle sharing, accidental needle sticks, and mother to unborn child are all common routes for HBV to spread. Despite massive measures to combat the threat, HBV has remained a global problem. Hepatitis B has infected 2 billion individuals worldwide (1 out of every 3 persons); 400 million people are chronically afflicted. Every year, an estimated 1 million people die from hepatitis B and its consequences, while 10-30 million individuals become infected (HBV Foundation, 2014). Hoofnagle et al, (2007) had claimed that 10% of HIV-positive patients (about four million people worldwide) are also infected with HBV.

Given that hepatitis B is on Nigeria's list of deadly illnesses, the Nigerian government felt compelled to move quickly on different control techniques. According to statistical indicators, at least 23 million Nigerians are infected with

the Hepatitis B virus (HBV), making Nigeria one of the countries with the highest HBV infection rates in the world (Akanni, 2014).

Vaccination, behavioral change campaigns, illness education campaigns, and other management techniques have all been implemented in an attempt to remove the threat or reduce its endemicity among the world population. As a result, the goal of this study is to build on an existing model created by Blessing and Iyama (2017) in order to develop a better control approach for reducing HBV infection transmission dynamics in Nigeria. As a result, the focus of this research is on combining vaccine, sanitarium, and immigration into the existing model.

## 2. Reviews on Mathematical Models on HBV

Emechebe et al (2009) conducted study to determine the prevalence of hepatitis B virus infection in Nigeria. The findings demonstrated that the incidence of hepatitis B virus infection has remained high in Nigeria for over 30 years, dating back to pioneering work done in the country. Hepatitis B virus transmission occurs mostly during infancy in Nigeria, and all of the risk factors (such as blood transfusion, sexual promiscuity, lower socioeconomic position, etc implicated elsewhere in the virus's spread in the general population also play a role in Nigeria. They concluded that, reduction in the incidence of hepatitis B virus infection could be achieved by public enlightenment campaign, mass immunization of the children and adults at risk while antiviral drugs and immune stimulatory therapy should be provided for those already infected.

Pang and Zhou (2010) developed a mathematical model to investigate the influence of HBV infection control methods such as vaccination. The numerical simulation findings indicated that vaccination is a very successful way to reduce infection, and they also provided some helpful suggestions for preventing HBV transmission.

Kalajdzievsk and Li (2011) examine the influence of carriers on transmission patterns, a generic mathematical model for infectious illnesses with asymptomatic carriers was devised. The model was applied to Chronic Hepatitis B, and the numerical findings suggested that, in countries with high HBV prevalence, testing and raising awareness of chronic carriers will have a considerably bigger impact on disease burden than improving vaccination rates.

Zhang and Zhou (2012) To characterize the transmission of hepatitis B, a mathematical model was developed. The stability of equilibriums and illness persistence were investigated. The finding demonstrates that the model's

dynamics are entirely driven by the basic reproductive number q0. When q0 is less than 1, the disease-free equilibrium is globally stable; when q0 is more than 1, the disease-free equilibrium is unstable, and the disease is uniformly persistent. Furthermore, it has been demonstrated that the endemic equilibrium is universally appealing under specific conditions. To show the theoretical conclusions, numerical simulations were run. In China, the model was used to study HBV transmission. The model's parameter values were calculated using data from China's HBV epidemic. The simulated result comes close to the HBV epidemic statistics in China.

Momoh, Ibrahim, Madu, and Asogwa, (2012) proposed an MSEIR model for the dynamics of HBV transmission using a model including passive vaccination, individual therapy, and infectious hepatitis B therapy. The disease-free and endemic equilibrium states were developed, and Bellman and Cooke's theorem was used to assess the stability of the epidemic equilibrium state. Their findings demonstrated that the epidemic equilibrium state was stable, implying that the model may be used to forecast the long-term efficacy of vaccination combined with exposed persons therapy and infectious hepatitis B medication in maintaining a population.

Abdulrahman, Akinwande, Awojoyogbe and Abubakar (2013a) developed a novel mathematical model including vertical transmission and sexual maturity for the dynamics of hepatitis B virus (HBV) transmission in a population with vital dynamics. The reproduction number was subjected to a sensitivity analysis in relation to the model parameters. The findings reveal that the birth rate, mortality removal rate, HBV sexual transmission probability per contact rate, and average total sexual contacts rate are all very sensitive characteristics that influence the dynamics of HBV transmission in any community. Vaccination, condom use, and a lower-than-average sexual partner(s) are all excellent measures for controlling HBV.

Khan, Islam, Arif and Haq (2013) modified the model for hepatitis B infection due to Pang and Zhou (2010) they studied the influence of immigrants in the model to explore the effect of immigrants on the host population by introducing the migrated class and adding the "transmission between migrated and exposed class" and "the transmission between migrated and acute class." They argued that, with the additional assumptions and the addition of the migrated class, the model informs government policy, allowing them to be aware of immigrants and subject them to tests to determine their status. To limit the number of diseased

immigrants, short-term visitors and students should be submitted to examinations.

Oladeinde, Omoregie, and Oladeinde (2013) carried out a research in Nigeria to establish the prevalence of HIV, HBV, and HCV infections, as well as the risk factors associated with them, among pregnant women getting prenatal care in traditional birth homes. HIV, HBV, and HCV infection rates were found to be 7.2 percent, 2.2 percent, and 0.8 percent, respectively. The study also found that intervention efforts should be promoted in Nigeria to reduce these illnesses and their complications among pregnant women getting prenatal care in traditional birth homes.

Abdulrahman, Akinwande, Abubakar and Awojoyogbe (2013b) using a mathematical model, the researchers investigated the transmission dynamics and control of hepatitis B virus (HBV) infection in Nigeria. Using six alternative control procedures, the effective basic reproduction number $R_0$ was obtained and its values were calculated. Their findings reveal that, with a 25-year fading rate of vaccine, HBV cannot be managed at any coverage rate with immunization of new births or vulnerable adults before sexual activity. The country's HBV infection problem has been solved in two ways. The first is a 20% condom usage rate among sexually active people, with a 66 percent vaccine coverage rate for new births and a 20% vaccination rate for sexually active and yet to be sexually active vulnerable people. The second viable option is to use condoms at a rate of 20% with vaccine coverage of 30% vaccination coverage of sexually active susceptible individuals.

Hamza, Samaila, Yakasai, Babashani, Borodo and Habib (2013) conducted a research to determine the prevalence of HBV and HCV infections among HIV-infected patients at Aminu Kano Teaching Hospital, Kano. The prevalence of HIV/HBV co-infection estimated was 12.5% and 1.6% for HIV/HCV co-infection. The prevalence of HIV/HBV co-infection was similar to the results obtained by Otegbayo, et al., (2008) and Adewole, et al., (2009). The result of their study further confirmed that HBV is a major co-morbid infection and a threat to HIV/AIDS patients in Nigeria.

Kamyad, Akbari, Heydari and Heydari (2014) proposed and studied S-E-I-C-R model of hepatitis B virus infection with two controls: vaccination and treatment. The results of the numerical simulation shown that optimal combination of vaccination and treatment is the most effective way to control hepatitis B virus infection.

Abdullahi (2015) developed and studied 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. He explored basic reproduction number $R_0$of disease-free equilibrium, and its local stability analysis. His results shown that the model is locally asymptotically stable (LAS) if the basic reproduction number $R_0 < 1$. His simulation results further revealed that, HBV transmission would be better curbed via applications of vaccination and behavioural change.

The transmission dynamics of HBV in the context of vaccination, where only newborn newborns are immunized as a control mechanism against HB, were studied using a deterministic MSEIR model. The disease-free equilibrium state was discovered, and the Routh-Hurwitz theorem was used to assess its local stability. Their investigation of the disease-free equilibrium state's stability revealed that it is locally asymptotically stable. It was also discovered that hepatitis B can be completely eradicated if the sum of latently infected individuals' recovery rates, the rate at which latently infected individuals become actively infected by the HB disease, and the natural death rate of individuals in the population is kept below a certain threshold. This provides the conditions under which HB may be eradicated totally in any community (Blessing & Iyama, 2017).

From the above literatures reviewed no work focused in combining vaccine on the spot, treatment, sanitarium and immigration factors into the dynamic of HBV and to study their effect on the dynamics of HBV. In this work, we set out to incorporate these factors to study the dynamics of HBV under the influence of these factors with a review to proffer a better control strategy.

## 3.0 HBV Model Formulation

We present standard SEIR model for the transmission dynamics of HBV in Nigeria, we partitioned the population into 7 compartments namely: Immunized M(t), Susceptible S(t), Latent L(t), Infectious I(t), Senatorium $S_1(t)$, Vaccinated V(t) and Recovered R(t). Recruitment into immunized M (t) compartment is done via the coming of immunized babies (new born) at a proportion cρ the coming individuals into this population are immunized through vaccination. The vaccine efficacy in their system reduces due to expiration of the vaccine at the rate$\phi$ and also asa result of natural death at the rate μ. The susceptible(t) component of the population grows due to coming in of new born babies not immunized against HBV at the rate $(1 - c)\rho$, recovered individuals are recruited into this population

after recovery from HBV, recovered become susceptible at the rateθ,vaccination expiration of adult at λrate, $I_s$ as an immigration rate. This component decreases due to the latent infection of individuals at the rateα, adult vaccinated against HBV at rate ω, and due to death from natural causes at the rate of μ. The population of the vaccinated grown adult vaccinated against HBV at rate ω and decreases with vaccination expiration of adult at rate λ alongside death from natural causes at the rate of μ. The population of the latent component grows as a result of infection of individuals in the susceptible class at the rate of αand $I_L$rate of immigration. This class reduces due to the progression of latently infected individuals to active HB infection at the rate of β, the successful treatment and cure of latent HB patients at the rate of δand as a result of death from natural causes at the rate of μ. The infectious compartment increases due to the progression of latently infected individuals to active HB infection at the rate of β and $I_I$rate of immigration. The component reduces as a result of successful cure of infectious HB patients at the rate of γ, death as a result of active HB infection at the rate of πand also due to death from natural causes at the rate of μ. The population of sanatorium increases by infected individuals movement at the rate σ.The population reduces by εrate at which sanitary infected individuals from HBV and as a result of death from natural causes at the rate μ. Lastly, recovered component grows as a result of successful treatment and curer of latent HB patients at the rate δand εrate at which sanitary infected individuals recovered from HBV and that of infectious HB patient at the rateγ. The recovered compartment decreases due to the fact that recovered individuals are not immunized against the infection and so they return to the susceptible class at the rate of θand also as the result of death from natural cause at the rate μ. The above assertions can be seen diagrammatically below.

### 3.1 Model Diagram

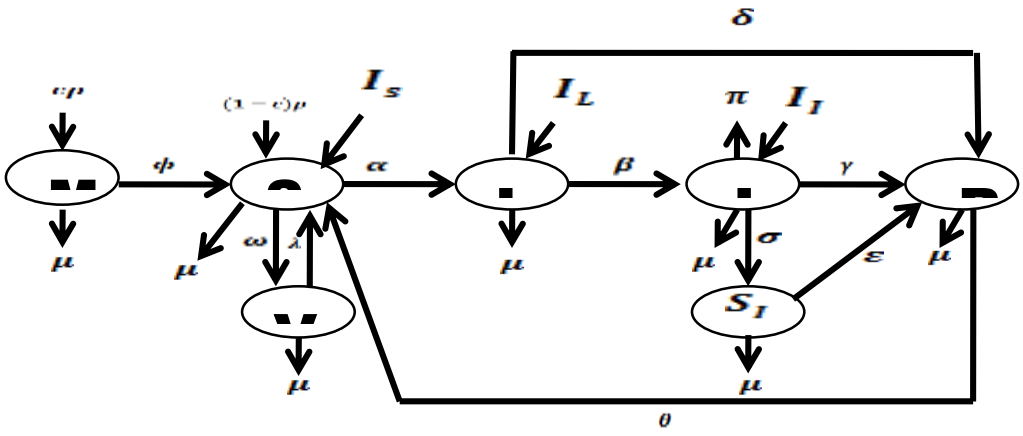


Fig. 1: Epidemiological flow diagram of the Model

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## 4. Model Equations

$$\frac{dM}{dt} = cp - (\phi + \mu)M \quad (1)$$

$$\frac{dS}{dt} = (1-c)\,p + I_s + \phi M + \theta R + \lambda V - (\alpha I + \omega + \mu)S \quad (2)$$

$$\frac{dV}{dt} = \omega S - (\lambda + \mu)V \quad (3)$$

$$\frac{dL}{dt} = \alpha SI + I_L - (\delta + \beta + \mu)L \quad (4)$$

$$\frac{dI}{dt} = \beta L + I_I - (\pi + \sigma + \gamma + \mu)I \quad (5)$$

$$\frac{dS_I}{dt} = \sigma I - (\varepsilon + \mu)S_I \quad (6)$$

$$\frac{dR}{dt} = \gamma I + \varepsilon S_I + \delta L - (\theta + \mu)R \quad (7)$$

Table 1: State Variables and Parameter Descriptions

| State Variable/Parameter | Description |
|---|---|
| M | Vaccinated infants at birth at time t |
| S | Susceptible individuals at time t |
| L | Latent individuals at time t |
| I | Infected individuals at time t |
| R | Recovered individuals at time t |
| V | Portion of those who escape vaccination at birth at time t |
| $S_1$ | Sanatorium compartment for infected individuals at time t |
| k | rate at which susceptible individuals become latently infected by HB |
| μ | the rate at which latently infected individuals become actively infected |
| ψ | rate at which actively infected individuals recover from HB infection |
| η | HB-induced mortality/death rate |
| β | natural mortality/death rate |
| p | Population of new births joining the population |
| N | the total population size |
| cp | the proportion of new births that have been immunized through vaccination |
| π | Loss of temporary immunity derived from recovery |
| ω | Proportion of adult being vaccinated against HBV |
| λ | Rate of expiration of the vaccine efficacy in Adult |
| ε | Rate at which sanitary infected individuals recovered from HBV |
| δ | Proportion of the exposed that recovered from HBV |
| θ | Rate of the recovered joining the susceptible |
| $I_{i,i=S,L,I}$ | Rate immigrant that are joining the latent class, susceptible class and infected |
| σ | Proportion of the infected that are taking into sanitary class |
| β | Rate of progression from latency to infected compartment |

## 5.Basic Analysis

It is important to show that the model (1) - (7) is mathematically and epidemiologically well posed before analyzing it. This is done below:

## 6. Positivity of the Solution of the Model

The model equations (1)-(7) monitors the dynamic within the human population, thus, all its associated parameters are nonnegative. It is instructive to show that all the state variables of the model are nonnegative at all time $t$. Hence, the following holds:

### Theorem 1

The solution set $\{M,S,V,L,I,S_I,R\}$ of the model (1)-(7) with nonnegative initial conditions remain nonnegative for all time $t>0$

### Proof

Given that the initial conditions of the model (1)-(7) are $M(0),S(0),V(0),L(0),I(0)$ , $S_I(0)$ and $R(0)$ are nonnegative. Then, the first equation of the system with only terms related to the state variable are picked, thus, we have:

$$\frac{dM}{dt}\geq -(\phi+\mu)M \tag{8}$$

Solve equation (4.1) by separation of variable to have

$$M(t)\geq M(0)e^{-(\phi+\mu)t}>0, \qquad \forall t>0$$

Again, the second equation of the system (1)-(7) becomes:

$$\frac{dS}{dt}\geq -(\alpha I+\omega+\mu)S \tag{9}$$

By separation of variables, equation (9) becomes:

$$S(t)\geq S(0)e^{-\left(\omega+\mu+\alpha\int I(\omega)d\omega\right)t}>0, \qquad \forall t>0$$

Similarly, the third equation and others have the following:

$$\frac{dV}{dt}\geq -(\lambda+\mu)V \tag{10}$$

$$\frac{dL}{dt}\geq -(\delta+\beta+\mu)L \tag{11}$$

$$\frac{dI}{dt}\geq -(\pi+\gamma+\sigma+\mu)I \tag{12}$$

$$\frac{dS_I}{dt} \geq -(\varepsilon+\mu)S_I \tag{13}$$

$$\frac{dR}{dt} \geq -(\theta+\mu)R \tag{14}$$

Solving equation (10) to (14) to obtain the following:

$$V(t) \geq V(0)e^{-(\lambda+\mu)t} > 0, \quad \forall t > 0$$

$$L(t) \geq L(0)e^{-(\delta+\beta+\mu)t} > 0, \quad \forall t > 0$$

$$I(t) \geq I(0)e^{-(\pi+\gamma+\sigma+\mu)t} > 0, \quad \forall t > 0$$

$$S_I(t) \geq S_I(0)e^{-(\varepsilon+\mu)t} > 0, \quad \forall t > 0$$

$$R(t) \geq R(0)e^{-(\theta+\mu)t} > 0, \quad \forall t > 0$$

Hence, the complete proof.

## 7. Invariant region

### Lemma 1

The closet set $G = \left\{(M,S,V,L,I,S_I,R) \in \Re_+^7 : M+S+V+L+I+S_I+R \leq \frac{p+I_S+I_L+I_I}{\mu}\right\}$ is positively invariant and attracting with respect to the model equation (1)-(7)

### Proof

The invariant region is obtained from the bounded solution of the total population $N = M+S+V+L+I+S_I+R$

Add all the equations in the system to get

$$\frac{dN}{dt} = p+I_S+I_L+I_I-\mu N-\pi I$$

Then,

$$\frac{dN}{dt} \leq p+I_S+I_L+I_I-\mu N \tag{15}$$

The Solution of (4.8) is expressed as

$$N(t) \leq \frac{\Omega}{\mu}+\left(N(0)-\frac{\Omega}{\mu}\right)e^{-\mu t}$$

Where $\Omega = p+I_S+I_L+I_I$

$N(0)$ is the initial condition for $N(t)$ at $t=0$

Again, as $t \to \infty$, $N(t) \to \frac{\Omega}{\mu}$. This implies that $N(t)$ is bounded as $0 \leq N(t) \leq \frac{\Omega}{\mu}$. Hence, the feasible region of the model in the system (4.8) is nonnegative region is defined.

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$$G=\left\{(M,S,V,L,I,S_I,R)\in\Re_+^7 : N\le\frac{\Omega}{\mu}\right\}$$

## 8. Existence of Equilibrium Point

The HBV model is at equilibrium when the system of equation (1)-(7) is set to zero, we have:

$$\frac{dM}{dt}=\frac{dS}{dt}=\frac{dV}{dt}=\frac{dM}{dt}=\frac{dV}{dt}=\frac{dL}{dt}=\frac{dI}{dt}=\frac{dS_I}{dt}=\frac{dR}{dt}=0$$

Thus, equation (1)-(7) becomes

$$cp-Q_1M=0 \tag{16}$$

$$(1-c)p+I_s+\phi M+\theta R+\lambda V-\alpha S_I-Q_2S=0 \tag{17}$$

$$\omega S-Q_3V=0 \tag{18}$$

$$\alpha SI+I_L-Q_4L=0 \tag{19}$$

$$\beta L+I_I-Q_5I=0 \tag{20}$$

$$\gamma I-Q_6S_I=0 \tag{21}$$

$$\gamma I+\varepsilon S_I+\delta L-Q_7R=0 \tag{22}$$

Where $Q_1=\phi+\mu$, $Q_2=\omega+\mu$, $Q_3=\lambda+\omega$, $Q_4=\delta+\beta+\omega$, $Q_5=\pi+\delta+\gamma+\omega$, $Q_6=\varepsilon+\mu$ and $Q_7=\theta+\mu$

From equation (16), (18), (19) and (21) we have the following:

$$M=\frac{cp}{Q_1} \tag{23}$$

$$V=\frac{\omega S}{Q_3} \tag{24}$$

$$S_I=\frac{\gamma I}{Q_6} \tag{25}$$

$$L=\frac{\alpha S_I+I_L}{Q_4} \tag{26}$$

Substitute equation (25) into (20), we have:

$$\left.\begin{aligned}&\beta\left(\frac{\alpha S_I+I_L}{Q_4}\right)+I_I-Q_5I=0\\&I=\frac{\beta(\alpha S_I+I_L)+Q_4I_I}{Q_4Q_5}\\&I=\frac{\beta\alpha S_I+\beta I_L+Q_4I_I}{Q_4Q_5}\end{aligned}\right\} \tag{27}$$

Substitute equation (25) and (26) into (22), we have:

$$\gamma I+\varepsilon\left(\frac{\delta I}{Q_6}\right)+\delta\left(\frac{\alpha S_I+I_I}{Q_4}\right)-Q_7R=0$$

$$\gamma Q_6Q_4I+\delta\varepsilon Q_4I+\delta\alpha Q_6S_I+\delta Q_6I_I-Q_4Q_6Q_7R=0$$

$$R=\frac{Q_4\left(\gamma Q_6+\delta\varepsilon\right)I+\delta\alpha Q_6S_I+\delta Q_6I_I}{Q_4Q_6Q_7} \quad (28)$$

Substitute equation (23), (28) and (24) into equation (17) to get:

$$(1-c)p+I_s+\phi\left(\frac{cp}{Q_1}\right)+\theta\left\{\frac{Q_4\left(\gamma Q_6+\delta\varepsilon\right)I+\delta\alpha Q_6S_I+\delta Q_6I_I}{Q_4Q_6Q_7}\right\}+\lambda\left(\frac{\omega S}{Q_3}\right)-\alpha SI-Q_2S=0$$

$$\{(1-c)p+I_s-\alpha SI-Q_2S\}Q_1Q_3Q_4Q_6Q_7+\phi cpQ_3Q_4Q_6Q_7+\phi cpQ_3Q_4Q_6Q_7+\theta Q_1Q_3\{Q_4(\gamma Q_6+\delta\varepsilon)+\delta Q_6(\alpha SI+I_L)\}+Q_1Q_4Q_6Q_7\lambda\omega S=0 \quad (29)$$

To make S the subject of the formula in equation (29) first, we rewrite equation (29) as below:

$$\{(1-c)p+I_s\}Q_1Q_3Q_4Q_6Q_7-Q_1Q_2Q_3Q_4Q_6Q+\theta Q_1Q_3\{Q_4(\gamma Q_6+\delta\varepsilon)+\delta Q_6(\alpha SI+I_L)\}-\alpha SIQ_1Q_2Q_3Q_4Q_6Q_7-Q_1Q_2Q_3Q_4Q_6Q_7S+\theta\alpha\delta Q_1Q_3Q_6SI+Q_1Q_4Q_6Q_7\lambda\omega S=0 \quad (30$$

Thus, equation (30) becomes:

$$S=\frac{\theta Q_1Q_3Q_4\left(\gamma Q_6+\delta\varepsilon\right)I+Q_1Q_3Q_6\left[Q_4Q_7+\theta\delta I_L\right]+Q_3Q_4Q_6Q_7\left(pQ_1(1-c)+\phi cp\right)}{\alpha Q_1Q_3Q_6I\left(Q_4Q_7-\theta\delta\right)+Q_1Q_4Q_6Q_7\left(Q_2Q_3-\lambda\omega\right)} \quad (31)$$

For simplicity in manipulating equation (31) is written as

$$S=\frac{b_1I+b_2+b_3}{b_4I+b_5} \quad (32)$$

Where:

$b_1=\theta Q_1Q_3Q_4\left(\gamma Q_6+\delta\varepsilon\right)$, $b_2=Q_1Q_3Q_6\left[Q_4Q_7+\theta\delta I_L\right]$, $b_3=Q_3Q_4Q_6Q_7\left(pQ_1(1-c)+\phi cp\right)$

$b_4=\alpha Q_1Q_3Q_6I\left(Q_4Q_7-\theta\delta\right)$ and $b_5=Q_1Q_4Q_6Q_7\left(Q_2Q_3-\lambda\omega\right)$

Substitute equation (32) into equation (27), we obtain:

$$I=\frac{\alpha\beta\left(b_1I+b_2+b_3\right)+\left(b_4+I+b_3\right)\left(\beta I_L+Q_4I_I\right)}{Q_4Q_5\left(b_4I+b_3\right)}$$

$$Q_4Q_5I\left(b_4I+b_3\right)=\alpha\beta b_1I^2+\alpha\beta I\left(+b_2+b_3\right)+b_4I\left(\beta I_L+Q_4I_I\right)+b_5\left(\beta I_L+Q_4I_I\right)$$

$$\left(b_4Q_4Q_5+\alpha\beta b_1\right)I^2-I\left[\alpha\beta\left(b_2+b_3\right)+b_4\left(\beta I_L+Q_4I_I\right)-b_5Q_4Q_5\right]-b_5\left(\beta I_L+Q_4I_I\right)=0 \quad (33)$$

If we conveniently resolve equation (33),we have:

$$A_2I^2-A_1I-A_0=0 \quad (34)$$

Where:

$$A_2=b_4Q_4Q_5-\alpha\beta b_1=\mu Q_5Q_6\left(Q_7+\delta\right)+p\left\{\mu\left(\gamma Q_7+\varepsilon\gamma+\gamma Q_6\right)+Q_6Q_7\pi\right\}>0$$

$$A_1=\alpha\beta b_3+\alpha pQ_1Q_3Q_4Q_6Q_7I_L+\alpha Q_1Q_3Q_4Q_6\left[\beta Q_7I_s+\left(\mu\left(Q_4+\theta\beta\right)I_I\right)\right]-b_5Q_4Q_5$$

$$A_0=b_5\left(\beta I_L+Q_4I_I\right)>0$$

Since all the parameters of the model are nonnegative, then it is important to note that $A_2 > 0$ and $A_0 > 0$. Thus by Descartes rules of sign, equation (34) has a unique positive root regardless of the sign of $A_1$. Hence, the following results are established.

## 9. Disease-Free Equilibrium Point and Computation of Reproduction Number

The equilibrium point that corresponds to the disease free equilibrium which is defines as a point at which disease is absent (hence all infected classes are zero) denoted by $E_0$ is given by (34)

Then by equation (34) and $I_L = I_I = 0$, equation (23), (24) and (26) becomes the following respectively

$$S^* = 0 \tag{35}$$

$$L^* = 0 \tag{36}$$

$$R^* = 0 \tag{37}$$

Similarly, equation (32) becomes

$$S^* = \frac{Q_3\left[Q_1 I_s + p\left(\theta + \mu(1-c)\right)\right]}{\mu Q_1\left(Q_2 + \lambda\right)} = \frac{(\lambda+\mu)(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)} \tag{38}$$

Substitute equation (38) into equation (24) to obtain

$$V^* = \frac{\omega\left[Q_1 I_s + p\left(\phi + \mu(1-c)\right)\right]}{\mu Q_1\left(Q_2 + \lambda\right)} = \frac{\omega(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)} \tag{39}$$

From equation (23)

$$M^* = \frac{cp}{Q_1} = \frac{cp}{\mu+\phi} \tag{40}$$

The component of disease-free equilibrium point is

$E_0\left(M^*, S^*, V^*, 0, 0, 0, 0\right)\left(\frac{cp}{\mu+\phi}, \frac{(\lambda+\mu)(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)}, \frac{\omega(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)}, 0, 0, 0, 0\right)$ are obtained by equations (40), (38), (39), (36), (35) and (37).

The local stability $E_0$ will be investigated using reproduction number ($R_0$). The reproduction number ($R_0$)is define as the average number of secondary HBV infection generated by infected individual when introduced into a susceptible population. The method for establishing the reproduction number ($R_0$) is next generation matrix. As outlined in Venn den Driessche and Wartmought (2002), where matrix $F$ and $V$ denote the new infection term and the transition term

associated with the system of equations (1 -7) in the absence of HBV infective immigrant at $E^*$

$F = \begin{pmatrix} 0 & \alpha S^* \\ 0 & 0 \end{pmatrix}$ and $V = \begin{pmatrix} Q_4 & 0 \\ -\beta & Q_5 \end{pmatrix}$

The inverse matrix $V$ becomes

$$V^{-1} = \begin{pmatrix} \frac{1}{Q_4} & 0 \\ \frac{\beta}{Q_4 Q_5} & \frac{1}{Q_5} \end{pmatrix}$$

It follows that $R_0 = \rho\left(FV^{-1}\right)$ is obtained as

$R_0 = \frac{\alpha\beta S^*}{Q_4 Q_5}$ which translate to

$$R_0 = \frac{\alpha\beta Q_3\left[Q_1 I_s + p\left(\theta + \mu(1-c)\right)\right]}{\mu Q_1 Q_4 Q_3\left(Q_2 + \lambda\right)} = \alpha\beta\left[\frac{(\lambda+\mu)(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)}\right] \quad (41)$$

## 10. Existence of Endemic Equilibrium Point in the absence of Immigration in Latency and Infected Compartments.

Let $E^*$ denote the endemic equilibrium (That is an equilibrium where at least one of the infected compartment is non-zero) of the model (1) – (7) with $I_L = I_I = 0$.

Thus (19) corresponds to the endemic equilibrium such that

$$I^* = \frac{B_0}{A_2}$$

$$I^* = \frac{\alpha\beta Q_3 Q_4 Q_6 Q_7 p\left\{\phi + \mu(1-c) + \alpha\beta Q_1 Q_3 Q_4 Q_6 Q_7 I_s - \mu Q_1 Q_4^2 Q_5 Q_6 Q_7\left(Q_2 + \lambda\right)\right\}}{A_2}$$

$$I^* = \frac{\mu Q_1 Q_4^2 Q_5 Q_6 Q_7\left(Q_2 + \lambda\right)\left\{\alpha\beta Q_3 Q_4 Q_6 Q_7\left(Q_1 I_s + p\left(\phi + \mu(1-c)\right)\right) - 1\right\}}{A_2} \quad (41)$$

Substituting equation (25) into equation (41) to have

$$I^* = \frac{\mu Q_1 Q_4^2 Q_5 Q_6 Q_7\left(Q_2 + \lambda\right)\left(R_0 - 1\right)}{A_2} \quad (42)$$

Then by (22) the following results are obtained from (23)

$$S_I^* = \frac{\gamma I^*}{Q_6} \quad (43)$$

From equation (32)

$$S_I^* = \frac{b_1 I^* + Q_3 Q_4 Q_6 Q_7 \left[ Q_1 I_s + p\left(\phi + \mu(1-c)\right)\right]}{b_4 I^* + b_5} \quad (44)$$

From equation (26)

$$L^* = \frac{\alpha S^* I^*}{Q_4} \quad (45)$$

Since $I_L = 0$

From (28)

$$R^* = \frac{Q_4\left(\gamma Q_6 + \varepsilon S\right) I^* + \alpha\delta Q_6 S^* I^*}{Q_4 Q_6 Q_7} \quad (46)$$

Since $I_L = 0$

From equation (23)

$$M^* = \frac{cp}{Q_1} \quad (47)$$

And

$$V^* = \frac{\omega S^*}{Q_3} \quad (48)$$

The component of the epidemic equilibrium of the model with $I_I = I_L = 0$ denoted by $E^*\left(M^*, S^*, V^*, L^*, I^*, R^*\right)$ must satisfy (47 )- (48).

## 11. Local Stability Analysis of Disease-Free Equilibrium Point $E^*$

Theorem: The disease free equilibrium point $E^*$ of the system of equation (1) – (7) in the absence of HBV infective immigrants is locally asymptotically stable at $R_0 < 1$and unstable otherwise. The variation matrix of model (1) – (7) corresponding to $E^*$

$$J\left(E^*\right) = \begin{pmatrix} -Q_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ Q & -Q_2 & \lambda & 0 & -\alpha S^* & 0 & 0 \\ 0 & \omega & -Q_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -Q_4 & -\alpha S^* & 0 & 0 \\ 0 & 0 & 0 & \beta & -Q_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -Q_6 & 0 \\ 0 & 0 & 0 & \delta & \alpha & \varepsilon & -Q_7 \end{pmatrix} \quad (49)$$

The characteristics equation (49) is denoted by

$$\left|J(E^*) - q_I I\right| = 0 \quad (50)$$

Where:

$q_I$ is eigen value

$I$ is an identity matrix of order 7 by 7

It obvious that equation (50) reduces to equation (51) below

$$\left|J(E^*)-q_I I\right|=\begin{vmatrix} -Q_1-q & 0 & 0 & 0 & 0 & 0 & 0 \\ Q & -Q_2-q & \lambda & 0 & -\alpha S^* & 0 & 0 \\ 0 & \omega & -Q_3-q & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -Q_4-q & -\alpha S^* & 0 & 0 \\ 0 & 0 & 0 & \beta & -Q_5-q & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha & -Q_6-q & 0 \\ 0 & 0 & 0 & \delta & \alpha & \varepsilon & -Q_7-q \end{vmatrix}=0 \quad (51)$$

$$-Q_1-q\begin{vmatrix} -Q_2-q & \lambda & 0 & -\alpha S^* & 0 & 0 \\ \omega & -Q_3-q & 0 & 0 & 0 & 0 \\ 0 & 0 & -Q_4-q & -\alpha S^* & 0 & 0 \\ 0 & 0 & \beta & -Q_5-q & 0 & 0 \\ 0 & 0 & 0 & \gamma & -Q_6-q & 0 \\ 0 & 0 & \delta & \alpha & \varepsilon & -Q_7-q \end{vmatrix} \quad (52)$$

Thus, the first equation $q_1$ is gotten as

$q_1=-Q_1<0$

Evaluate the reduce determinant (that is order 6 × 6) across the first column of (52) to have

$$-Q_2-q\begin{vmatrix} -Q_3-q & 0 & 0 & 0 & 0 \\ 0 & -Q_4-q & -\alpha S^* & 0 & 0 \\ 0 & \beta & -Q_5-q & 0 & 0 \\ 0 & 0 & \gamma & -Q_6-q & 0 \\ 0 & \delta & \alpha & \varepsilon & -Q_7-q \end{vmatrix} \quad (53)$$

$$-\omega\begin{vmatrix} \lambda & 0 & -\alpha S^* & 0 & 0 \\ 0 & -Q_4-q & 0 & 0 & 0 \\ 0 & \beta & -Q_5-q & 0 & 0 \\ 0 & 0 & 0 & -Q_6-q & 0 \\ 0 & \delta & \alpha & \varepsilon & -Q_7-q \end{vmatrix}=0 \quad (54)$$

Further simplification of (53)

$$(-Q_2-q)(-Q_3-q)\begin{vmatrix} -Q_4-q & -\alpha S^* & 0 & 0 \\ \beta & -Q_5-q & 0 & 0 \\ 0 & \gamma & -Q_6-q & 0 \\ \delta & \alpha & \varepsilon & -Q_7-q \end{vmatrix}$$

$$-\omega\lambda\begin{vmatrix} -Q_4-q & -\alpha S^* & 0 & 0 \\ \beta & -Q_5-q & 0 & 0 \\ 0 & \gamma & -Q_6-q & 0 \\ \delta & \alpha & \varepsilon & -Q_7-q \end{vmatrix}=0 \quad (55)$$

$$-(Q_2+q)(Q_3+q)-\omega\lambda\begin{vmatrix} -Q_4-q & -\alpha S^* & 0 & 0 \\ \beta & -Q_5-q & 0 & 0 \\ 0 & \gamma & -Q_6-q & 0 \\ \delta & \alpha & \varepsilon & -Q_7-q \end{vmatrix}=0 \quad (56)$$

$$-(Q_6+q)(Q_7+q)\{(Q_2+q)(Q_3+q)-\omega\lambda\}\{(Q_4+q)(Q_5+q)-\alpha\beta S^*\}=0 \quad (57)$$

The second $q_2$ and third $q_3$ are obtained as

$q_2=-Q_6<0$

$q_3=-Q_7<0$

The equation (57) becomes:

$$\{q^2+q(Q_2+Q_3)+Q_2Q_3-\omega\lambda\}\{q^2+q(Q_4+Q_5)-\alpha\beta S^*\}=0 \quad (58)$$

For convenience sake (51) is written as

$$\{q^2+qB_1+B_2\}\{q^2+qB_3-B_4\}=0 \quad (59)$$

Where

$B_1=Q_2+Q_3>0$

$B_2=Q_2Q_3-\omega\lambda>0$, since $Q_2Q_3-\omega\lambda=\mu(\mu+\omega+\lambda)$

$B_3=Q_4+Q_5$

$$B_4=Q_4+Q_5-\alpha\beta S^*=Q_4Q_5\left(1-\frac{\alpha\beta S^*}{Q_4Q_5}\right)$$

$=Q_4Q_5(1-R_0)>0$, if and only if $R_0<1$

Now by Routh-Hurwitz criteria the quadratic expression in each curly bracket of (59) will be stable that is real part of Eigen value if $B_i>0$, $i=1,2,3,4$

It is readily seen that $B_1>0, B_2>0$ and $B_3>0$ but $R_0<1$. Thus, the $E^*$ of system of equations (1) – (7) in the absence of HBV infective immigrant is locally asymptotically stable whenever $R_0<1$. This completes the proof.

## 12. Global Stability Analysis of Disease Free Equilibrium Point $E^*$

We used Castillo-Chavez, Feng and Huang (2002) method to investigate the global asymptotic stability of the disease-free and infestation-free steady state. In this section, we highlight two conditions that if met, guarantee the global asymptotic

stability of the disease-free and infestation-free steady state. First, the system of equation (1) - (7) must be written in the form:

$$\left.\begin{aligned}\frac{dX}{dt}&=H(X,Z)\\ \frac{dZ}{dt}&=G(X,Z),G(X,Z)=0\end{aligned}\right\} \qquad (60)$$

Where $X\in\Re^m$ denotes (its components) the number of uninfected individuals and $Z\in\Re^n$ denotes (its components) the number of infected individuals. $E_1^*=(X^*,0)$ denotes the disease-free and infestation-free steady state of the system (1)-(7). The conditions $(H_1)$ and $(H_2)$ below must be met to guarantee global asymptotic stability.

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

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

Where $P=D_1G(X^*,0)$ is an M-matrix (the off diagonal elements of $P$ are nonnegative) and $\Omega$ is the region where the modified model makes biological sense.

If the system of equation (1) - (7) satisfies the above two conditions the following theorem holds.

**Theorem 2.** The fixed point $E_1^*=(X^*,0)$ is a globally asymptotic stable (G.A.S) of disease-free and infestation-free steady state of the system (1)-(7) provided that $R_0<1$ is Locally Asymptotically Stable (L.A.S) and that $(H_1)$ and $(H_2)$ are satisfied.

**Proof:**

Let $X=(M,S,V,R)$, $Z=(L,I,S_I)$, $X\in\Re^4$, $Z\in\Re^4$, $E_0=(X^*,0)$ and

$$X^*=\left(\frac{cp}{\mu+\phi},\frac{(\lambda+\mu)(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)},\frac{\omega(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)},0\right)$$

Thus, the uninfected compartments from the system of equation (1)-(7), we have:

$$H(X,Z)=\begin{bmatrix}cp-(\phi+\mu)M\\ (1-c)p+\phi M+\lambda V-(\alpha I+\omega+\mu)S\\ \omega S-(\lambda+\mu)V\\ 0\end{bmatrix} \qquad (61)$$

Evaluating (61) at the disease-free equilibrium point, we have:

$$H(X,0)=\begin{bmatrix}\frac{(\lambda+\mu)(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)}\\0\end{bmatrix} \quad (62)$$

Taking the infected compartment of the system (1)-(7), we have:

$$G(X,Z)=\begin{bmatrix}\alpha SI+I_L-(\delta+\beta+\mu)L\\ \beta L+I_I-(\pi+\sigma+\gamma+\mu)I\\ \sigma I-(\varepsilon+\mu)S_I\end{bmatrix} \quad (63)$$

Taking the partial derivatives of the state variables in an infected compartment sand evaluating equation (63) at disease-free and infestation-free steady state, it gives:

$$P=\begin{pmatrix}-(\delta+\beta+\mu) & \alpha S & 0\\ \beta & -(\pi+\sigma+\gamma+\mu) & 0\\ 0 & \sigma & -(\varepsilon+\mu)\end{pmatrix} \quad (64)$$

Multiply equation (64) with $Z=(L,I,S_I)^T$, we have:

$$PZ=\begin{pmatrix}-(\delta+\beta+\mu)L+\alpha SI\\ \beta L-(\pi+\sigma+\gamma+\mu)I\\ \sigma I-(\varepsilon+\mu)S_I\end{pmatrix} \quad (65)$$

Thus, subtracting (65) from (63), we have:

$$G(X,Z)=PZ-\hat{G}(X,Z)=\begin{bmatrix}I_L\\ I_I\\ 0\end{bmatrix}$$

Since $I_L$ and $I_I$ are strictly positive. Then, $\hat{G}(X,Z)\geq 0$. The global stability of $X^*=\left(\frac{(\lambda+\mu)(p(-c\mu+\mu+\phi)+I(\mu+\phi))}{\mu(\mu+\phi)(\lambda+\mu+\omega)},0\right)$ of the system (1)-(7). Hence, the two conditions have been met and thus $E_1^*$ is Globally Asymptotically Stable (G.A.S).

## 13. Numerical Experiments of the Modified Model

In order to investigate the impact of using vaccination or sanatorium or combination of both control strategies on fighting HBV, we used the baseline values for the parameters as in Table 4.1 and implored the use of MATLAB Codes in computing the numerical values. The following cases are hitherto investigated:

i. A case where vaccination is used only

ii. A case where sanatorium is used only

iii. A case where both vaccination and sanatorium are used

Table 4.1: The Baseline Values for Variables and Parameters of the Models.

| Parameter | parameter description | value | Source |
|---|---|---|---|
| $\omega$ | Proportion of adult being vaccinated | 0 - 1 | Pang *et al.*, (2010) |
| $\lambda$ | Rate of vaccination expiration in adult | 0.045 | Shepard *et al.*, (2006) |
| $\varepsilon$ | Rate of sanitary individuals recovering | 0.5 | Assumed |
| $\delta$ | Rate of latent individuals recovering | 0.001 | Assumed |
| $\theta$ | Rate of recovered becoming susceptible | 0.3 | Assumed |
| $I_i, i = S, L, I$ | Rate of immigrant into susceptible, Latent and Infected class | 0.1-0.9 | Altaf Khan et al 2013 |
| $\sigma$ | Rate of infected joining sanatorium class | 0.6 | Assumed |
| $\beta$ | Proportion of latency becoming infected | 6 | Medley *et al.*, (2001) |
| $\gamma$ | Rate at which infected recover | 0.025 | Medley *et al.*, (2001) |
| $\pi$ | Disease induced death rate | 0.007 | Abdulrahman *et al.*, (2013a) |
| $\mu$ | Natural death rate for all the subpopulation | 0.011 | Abdulrahman *et al.*, (2013a) |
| $c$ | Proportion of immunized babies | 0.8 | Assumed |
| $\phi$ | Rate of vaccination expiration in babies | 0.055 | Assumed |
| $\rho$ | population of new births joining the population N | 0.5 | Assumed |
| $\alpha$ | Transmission coefficient | 0.85 | Edmunds *et al.*, (1996) |

## 14. The Impact of Vaccination Only on The Dynamics of The Populations of The Modified Model

Vaccination of the susceptible individual is one among the available control strategies that we have in curbing a menace of HBV. As such, a parameter value for vaccination is varied to observe the behavior of the population curves when simulation is taken. From figure 1 to figure 2, they indicate that, the infected population is significantly higher than the other population densities even when vaccination is administered in relatively small amount. From figure 3, when the vaccination rate is increased to $\omega = 0.9$, the infected population has drastically declined.

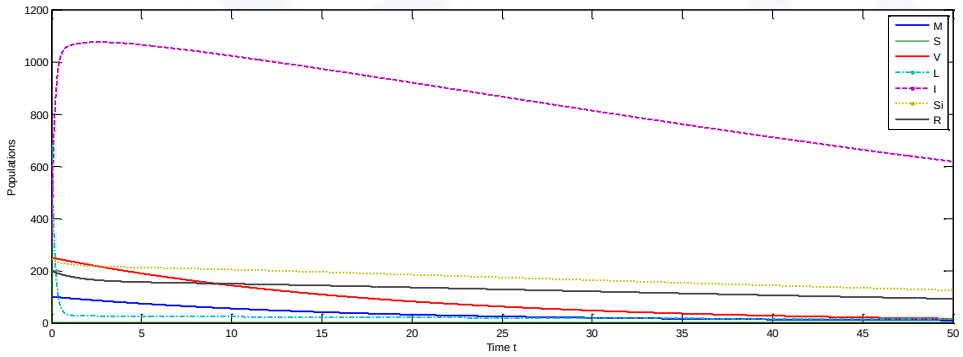


Figure 1: The impact of vaccination on the dynamics of the population with $\omega = 0.1$

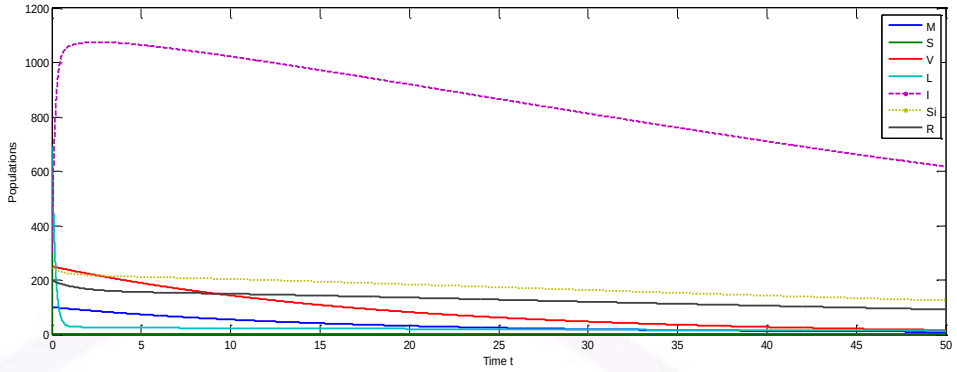


Figure 2: The impact of vaccination on the dynamics of the population with $\omega = 0.2$

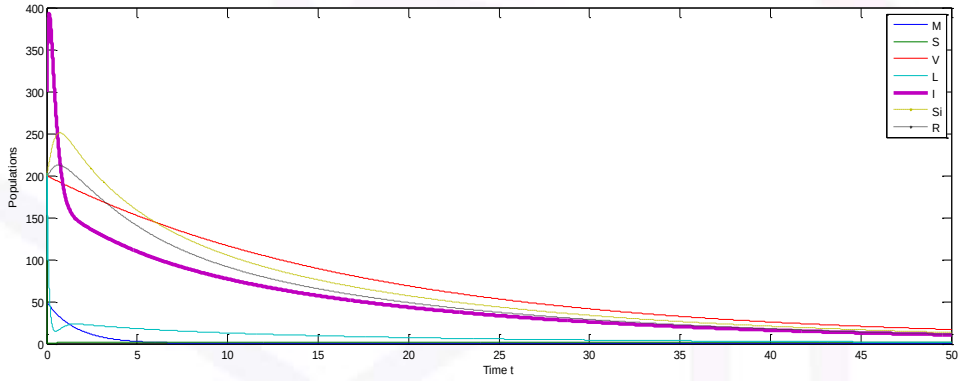


Figure 3: The impact of vaccination on the dynamics of the population with $\omega = 0.9$

## 15. The Impact of Vaccination And Sanitarium on The Dynamics of The Population of The Modified Model

Combining both vaccination and sanitarium as strategies to curb the menace of HBV, we vary these parameter values and simulated the scenarios as presented in figure 4 to figure7. In figure 4, more infected are put on sanitary and less vaccine is administered in a susceptible population. But, the infected population drop slightly below sanatorium population and above all other subpopulations (i.e vaccinated, susceptible, recovered, latent and immunized). Similarly, in figure 5, sanatorium parameter is increased while holding vaccination parameter constant. It shows that more recovered and sanitary individuals sharply grow above infected individuals, but, vaccinated individuals are relatively minute. In figure 6, more vaccination rate is considered than the rate of sanatorium. The recovered and sanatorium curves sharply grow while infected curve drop sharply

and converge to zero. As for the vaccinated curve drop slightly and then set to a steady level above the recovered curve. Finally, from figure 7, both vaccination and sanitorium rates were increased significantly at the same or almost the same higher value. The dynamical behviour of the curves clearly indicated that, combining these two strategies at some significantly higher rate would go along in curbing the menance of HBV.

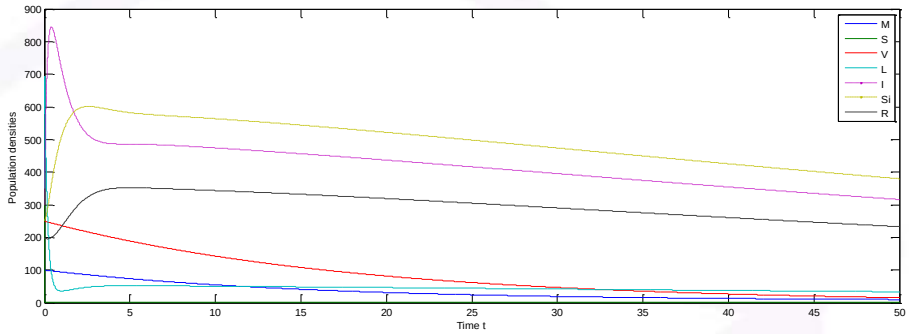


Figure 4: impact of vaccination and sanatorium on the dynamics of the population with $\sigma = 0.4$ and $\omega = 0.1$

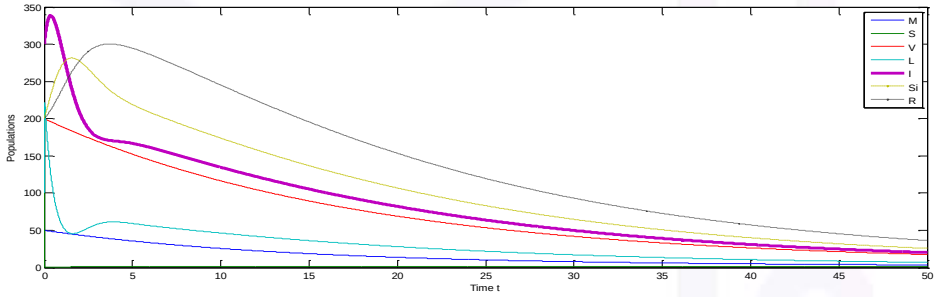


Figure 5: impact of vaccination and sanitorium on the dynamics of the population with $\sigma = 0.6$ and $\omega = 0.1$

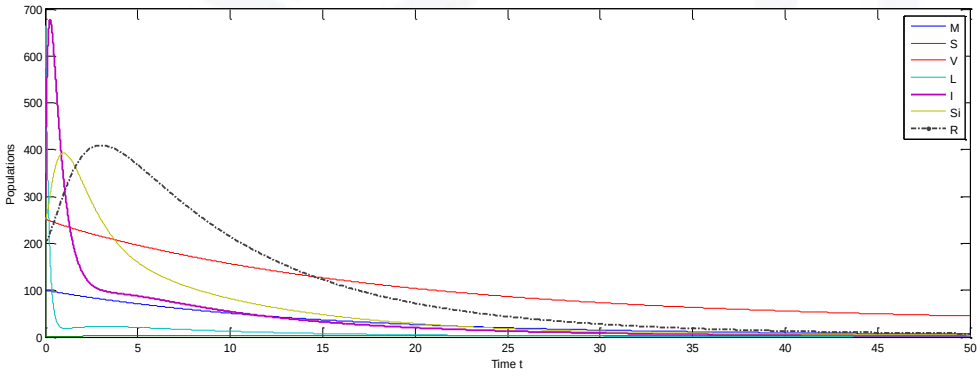


Figure 6: impact of vaccination and sanitorium on the dynamics of the population with $\sigma = 0.5$ and $\omega = 0.8$

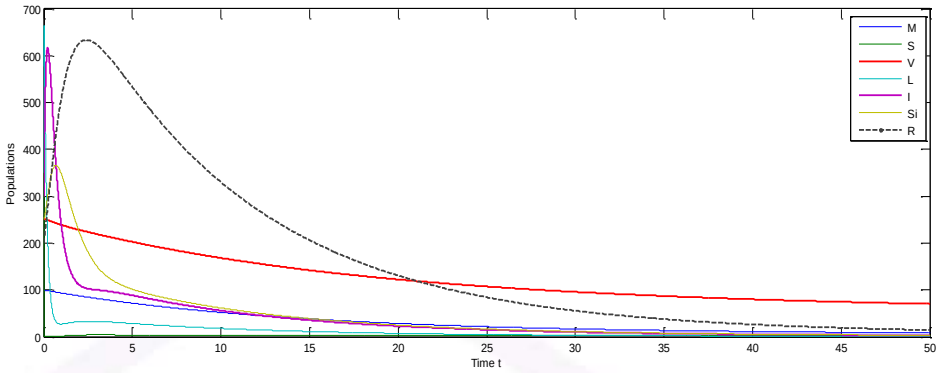


Figure 7: impact of vaccination and sanitorium on the dynamics of the population with $\sigma = 0.8$ and $\omega = 0.9$

## 16. Conclusion

Conclusively, HBV is a vaccine-preventable liver infection illness that is transmitted from an infected person to an uninfected person by blood, sperm, or other bodily fluids. Progression to active HBV in the community is epidemiologically significant, and interventions should focus on vaccination, treatment, and sanitorium on population dynamics. As a result, every effort should be taken to reduce the disease's contracting rate in any given population.

## 17. Recommendations

Vaccination and control of HBV could be achieved if the following measures are adopted:

1. Ensure that excellent, appropriate health hygiene procedures are in place.
2. In high-population areas, all individuals should be considered at risk for HBV infection and should be provided HBV vaccine if they have not already done so.
3. Health care professionals should establish standing orders to provide HVB vaccine to people who have not completed the vaccine series as part of normal treatments, and make HVB vaccination a standard component of STI and HIV/AIDS assessment and treatment.
4. HBV vaccine should be available in outreach and other venues where people at risk of HBV infection get assistance (e.g., needle-exchange programs, HIV testing sites, HIV prevention programs, and homeless shelters)
5. The government should start tracing newly affected people and nursing moms who have fallen behind on their payments so that they can get help.

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