ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

Mathematical Analysis of Hepatitis B Virus Dynamics with Vertical Transmission

Afolabi, Williams Oluwasegun[1]; Johnson, Adedayo Simeon[2]

[1]Department of Pure and Applied Mathematics Faculty of Pure and Applied Sciences Ladoke Akintola University of Science and Technology, Ogbomoso, Oyo State, Nigeria
[2]Department of Statistics Faculty of Natural and Applied Science Nasarawa State University, Keffi, Nasarawa State, Nigeria

Publication Date: 2025/12/01

Abstract: The dynamics of Hepatitis B virus remains endemic in the population despite the availability of a potent vaccine against the infection. Thus, there is the need for continuous efforts to eradicate the disease in order to forestall its spread. In this study, a model that explains the dynamics of Hepatitis B with vertical transmission was formulated, and an intervention to minimize its effects was proferred. A six-compartmental model comprising of a Susceptible S(t), Exposed E(t), Infected I(t), Treated T(t), Vaccinated V(t), and Recovered R(t) was formulated. The model's well-posedness was established through positivity, boundedness, and uniqueness of solutions. The disease-free equilibrium was determined by setting the infection force to zero, and the basic reproduction number (R_0) was derived using the next generation matrix method. Local stability analysis showed the disease-free equilibrium is stable when $R_0 < 1$. Presence of disease was confirmed by analyzing the endemic equilibrium, which was globally stable when $R_0 > 1$, as demonstrated using Lyapunov functions. The global stability analysis of endemic equilibrium point of the model was obtained using the Lyapunov functions and the sensitivity analysis identified the birth rate and vaccination rate as the most influential parameters on R_0, with birth rate increasing and vaccination decreasing disease spread. Graphical results highlighted that failure to vaccinate newborns significantly raises the risk of chronic infection. The study emphasizes vertical transmission as the primary and deadliest infection pathway. Consequently, it recommends enhanced public awareness, focused screening, and vaccination efforts, especially targeting pregnant women, to reduce Hepatitis B transmission and aid eradication.

Keywords: *Hepatitis B Virus, Analysis, Lipschitz Condition, Lyapunov Function, Disease.*

How to Cite: Afolabi, Williams Oluwasegun; Johnson, Adedayo Simeon (2025). Mathematical Analysis of Hepatitis B Virus Dynamics with Vertical Transmission. *International Journal of Innovative Science and Research Technology*, 10(11), 2062-2079. https://doi.org/10.38124/ijisrt/25nov207

I. INTRODUCTION

➢ *Background to the Study*

Hepatitis B virus (HBV) infection remains a significant global health challenge, particularly because of its complex transmission dynamics, including vertical transmission from mother to child. Hepatitis means inflammation of the liver. Hepatitis B is a contagious liver disease that results from infection with the Hepatitis B virus (CDC, 2013). The most common is viral infection, particularly by the hepatitis A, B, C, D, and E. Hepatitis can be acute or chronic, with acute lasting less than six months and chronic lasting longer. The condition varies in severity, ranging from mild, self-limiting inflammation to severe liver damage that may result in fibrosis, cirrhosis, or liver cancer. When first infected, a person can develop an illness which can be mild with few or no symptoms, or an illness that is serious, requiring hospitalization sometimes leading to liver failure (WHO, 2012). The Hepatitis B virus remains endemic despite an effective vaccine, necessitating ongoing efforts to control its spread. The study develops a six-compartment mathematical model incorporating Susceptible (S), Exposed (E), Infected (I), Treated (T), Vaccinated 9V), and Recovered (R) populations to describe Hepatitis B dynamics with vertical transmission. Mathematical modeling has been increasingly employed to understand and predict the spread and control of HBV in populations. There are safe and effective vaccines since 1982 for the prevention of hepatitis B infection. The vaccines work by activating the body to produce antibodies that protect against contacting the virus. Research is still in progress to find cure for chronic hepatitis B infection (WHO, 2023).

Recent studies have focused on incorporating key aspects such as vaccination, treatment, immune response, and vertical transmission in their models to provide insights into effective intervention strategies. Several authors have contributed to the development of HBV transmission models.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

Xu et al., (2023) constructed a dynamic model incorporating vaccination and vertical transmission, highlighting vaccination's critical role in reducing infection prevalence. Oluyo and Adejumo (2024) developed a vertical transmission model and nonlinear treatment model in transmission dynamics of Lassa fever using a mathematical study approach. Adepoju, et al., (2024) presented a mathematical assessment of HIV/AIDS dynamics epidemic with vertical transmission and treatment.

Despite these advances, vertical transmission remains a significant contributor to HBV persistence globally, making it imperative to develop and analyze models that explicitly capture this route alongside other modes of transmission and interventions. This study builds on these foundations to propose a mathematical model that focuses on the dynamics of HBV with vertical transmission, aiming to provide deeper insight into the infection's control and potential eradication strategies.

II. LITERATURE REVIEW

Wodajo, et al., (2023) worked on a model of HBV transmission dynamics with vertical transmission and assess combined intervention impacts. Their method of research design was a nonlinear mathematical compartmental model including vaccination, treatment, migration, and screening. The research was carried out in Ethiopia and the parameters were estimated from Ethiopian epidemiological data.

The result of the study was that combination of vaccine with treatment and screening effectively reduced HBV prevalence; migration influenced transmission dynamics; disease-free and endemic equilibriums stability was revealed. The knowledge gap that was filled encompass the inclusion of migration and dual-stage treatment and screening, which were lacking in prior models, providing a more comprehensive transmission dynamic understanding.

Yavuz, et al., (2024) also made research on the simulation of HBV transmission considering vertical transmission, migration, and fractional dynamics. Their method of research design was fractional-order differential equations model incorporating memory effects. The research was carried out in USA, and employed the data parameterized from US data.

The result of the study showed that the fractional order models better captured long-term infection trends; vertical transmission significant in persistence of HBV. The knowledge gap filled was an advanced fractional modeling addressing non-local and memory effects in HBV epidemiology.

Khan et al., (2016) worked on the transmission dynamic and optimal control of acute and chronic Hepatitis B. In their study, they presented the transmission dynamic of the acute and chronic hepatitis B epidemic problem and developed an optimal control strategy to control the spread of hepatitis B in a community. In order to do this, they firstly presented the model formulation and found the basic reproduction number R_0, and showed that if $R_0 \leq 1$, then the disease-free equilibrium is both locally as well as globally asymptotically stable. Then, they proceeded by proving that the model is locally and globally asymptotically stable, if $R_0 > 1$. To control the spread of this infection, they developed a control strategy by applying three control variables such as isolation of infected and non-infected individuals, treatment and vaccination to minimize the number of acute infected, chronically infected with hepatitis B individuals, and maximize the number of susceptible and recovered. Numerical simulation was later introduced to illustrate the feasibility of the control strategy.

Anley and Tadesse (2023) designed a model on vertical transmission of HBV and evaluated intervention strategies on infection dynamics. Their method of research design was Deterministic S-I-C-R compartmental model. The research was carried out in Ethiopia and the population parameters were a model compartment for susceptible, infected, carrier, and recovered.

The result of the research showed that the vertical transmission major contributor to HBV burden; suggested targeted interventions to reduce infections and mortality. The knowledge gap that was filled focused on specific vertical transmission quantification in a high-burden setting.

Ijalana et al., (2017) investigated an optimal control strategy for Hepatitis B virus epidemic in areas of high epidemicity. It was discovered that Hepatitis B virus infection remains endemic in some parts of northern Nigeria despite the availability of a potent vaccine against the infection. There is therefore the need for continuous efforts to eradicate the disease in order to forestall its spread to other parts of the country, more so that the disease is highly infectious, and this lead them to present an improved deterministic model to describe the spread of Hepatitis B virus infection in areas of high endemicity using vaccination of the susceptible and early detection cum effective treatment of acute Hepatitis B virus infected individuals as control measures. They formulated an optimal control problem subject to the model dynamics incorporating the two control measures with the goal of finding the optimal combination of the two control measures that will minimize the cost of implementing the control measures as well as reduce the incidences and prevalence of the disease. Using Pontryagin (2018) maximum principle, it was obtained that the optimal system can solve the system numerically. Their numerical simulations showed that starting treatment for acutely infected individuals early enough and improving the potency of the vaccines reduces the incidence and prevalence of Hepatitis B virus infection drastically within ten years.

Kuei and Gatoto (2021) examined the combined vaccination and treatment effects on vertical transmission of HBV. Their method of research design was a six-compartment differential equation model incorporating vaccination and treatment. The research was carried out in Kenya, and the population includes newborns and susceptible adults.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

The result of the research revealed that the combination approaches of vaccination and treatment effects showed reduction in vertical transmission rates. The knowledge gap filled was the integration of vaccination and treatment strategies in a single vertical transmission model.

Riches (2025) assess efficacy of WHO vertical transmission interventions. His method of research design was systematic review through meta-analysis of vertical transmission programs. The research was carried out in Africa, and the population consists of a large cohort of African pregnant women and newborns.

The result of the research showed that the prevention programs were effective but residual risk in high viral load cases. The knowledge gaps filled were the need for region-tailored strategies accounting for HBV genotype variability and viral load.

III. METHODS

➢ *Model Formulation*

The total human population at time t, denoted by $N(t)$, is subdivided into six mutually exclusive compartments of susceptible human, denoted by $(S(t))$, exposed human, denoted by $(E(t))$, infectious human, denoted by $(I(t))$, treated human, denoted by $(T(t))$, vaccinated human, denoted by $(V(t))$ and recovered human, denoted by $(R(t))$, respectively. Then, the total human population is obtained as:

$$N(t)=S(t)+E(t)+I(t)+T(t)+V(t)+R(t) \quad (2.1)$$

The susceptible component of the population increases due to the coming in of new born babies into the population with the assumption that infected mothers gave birth to the babies at a rate $\mu_1(1-P_2I)$. The population is further increased based on unsuccessful vaccination at a rate ηV and progression of recovered individuals due to loss of immunity at a rate $\sigma_3 R$ and fraction of new born babies that comes in contact with infectious human at a rate $\varepsilon\mu_1 I$. This component decreases due to the infection of individuals at a rate $(\beta_1 I+\beta_2 T)$. The population is further decreased by vaccination and natural death at a rate σ_1 and μ_2, respectively. Then, the rate of change of susceptible individuals is given by

$$\frac{dS}{dt}=\mu_1(1-P_2I)+\eta V+\sigma_3 R-\varepsilon\mu_1 I-(\beta_1 I+\beta_2 T)S-(\sigma_1+\mu_2)S \quad (2.2)$$

The population of the exposed individuals increases as a result of infection of individuals in the susceptible class at a rate $(\beta_1 I+\beta_2 T)S$ and further increased by the fraction of those born by infected mothers at a rate $\mu_1 P_2 I$. The class reduces due to the progression of exposed or latently infected individuals to active HB infection at a rate α. The population is further decreased by natural death at a rate μ. Then, the rate of change of exposed individuals is given by:

$$\frac{dE}{dt}=(\beta_1 I+\beta_2 T)S+\mu_1 P_2 I-(\alpha+\mu_2)E \quad (2.3)$$

The population of infectious individuals increases based on the progression of exposed or latently infected individuals to active HB infection at a rate α and further increased by the fraction of new born babies that comes in contact with infectious human at a rate $\varepsilon\mu_1 I$. The population reduces due to the probability that infected individuals clear the virus at a rate $p_1\lambda$ and further reduced by a fraction of those who failed to clear off the virus at a rate $(1-p_1)\lambda$. The population finally reduced due to death from natural causes at a rate μ_2. Then, the rate of change of infectious individuals is given by

$$\frac{dI}{dt}=\alpha E-\varepsilon\mu_1 I-(P_1\lambda+(1-P_1)\lambda+\mu_2)I \quad (2.4)$$

The population of treated infectious individuals increases based on the progression of fraction of those who failed to clear off the virus at a rate $(1-P_1)\lambda$. The population is reduced by the progression of treated individuals to the recovered class at a rate σ_4 and further reduced due to death from natural causes at a rate μ_2. Then, the rate of change of treated individuals is given by

$$\frac{dT}{dt}=(1-P_1)\lambda I-(\sigma_4+\mu_2)T \quad (2.5)$$

The vaccinated component increases as a result of successful vaccination of susceptible individuals at a rate σ_1 and decreases due to unsuccessful vaccination at a rate ηV and progression of vaccinated individuals to the recovered class at a rate σ_2. The population is further reduced by death from natural cause at a rate μ_2. Thus, the rate of change of vaccinated individuals is given by

$$\frac{dV}{dt}=\sigma_1 S-(\eta+\sigma_2+\mu_2)V \quad (2.6)$$

The population of recovered humans increases due to the progression of vaccinated individuals to the recovered class at a rate σ_2 and also reduces by the probability that an

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

infected individual clear the virus at a rate $p_1\lambda$. The population is further increased by the progression of treated individuals to the recovered class at a rate σ_4 .

The population is reduced by the progression of recovered individuals due to loss of immunity at a rate $\sigma_3 R$ further reduced by the natural death at a rate μ . Therefore, the rate of change of the population of recovered infectious human is given by

$$\frac{dR}{dt} = \sigma_2 V + P_1\lambda I + \sigma_4 T - (\sigma_3 + \mu_2)R \tag{2.7}$$

Thus, the mathematical model describing the transmission dynamics of human hepatitis B virus (HBV) in human population as written in equations (2.2 - 2.7) given as follows:

$$\left.\begin{aligned}
\frac{dS}{dt} &= \mu_1(1-P_2 I) + \eta V + \sigma_3 R - \varepsilon\mu_1 I - (\beta_1 I + \beta_2 T)S - (\sigma_1 + \mu_2)S \\
\frac{dE}{dt} &= (\beta_1 I + \beta_2 T)S + \mu_1 P_2 I - (\alpha + \mu_2)E \\
\frac{dI}{dt} &= \alpha E + \varepsilon\mu_1 I - (P_1\lambda + (1-P_1)\lambda + \mu_2)I \\
\frac{dT}{dt} &= (1-P_1)\lambda I - (\sigma_4 + \mu_2)T \\
\frac{dV}{dt} &= \sigma_1 S - (\eta + \sigma_2 + \mu_2)V \\
\frac{dR}{dt} &= \sigma_2 V + P_1\lambda I + \sigma_4 T - (\sigma_3 + \mu_2)R
\end{aligned}\right\} \tag{2.8}$$

Let $\lambda^* = (\beta_1 I + \beta_2 T)$

Table 1 Description of Variables

Variables	Description
$S(t)$	Population of susceptible humans at a given time.
$E(t)$	Population of exposed humans at a given time.
$I(t)$	Population of infectious humans at a given time.
$T(t)$	Population of treated humans at a given time.
$V(t)$	Population of vaccinated humans at a given time.
$R(t)$	Population of recovered humans at a given time.
$N(t)$	Total human population at a given time.

Table 2 Description of Parameters of the HBV Model

Parameter	Description
μ_1	The rate at which people give birth
μ_2	The rate at which people die
σ_1	Vaccination rate of susceptible individuals
η	Unsuccessful vaccination rate
β_1	Horizontal transmission rate control by the infected compartment
β_2	The rate of transmission for the treated population
α	The rate at which the expose population move to the infected population.
P_1	The probability that an infected individuals clear the virus

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

P_2	The probability that infected mothers give birth to infected babies
σ_2	Rate of moving from the vaccinated class to the recovered class
σ_3	The rate at which the recovered population move to the susceptible population due to loss of immunity.
σ_4	The rate at which the treated population move to the recovered population
$(1-P_1)$	The probability that an infected fail to clear the virus
λ λ^*	The rate at which infected population move to any other class. The force of infection

➢ *Mathematical Analysis of the Model*

- *Positivity and Boundedness of Solutions of the Model*

Since the mathematical model (2.8) governing the transmission dynamics of HBV consider human population, then it is important that all its state variables and associated parameters are non-negative for all time, t. Hence, the following result holds for all the state variables in the mathematical model (2.8).

✓ Theorem 2.1: let the initial data for the HBV mathematical model (2.8) be $S(0)>0; E(0)>0; I(0)>0; T_h(0)>0; V(0)>0$ and $R(0)>0$. Then, the solution $(S(t), E(t), I(t), T(t), V(t), R(t))$ of the HBV mathematical model (2.8), remains non-negative for all $t>0$.

- *Existence and Uniqueness of Solution*

✓ Theorem 2.2: There exists a unique solution for the transmission of Hepatitis B model (2.8).

Consider the system of equations below:

$$\left.\begin{aligned} x_1' &= f_1(x_1, x_2, \cdots, x_n, t), x_1(t_0) = (x_1)_0 \\ x_2' &= f_2(x_1, x_2, \cdots, x_n, t), x_2(t_0) = (x_2)_0 \\ &\vdots \\ x_n' &= f_n(x_1, x_2, \cdots, x_n, t), x_n(t_0) = (x_n)_0 \end{aligned}\right\} \quad (2.32)$$

The model can be written in a compact form as follows;

$$x' = f(t, x), x(t_0) = x_0 \quad (2.33)$$

✓ Theorem 2.3: Let f be continuous in a domain

$$D = \{(x,t) : |t-t_0| \le a, \|x-x_0\| \le b, x = (x_1, x_2, \ldots x_n), x_0 = [(x_1)_0, (x_2)_0, \cdots, (x_n)_0]\} \quad (2.34)$$

Suppose that $f(t,x)$ satisfies Lipschitz condition

$$\|f(x_1,t) - f(x_2,t)\| \le L\|x_1 - x_2\| \quad (2.35)$$

Then, system (2) has a unique solution in D, where the pairs $f(x_1,t)$ and $f(x_2,t)$ belongs to D and L (Lipschitz constant) is a positive constant.

It is of great importance to note that the Lipschitz condition is satisfied by the requirement that $\frac{\partial f_1}{\partial f_j}, i, j = 1,2,\cdots,n$ be continuous and bounded in D.

- *Existence of Disease-Free Equilibrium*

Disease free equilibrium point is a stable position where the entire population has no infection. Thus, at disease free equilibrium, the force of infection is set to zero (i.e. $\lambda^* = 0$) which means there is no infection such that $E = I = T = R = 0$. Let the disease-free equilibrium (DFE) of the HBV model be denoted by ε_0 .

Also, at critical points,

$$\frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dT}{dt} + \frac{dV}{dt} + \frac{dR}{dt} = 0 \quad (2.36)$$

Then, the equation becomes

$$\mu_1 + \eta V - (\sigma_1 + \mu_2)S = 0 \quad (2.37a)$$

$$\sigma_1 S - (\eta + \sigma_2 + \mu_2)V = 0 \quad (2.37b)$$

Now, from (2.37b),

$$\sigma_1 S - (\eta + \sigma_2 + \mu_2)V = 0$$

$$V = \frac{\sigma_1 S}{(\eta + \sigma_2 + \mu_2)} \quad (2.38)$$

From (2.37a), make S the subject of the formula, such that

$$\mu_1 + \eta V - (\sigma_1 + \mu_2)S = 0$$

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

$$S = \frac{\mu_1 + \eta V}{(\sigma_1 + \mu_2)} \tag{2.39}$$

Substitute (2.38) into (2.39), then

$$S = \frac{\mu_1 + \eta \dfrac{\sigma_1 S}{(\eta + \sigma_2 + \mu_2)}}{(\sigma_1 + \mu_2)}$$

$$S = \frac{\mu_1(\eta + \sigma_2 + \mu_2) + \eta\sigma_1 S}{(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2)}$$

$$(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2)S = \mu_1(\eta + \sigma_2 + \mu_2) + \eta\sigma_1 S$$

$$(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2)S - \eta\sigma_1 S = \mu_1(\eta + \sigma_2 + \mu_2)$$

$$\{(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2) - \eta\sigma_1\}S = \mu_1(\eta + \sigma_2 + \mu_2)$$

$$S = \frac{\mu_1(\eta + \sigma_2 + \mu_2)}{\{(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2) - \eta\sigma_1\}} \tag{2.40}$$

Substitute (2.40) into (2.38), then

$$V = \frac{\sigma_1 S}{(\eta + \sigma_2 + \mu_2)}$$

$$V = \frac{\sigma_1 \dfrac{\mu_1(\eta + \sigma_2 + \mu_2)}{\{(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2) - \eta\sigma_1\}}}{(\eta + \sigma_2 + \mu_2)}$$

$$V = \frac{\sigma_1\mu_1}{\{(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2) - \eta\sigma_1\}} \tag{2.41}$$

Therefore, the disease-free equilibrium is obtained as

$$\varepsilon_0 = (S_0, 0, 0, 0, V_0, 0) \tag{2.42}$$

where;

$$S_0 = \frac{\mu_1(\eta + \sigma_2 + \mu_2)}{\{(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2) - \eta\sigma_1\}}$$

$$V_0 = \frac{\sigma_1\mu_1}{\{(\sigma_1 + \mu_2)(\eta + \sigma_2 + \mu_2) - \eta\sigma_1\}}$$

- *Local Stability Analysis of Disease-Free Equilibrium of the Model*

✓ Theorem 2.4: The disease-free equilibrium of the Hepatitis B virus model (2.8) is locally asymptotically stable (LAS) whenever the associated threshold parameter (R_0) is less than one.

✓ *Proof:*

To study the stability of the disease-free equilibrium point (2.36), then, the following Jacobian matrix J of the system equation (2.8) is calculated.

Now, considering the stability of the infection-free equilibrium at critical point for the model equation (3.8), then: Let $f = (f_1, f_2, \cdots f_n)$, such that the model equation (2.8) becomes:

$$\left.\begin{aligned}
f_1 &= \frac{dS}{dt} = \mu_1(1 - P_2 I) + \eta V + \sigma_3 R - \varepsilon\mu_1 I - (\beta_1 I + \beta_2 T)S - (\sigma_1 + \mu_2)S \\
f_2 &= \frac{dE}{dt} = (\beta_1 I + \beta_2 T)S + \mu_1 P_2 I - (\alpha + \mu_2)E \\
f_3 &= \frac{dI}{dt} = \alpha E + \varepsilon\mu_1 I - (P_1\lambda + (1 - P_1)\lambda + \mu_2)I \\
f_4 &= \frac{dT}{dt} = (1 - P_1)\lambda I - (\sigma_4 + \mu_2)T \\
f_5 &= \frac{dV}{dt} = \sigma_1 S - (\eta + \sigma_2 + \mu_2)V \\
f_6 &= \frac{dR}{dt} = \sigma_2 V + P_1\lambda I + \sigma_4 T - (\sigma_3 + \mu_2)R
\end{aligned}\right\} \tag{2.43}$$

Now, defining the Jacobian matrix of the system (2.43)

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

$$J = \begin{pmatrix} \frac{\partial f_1}{\partial S} & \frac{\partial f_1}{\partial E} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial V} & \frac{\partial f_1}{\partial R} \\ \frac{\partial f_2}{\partial S} & \frac{\partial f_2}{\partial E} & \frac{\partial f_2}{\partial I} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial R} \\ \frac{\partial f_3}{\partial S} & \frac{\partial f_3}{\partial E} & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial T} & \frac{\partial f_3}{\partial R} \\ \frac{\partial f_4}{\partial S} & \frac{\partial f_4}{\partial E} & \frac{\partial f_4}{\partial I} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial T} & \frac{\partial f_4}{\partial R} \\ \frac{\partial f_5}{\partial S} & \frac{\partial f_5}{\partial E} & \frac{\partial f_5}{\partial I} & \frac{\partial f_5}{\partial T} & \frac{\partial f_5}{\partial T} & \frac{\partial f_5}{\partial R} \\ \frac{\partial f_6}{\partial S} & \frac{\partial f_6}{\partial E} & \frac{\partial f_6}{\partial I} & \frac{\partial f_6}{\partial T} & \frac{\partial f_6}{\partial T} & \frac{\partial f_6}{\partial R} \end{pmatrix} \tag{2.44}$$

Then, the Jacobian of (2.43) is obtained using (2.44)

$$J = \begin{pmatrix} -(\beta_1 I + \beta_2 T + \sigma_1 + \mu_2) & 0 & -(\mu_1 P_2 + \varepsilon\mu_1 + \beta_1 S) & -\beta_2 S & \eta & \sigma_3 \\ \beta_1 I + \beta_2 T & -k_2 & \mu_1 P_2 + \beta_1 S & \beta_2 S & 0 & 0 \\ 0 & \alpha & \varepsilon\mu_1 - k_3 & 0 & 0 & 0 \\ 0 & 0 & (1-P_1)\lambda & -k_4 & 0 & 0 \\ \sigma_1 & 0 & 0 & 0 & -k_5 & 0 \\ 0 & 0 & P_1\lambda & \sigma_4 & \sigma_2 & -k_6 \end{pmatrix} \tag{2.45}$$

Then, evaluating the Jacobian matrix at disease-free gives:

$$J|(\varepsilon_0) = \begin{pmatrix} -(\sigma_1 + \mu_2) & 0 & -(\mu_1 P_2 + \varepsilon\mu_1 + \beta_1 S_0) & -\beta_2 S_0 & \eta & \sigma_3 \\ 0 & -k_2 & \mu_1 P_2 + \beta_1 S_0 & \beta_2 S_0 & 0 & 0 \\ 0 & \alpha & \varepsilon\mu_1 - k_3 & 0 & 0 & 0 \\ 0 & 0 & (1-P_1)\lambda & -k_4 & 0 & 0 \\ \sigma_1 & 0 & 0 & 0 & -k_5 & 0 \\ 0 & 0 & P_1\lambda & \sigma_4 & \sigma_2 & -k_6 \end{pmatrix} \tag{2.46}$$

To avoid confusion,

Let $k_7 = (\mu_1 P_2 + \varepsilon\mu_1 + \beta_1 S_0)$, $k_8 = \mu_1 P_2 + \beta_1 S_0$, $k_9 = (\varepsilon\mu_1 - k_3)$, and $(1-P_1)\lambda = (1-P_1)\Lambda$, such that the λ in the model will become Λ, thereby making it different from the eigenvalue. Then, using the characteristics equation $\left|J_{(\varepsilon_0)} - \lambda I\right| = 0$, equation (2.48) becomes:

$$\begin{vmatrix} -k_1 - \lambda & 0 & -(\mu_1 P_2 + \varepsilon\mu_1 + \beta_1 S_0) & -\beta_2 S_0 & \eta & \sigma_3 \\ 0 & -k_2 - \lambda & \mu_1 P_2 + \beta_1 S_0 & \beta_2 S_0 & 0 & 0 \\ 0 & \alpha & (\varepsilon\mu_1 - k_3) - \lambda & 0 & 0 & 0 \\ 0 & 0 & (1-P_1)\lambda & -k_4 - \lambda & 0 & 0 \\ \sigma_1 & 0 & 0 & 0 & -k_5 - \lambda & 0 \\ 0 & 0 & P_1\lambda & \sigma_4 & \sigma_2 & -k_6 - \lambda \end{vmatrix} = 0 \tag{2.47}$$

Reducing the matrix (2.47) gives the following results

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

$$\left|\begin{array}{l}(-k_1-\lambda)\begin{pmatrix}-k_2-\lambda & k_8 & \beta_2 S_0 & 0 & 0\\ \alpha & k_9-\lambda & 0 & 0 & 0\\ 0 & (1-P_1)\Lambda & -k_4-\lambda & 0 & 0\\ 0 & 0 & 0 & -k_5-\lambda & 0\\ 0 & P_1\Lambda & \sigma_4 & \sigma_2 & -k_6-\lambda\end{pmatrix}-k_7\begin{pmatrix}0 & -k_2-\lambda & \beta_2 S_0 & 0 & 0\\ 0 & \alpha & 0 & 0 & 0\\ 0 & 0 & -k_4-\lambda & 0 & 0\\ \sigma_1 & 0 & 0 & -k_5-\lambda & 0\\ 0 & 0 & \sigma_4 & \sigma_2 & -k_6-\lambda\end{pmatrix}\\ +\beta_2 S_0\begin{pmatrix}0 & -k_2-\lambda & k_8 & 0 & 0\\ 0 & \alpha & k_9-\lambda & 0 & 0\\ 0 & 0 & (1-P_1)\Lambda & 0 & 0\\ \sigma_1 & 0 & 0 & -k_5-\lambda & 0\\ 0 & 0 & P_1\Lambda & \sigma_2 & -k_6-\lambda\end{pmatrix}+\eta\begin{pmatrix}0 & -k_2-\lambda & k_8 & \beta_2 S_0 & 0\\ 0 & \alpha & k_9-\lambda & 0 & 0\\ 0 & 0 & (1-P_1)\Lambda & -k_4-\lambda & 0\\ \sigma_1 & 0 & 0 & 0 & 0\\ 0 & 0 & P_1\Lambda & \sigma_4 & -k_6-\lambda\end{pmatrix}\\ -\sigma_3\begin{pmatrix}0 & -k_2-\lambda & k_8 & \beta_2 S_0 & 0\\ 0 & \alpha & k_9-\lambda & 0 & 0\\ 0 & 0 & (1-P_1)\Lambda & -k_4-\lambda & 0\\ \sigma_1 & 0 & 0 & 0 & -k_5-\lambda\\ 0 & 0 & P_1\Lambda & \sigma_4 & \sigma_2\end{pmatrix}\end{array}\right|=0$$

This gives:

$$\left|\begin{array}{l}(-k_1-\lambda)\left\{\begin{array}{l}(-k_2-\lambda)\begin{pmatrix}k_9-\lambda & 0 & 0 & 0\\ (1-P_1)\Lambda & -k_4-\lambda & 0 & 0\\ 0 & 0 & -k_5-\lambda & 0\\ P_1\Lambda & \sigma_4 & \sigma_2 & -k_6-\lambda\end{pmatrix}-k_8\begin{pmatrix}\alpha & 0 & 0 & 0\\ 0 & -k_4-\lambda & 0 & 0\\ 0 & 0 & -k_5-\lambda & 0\\ 0 & \sigma_4 & \sigma_2 & -k_6-\lambda\end{pmatrix}\\ +\beta_2 S_0\begin{pmatrix}\alpha & k_9-\lambda & 0 & 0\\ 0 & (1-P_1)\Lambda & 0 & 0\\ 0 & 0 & -k_5-\lambda & 0\\ 0 & P_1\Lambda & \sigma_2 & -k_6-\lambda\end{pmatrix}\end{array}\right.\\ -k_7\left\{-(-k_2-\lambda)\begin{pmatrix}0 & 0 & 0 & 0\\ 0 & -k_4-\lambda & 0 & 0\\ \sigma_1 & 0 & -k_5-\lambda & 0\\ 0 & \sigma_4 & \sigma_2 & -k_6-\lambda\end{pmatrix}+\beta_2 S_0\begin{pmatrix}0 & \alpha & 0 & 0\\ 0 & 0 & 0 & 0\\ \sigma_1 & 0 & -k_5-\lambda & 0\\ 0 & 0 & \sigma_2 & -k_6-\lambda\end{pmatrix}\right\}\\ +\beta_2 S_0\left\{-(-k_2-\lambda)\begin{pmatrix}0 & k_9-\lambda & 0 & 0\\ 0 & (1-P_1)\Lambda & 0 & 0\\ \sigma_1 & 0 & -k_5-\lambda & 0\\ 0 & P_1\Lambda & \sigma_2 & -k_6-\lambda\end{pmatrix}+k_8\begin{pmatrix}0 & \alpha & 0 & 0\\ 0 & 0 & 0 & 0\\ \sigma_1 & 0 & -k_5-\lambda & 0\\ 0 & 0 & \sigma_2 & -k_6-\lambda\end{pmatrix}\right\}\end{array}\right|$$

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

$$+\eta\left\{\begin{array}{l}-(-k_2-\lambda)\begin{pmatrix}0 & k_9-\lambda & 0 & 0\\ 0 & (1-P_1)\Lambda & -k_4-\lambda & 0\\ \sigma_1 & 0 & 0 & 0\\ 0 & P_1\Lambda & \sigma_4 & -k_6-\lambda\end{pmatrix}+k_8\begin{pmatrix}0 & \alpha & 0 & 0\\ 0 & 0 & -k_4-\lambda & 0\\ \sigma_1 & 0 & 0 & 0\\ 0 & 0 & \sigma_4 & -k_6-\lambda\end{pmatrix}\\ -\beta_2 S_0\begin{pmatrix}0 & \alpha & k_9-\lambda & 0\\ 0 & 0 & (1-P_1)\Lambda & 0\\ \sigma_1 & 0 & 0 & 0\\ 0 & 0 & P_1\Lambda & -k_6-\lambda\end{pmatrix}\end{array}\right\}$$

$$-\sigma_3\left\{\begin{array}{l}-(-k_2-\lambda)\begin{pmatrix}0 & k_9-\lambda & 0 & 0\\ 0 & (1-P_1)\Lambda & -k_4-\lambda & 0\\ \sigma_1 & 0 & 0 & -k_5-\lambda\\ 0 & P_1\Lambda & \sigma_4 & \sigma_2\end{pmatrix}+k_8\begin{pmatrix}0 & \alpha & 0 & 0\\ 0 & 0 & -k_4-\lambda & 0\\ \sigma_1 & 0 & 0 & -k_5-\lambda\\ 0 & 0 & \sigma_4 & \sigma_2\end{pmatrix}\\ -\beta_2 S_0\begin{pmatrix}0 & \alpha & k_9-\lambda & 0\\ 0 & 0 & (1-P_1)\Lambda & 0\\ \sigma_1 & 0 & 0 & -k_5-\lambda\\ 0 & 0 & P_1\Lambda & \sigma_2\end{pmatrix}\end{array}\right\}=0$$

Further simplification gives the following result:

$$\left|(-k_1-\lambda)\left\{\begin{array}{l}(-k_2-\lambda)(k_9-\lambda)\begin{pmatrix}-k_4-\lambda & 0 & 0\\ 0 & -k_5-\lambda & 0\\ \sigma_4 & \sigma_2 & -k_6-\lambda\end{pmatrix}-\alpha k_8\begin{pmatrix}-k_4-\lambda & 0 & 0\\ 0 & -k_5-\lambda & 0\\ \sigma_4 & \sigma_2 & -k_6-\lambda\end{pmatrix}\\ +\alpha\beta_2 S_0\begin{pmatrix}(1-P_1)\Lambda & 0 & 0\\ 0 & -k_5-\lambda & 0\\ P_1\Lambda & \sigma_2 & -k_6-\lambda\end{pmatrix}\end{array}\right\}\right.$$

$$+\beta_2 S_0\left\{(-k_2-\lambda)k_9-\lambda\begin{pmatrix}0 & 0 & 0\\ \sigma_1 & -k_5-\lambda & 0\\ 0 & \sigma_2 & -k_6-\lambda\end{pmatrix}-\alpha k_8(0)\right\}$$

$$+\eta\left\{(k_9-\lambda)(-k_2-\lambda)\begin{pmatrix}0 & -k_4-\lambda & 0\\ \sigma_1 & 0 & 0\\ 0 & \sigma_4 & -k_6-\lambda\end{pmatrix}+\beta_2 S_0\alpha\begin{pmatrix}0 & (1-P_1)\Lambda & 0\\ \sigma_1 & 0 & 0\\ 0 & P_1\Lambda & -k_6-\lambda\end{pmatrix}\right\}$$

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

$$-\sigma_3\left\{\begin{array}{l}(k_9-\lambda)(-k_2-\lambda)\begin{pmatrix}0 & -k_4-\lambda & 0\\ \sigma_1 & 0 & -k_5-\lambda\\ 0 & \sigma_4 & \sigma_2\end{pmatrix}-\alpha k_8\begin{pmatrix}0 & -k_4-\lambda & 0\\ \sigma_1 & 0 & -k_5-\lambda\\ 0 & \sigma_4 & \sigma_2\end{pmatrix}\\ -\beta_2 S_0\left|-\alpha\begin{pmatrix}0 & (1-P_1)\Lambda & 0\\ \sigma_1 & 0 & -k_5-\lambda\\ 0 & P_1\Lambda & \sigma_2\end{pmatrix}+(k_9-\lambda)\begin{pmatrix}0 & 0 & 0\\ \sigma_1 & 0 & -k_5-\lambda\\ 0 & 0 & \sigma_2\end{pmatrix}\right|\end{array}\right\}=0 \quad (2.48)$$

Then,

$$\left|\begin{array}{l}(-k_1-\lambda)\left\{\begin{array}{l}(-k_2-\lambda)(-k_4-\lambda)(-k_5-\lambda)(-k_6-\lambda)(k_9-\lambda)-\alpha k_8(-k_4-\lambda)(-k_5-\lambda)(-k_6-\lambda)\\ +\alpha\beta_2 S_0(1-P_1)\Lambda(-k_5-\lambda)(-k_6-\lambda)\end{array}\right\}\\ -\eta\{\sigma_1(k_9-\lambda)(-k_2-\lambda)(-k_4-\lambda)(-k_6-\lambda)-\sigma_1\beta_2 S_0\alpha(1-P_1)\Lambda(-k_6-\lambda)\}-\\ \sigma_3\{-\sigma_1\sigma_2(k_9-\lambda)(-k_2-\lambda)(-k_4-\lambda)+\sigma_1\sigma_2\alpha k_8(-k_4-\lambda)-\beta_2 S_0\alpha(1-P_1)\Lambda\sigma_1\sigma_2\}\end{array}\right|=0$$

Then, the following result is obtained

$$\lambda^6+A_1\lambda^5+A_2\lambda^4+A_3\lambda^3+A_4\lambda^2+A_5\lambda+A_6=0 \quad (2.49)$$

where;

$$A_1=a_1-a_3>0,$$

$$A_2=(a_2-(a_4+a_1a_3)-\eta\sigma_1)>0,$$

$$A_3=(k_1k_5k_6-(a_5+a_1a_4+a_2a_3)-\sigma_1[\eta(k_6-b_1)+\sigma_2\sigma_3])>0,$$

$$A_4=\sigma_1[\eta(k_6b_1+b_2)+C_1\sigma_3]-(a_1(1+k_1k_5k_6)+a_1a_4)>0,$$

$$A_5=\sigma_1[\eta(k_6b_2+b_3)+C_2\sigma_3]-(a_2a_5+a_1k_1k_5k_6)>0,$$

$$A_6=-(k_1k_5k_6a_5+\eta\sigma_1k_6b_3+\sigma_1\sigma_3C_3)>0,$$

And $A=\varepsilon\mu_1-k_3,$

$$a_1=-(k_1+k_2+k_6),$$

$$a_2=-(k_1(k_5+k_6)+k_5k_6),$$

$$a_3=A-(k_2+k_4),$$

$$a_4=A(k_2+k_4)-k_2k_4+\alpha k_8,$$

$$a_5=(Ak_2k_4\alpha(k_4k_8+(1-p_1)\Lambda\beta_2S_0)),$$

$$b_1=(A-(k_2+k_4)),$$

$$b_2=(A(k_2+k_4)-k_2k_4+\alpha k_8),$$

$$b_3=(Ak_2k_4+\alpha(k_4k_8+(1-p_1)\Lambda\beta_2S_0)),$$

$$C_1=\sigma_2(A-(k_2+k_4)),$$

$$C_2=(\sigma_2A(k_2+k_4)-\sigma_2k_2k_4+\alpha\sigma_2k_8),$$

$$C_3=(\sigma_2Ak_2k_4+\alpha\sigma_2(k_4k_8+(1-p_1)\Lambda\beta_2S_0)).$$

Therefore, the polynomial equation (2.49) is the solution of the Jacobian matrix. Then, following Descarte's rule of signs, since all the coefficient of the polynomial are positive, then it can be concluded that all the eigenvalues of the Jacobian matrix are negative, real and distinct.

Hence, the disease-free equilibrium of the HBV model (2.8) is locally asymptotically stable.

- *Global Stability of Endemic Equilibrium*

- ✓ Theorem 3.6: If $R_0>1$, then the endemic equilibrium ε_0^{**} of the HBV model (2.8) is globally asymptotically stable.

- ✓ Proof:

The global asymptotic stability of the endemic equilibrium can be proved using the Lyapunov functions (Cai and Li, 2000) defined as:

$$G=(S^{**},\ E^{**},\ I^{**},T^{**},V^{**},\ R^{**})$$

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

$$
\begin{aligned}
&= \left(S - S^{**} - S^{**} \log \frac{S}{S^{**}} \right) + \left(E - E^{**} - E^{**} \log \frac{E}{E^{**}} \right) + \left(I - I^{**} - I^{**} \log \frac{I}{I^{**}} \right) \\
&+ \left(T - T^{**} - T^{**} \log \frac{T}{T^{**}} \right) + \left(V - V^{**} - V^{**} \log \frac{V}{V^{**}} \right) + \left(R - R^{**} - R^{**} \log \frac{R}{R^{**}} \right)
\end{aligned} \tag{2.50}
$$

Then, by direct calculation, the derivation of the Lyapunov solution for the HBV model is obtained as:

$$
\begin{aligned}
\frac{dG}{dt} &= \frac{dS}{dt} - \left(\frac{S^{**}}{S} \right) \frac{dS}{dt} + \frac{dE}{dt} - \left(\frac{E^{**}}{E} \right) \frac{dE}{dt} + \frac{dI}{dt} - \left(\frac{I^{**}}{I} \right) \frac{dI}{dt} + \frac{dT}{dt} - \left(\frac{T^{**}}{T} \right) \frac{dT}{dt} \\
&+ \frac{dV}{dt} - \left(\frac{V^{**}}{V} \right) \frac{dV}{dt} + \frac{dR}{dt} - \left(\frac{R^{**}}{R} \right) \frac{dR}{dt}
\end{aligned} \tag{2.51}
$$

Substitute the right-hand side of system (2.8) into (2.51). Then (2.51) becomes:

$$
\begin{aligned}
\frac{dG}{dt} &= \mu_1 (1 - P_2 I) + \eta V + \sigma_3 R - \varepsilon \mu_1 I - (\beta_1 I + \beta_2 T) S - (\sigma_1 + \mu_2) S \\
&+ (\beta_1 I + \beta_2 T) S + \mu_1 P_2 I - (\alpha + \mu_2) E + \alpha E + \varepsilon \mu_1 I - (P_1 \lambda + (1 - P_1) \lambda + \mu_2) I \\
&+ (1 - P_1) \lambda I - (\sigma_4 + \mu_2) T + \sigma_1 S - (\eta + \sigma_2 + \mu_2) V + \sigma_2 V + P_1 \lambda I + \sigma_4 T - (\sigma_3 + \mu_2) R \\
&- \left(\frac{S^{**}}{S} \right) \{ \mu_1 (1 - P_2 I) + \eta V + \sigma_3 R - \varepsilon \mu_1 I - (\beta_1 I + \beta_2 T) S - (\sigma_1 + \mu_2) S \} \\
&- \left(\frac{E^{**}}{E} \right) \{ (\beta_1 I + \beta_2 T) S + \mu_1 P_2 I - (\alpha + \mu_2) E \} \\
&- \left(\frac{I^{**}}{I} \right) \{ \varepsilon \mu_1 I - (P_1 \lambda + (1 - P_1) \lambda + \mu_2) I \} \\
&- \left(\frac{T^{**}}{T} \right) \{ (1 - P_1) \lambda I - (\sigma_4 + \mu_2) T \} \\
&- \left(\frac{V^{**}}{V} \right) \{ \sigma_1 S - (\eta + \sigma_2 + \mu_2) V \} \\
&- \left(\frac{R^{**}}{R} \right) \{ \sigma_2 V + P_1 \lambda I + \sigma_4 T - (\sigma_3 + \mu_2) R \}
\end{aligned} \tag{2.52}
$$

Simplifying (2.52) gives the following result:

$$
\begin{aligned}
\frac{dG}{dt} &= \mu_1 - (\mu_1 (1 - P_2 I) - \eta V - \sigma_3 R + \varepsilon \mu_1 I) \left(\frac{S^{**}}{S} \right) + ((\beta_1 I + \beta_2 T) + \mu_2 + \sigma_1) S^{**} - \mu_2 S \\
&- ((\beta_1 I + \beta_2 T) S + \mu_1 P_2 I) \left(\frac{E^{**}}{E} \right) + (\alpha + \mu_2) E^{**} + (\alpha - (\alpha + \mu_2)) E - \alpha E \left(\frac{I^{**}}{I} \right) \\
&+ ((\alpha + \mu_2) - \varepsilon \mu_1) I^{**} - (P_1 \lambda + (1 - P_1) \lambda + \mu_2) I + P_1 \lambda \left(\frac{I^{**}}{I} \right) + (\sigma_4 + \mu_2) T^{**} \\
&+ (\sigma_4 - (\sigma_4 + \mu_2)) T - \sigma_1 S \left(\frac{V^{**}}{V} \right) + (\eta + \sigma_2 + \mu_2) V^{**} + (\eta + \sigma_2 - (\eta + \sigma_2 + \mu_2)) V \\
&- (\sigma_2 V + P_1 \lambda I + \sigma_4 T) \left(\frac{R^{**}}{R} \right) + (\sigma_3 + \mu_2) R^{**} + (\sigma_3 - (\sigma_3 + \mu_2)) R
\end{aligned} \tag{2.53}
$$

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

Re-arranging the postive and negative terms in (3.112) using the equation below gives

$$\frac{dG}{dt} = M - N \tag{2.54}$$

Then,

$$\begin{aligned} M = {} & \mu_1 + \left(\left(\beta_1 I + \beta_2 T\right) + \mu_2 + \sigma_1\right)S^{**} + \left(\alpha + \mu_2\right)E^{**} + \left(\alpha - \left(\alpha + \mu_2\right)\right)E \\ & + \left(\left(\alpha + \mu_2\right) - \varepsilon\mu_1\right)I^{**} + P_1\lambda\left(\frac{I^{**}}{I}\right) + \left(\sigma_4 + \mu_2\right)T^{**} + \left(\sigma_4 - \left(\sigma_4 + \mu_2\right)\right)T \\ & + \left(\eta + \sigma_2 + \mu_2\right)V^{**} + \left(\eta + \sigma_2 - \left(\eta + \sigma_2 + \mu_2\right)\right)V + \left(\sigma_3 + \mu_2\right)R^{**} \\ & + \left(\sigma_3 - \left(\sigma_3 + \mu_2\right)\right)R \end{aligned}$$

$$\begin{aligned} N = {} & -\left(\mu_1\left(1 - P_2 I\right) - \eta V - \sigma_3 R + \varepsilon\mu_1 I\right)\left(\frac{S^{**}}{S}\right) - \mu_2 S - \left(\left(\beta_1 I + \beta_2 T\right)S + \mu_1 P_2 I\right)\left(\frac{E^{**}}{E}\right) \\ & - \alpha E\left(\frac{I^{**}}{I}\right) - \left(P_1\lambda + \left(1 - P_1\right)\lambda + \mu_2\right)I - \sigma_1 S\left(\frac{V^{**}}{V}\right) - \left(\sigma_2 V + P_1\lambda I + \sigma_4 T\right)\left(\frac{R^{**}}{R}\right) \end{aligned}$$

Therefore, if $M < N$, then we obtain $\frac{dG}{dt} = 0$, noting that $\frac{dG}{dt} = 0$ if and only if $S = S^{**},\ E = E^{**},\ I = I^{**}, T = T^{**}, V = V^{**},\ R = R^{**}$. Then, the largest compact invariant set $\left\{\left(S^{**},\ E^{**},\ I^{**}, T^{**}, V^{**},\ R^{**}\right) \in D,\ \frac{dG}{dt} = 0\right\}$ is the singleton $\left\{\varepsilon_0^{**}\right\}$ where ε_0^{**} is the endemic equilibrium. Hence, by La' Salles invariance principle (La' Salles, 1976), it applies that the endemic equilibrium point is globally asymptotically stable in the region D if $M < N$.

➢ *Sensitivity Analysis*

Here, the changes occurring in HBV infection transmission dynamics by computing the sensitivity indices of the parameters relative to the basic reproduction number. The normalized forward sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter (Chitnis *et al.,* (2008)). The normalized forward sensitivity index of a variable X that depends differentially on a parameter P is defined as:

$$\xi_P^X = \frac{\partial X}{\partial P} \times \frac{P}{X} \tag{2.55}$$

Using (2.50), the sensitivity indices of the parameters associated with basic reproduction number is calculated and the result is presented in Table 3 below:

Table 3 Sensitivity Indices of Basic Reproduction Number Relative to its Parameters (Otoo et al., (2021))

Parameters	Values	Sensitivity Indices
α	0.055	0.1772
μ_1	0.0196	+1.0044
μ_2	0.0096	-0.5751
η	0.001	+0.0616
β_1	0.04	+0.1472
β_2	0.002	+0.3948
λ	0.450	-0.5874
ε	0.1	-0.1305
P_1	0.65	-0.7332
P_2	0.0025	+0.0001

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

σ_1	0.25	-0.9601
σ_2	0.005	-0.0211
σ_4	0.0025	-0.0816

IV. RESULTS

A new mathematical model for the transmission dynamics of Hepatitis B virus governed by a system of ordinary differential equations was formulated and analyzed. The model subdivided the total human population into six mutually exclusive compartments of susceptible humans, exposed humans, infected humans, treated, vaccinated and recovered humans, respectively. The mathematical model was shown to be mathematically and epidemiologically meaningful through the positivity and boundedness of solution of the model. The equilibriums points of the model were obtained and the local stability of the disease-free equilibrium was investigated. The HBV reproduction number was calculated using the next generation matrix method. The endemic equilibrium point of the model was shown to be globally asymptotically stable. Using sensitivity analysis, the effect of the parameters of the model relative to the basic reproduction number was calculated.

It can be seen from the table 3, the indices with positive sign shows that the value of the basic reproduction number will increase when the corresponding parameters are increased and indices with negative signs indicates that, the value of the basic reproduction number will decrease with increase in the corresponding parameters. Therefore, it is clear from the Table 3 above, that the value of the basic reproduction number will decrease with increase in the values of the parameters, since the sensitivity indices of these parameters are negative.

To validate theoretical findings of model system of (2.8), the numerical simulations were performed using MATLAB so as to gain more insights in the equilibrium dynamics and the most sensitive parameters associated to the basic reproduction number as well as on time profiles.

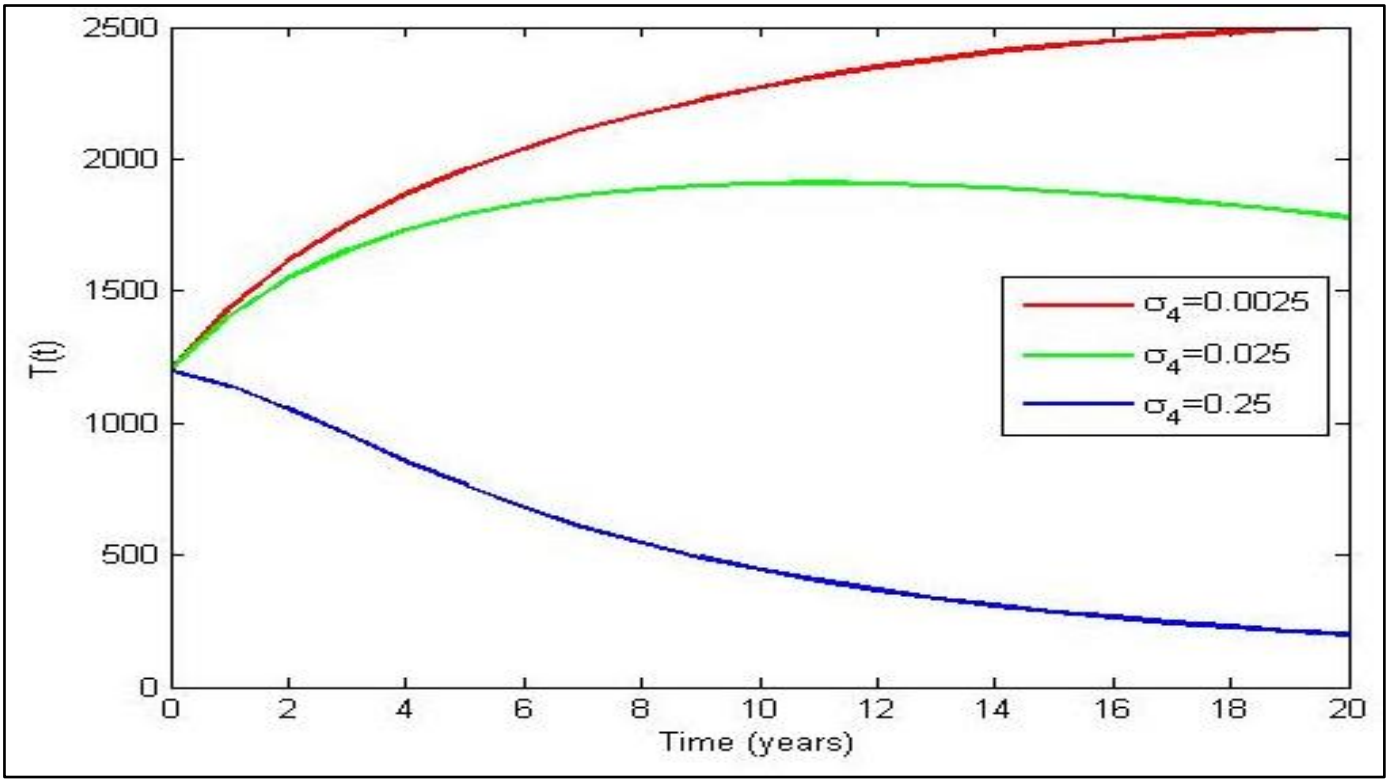

Fig 1 Effect of the Recovery Rate on the Population of Treated Human

Fig. 1 shows the rate of movement from treated class to recovered class. This can only happen when the treatment is sufficient enough to suppress or reduce the viral load, at σ_4=0·0025, the treatment melted is inactive and there is a progression in the treated population. This could be due to possibly, ignorance in the side of the treated patients, wrong diagnosis or self-medication. At σ_4=0.025, the treatment shows a partial effect on the treated class and was not adequately enough, but over a long period of time, there could be significant impact on the treatment class which tends to suppress the viral load. At σ_4=0.25, it was observed that the treatment is adequately enough to remove the patient from the treated class to the recovered class. This is achievable when there is proper diagnosis, understanding of the mechanism of the disease, adequate information and qualified personnel to manage the diseases.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

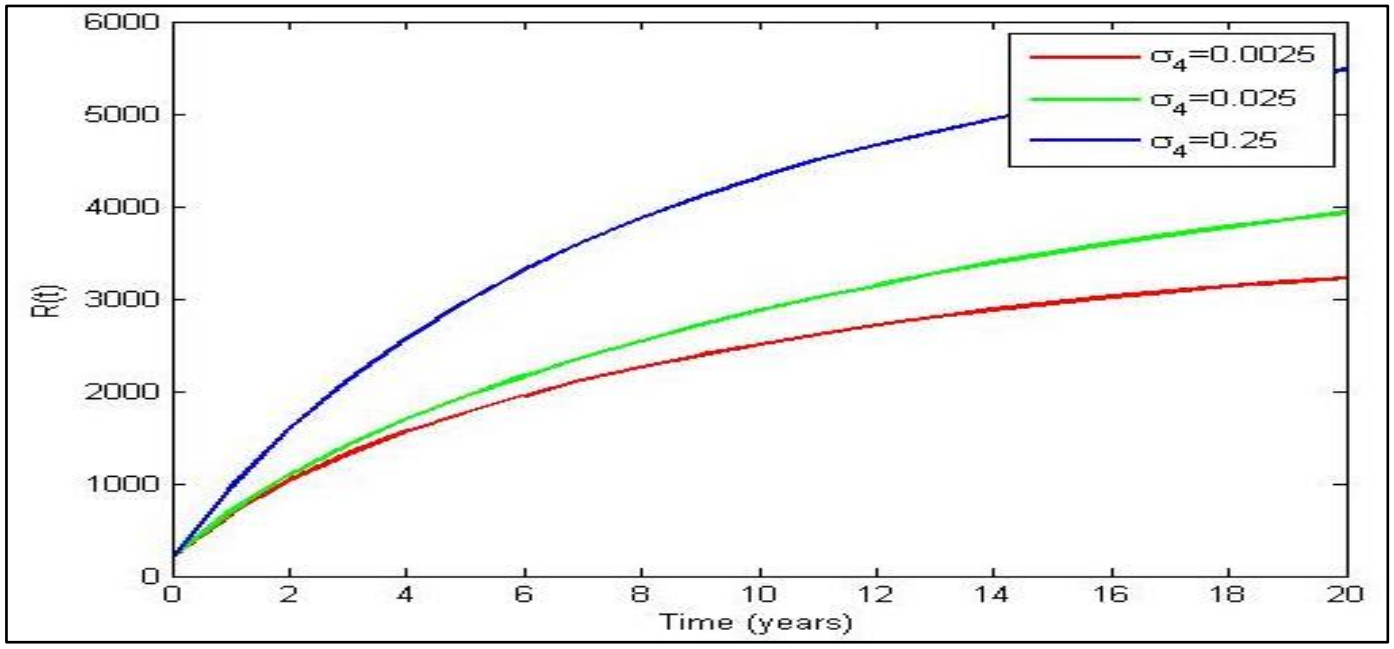

Fig 2 Effect of σ_4 on the population of recovered Human

Fig 2 represents the effect of σ4 on the population of the recovered class. It was noted that, at σ_4= 0.25, there is an outright increase in the recovered population, as many treated persons join the recovered population. This was as a result of the adequate treatment, proper diagnosis, and understanding of the mechanism of the disease. At σ_4 = 0.025, it is noticeable that there is a slight drop in the number of those that are recovered; and there is a little decrease in the rate at which people recover.

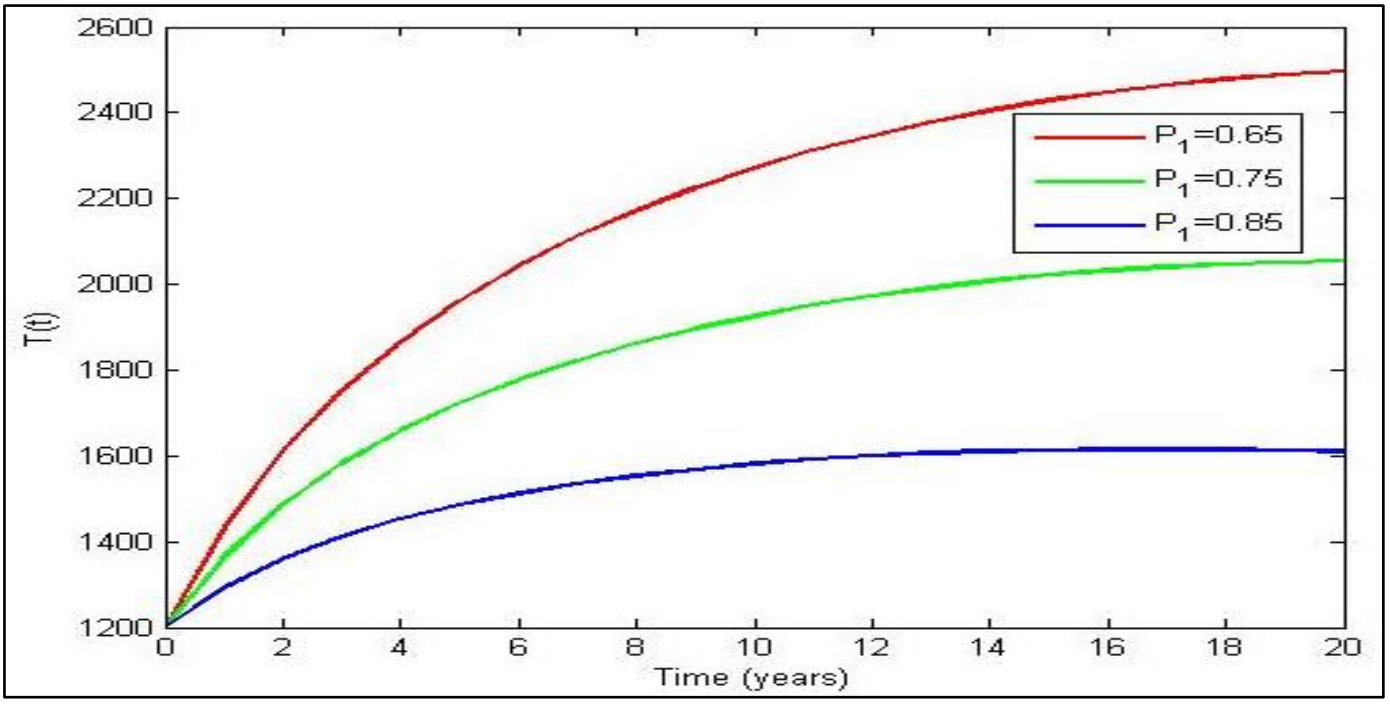

Fig 3 Effect of P_1 on the Population of Treated Human

Fig. 3 gives the physical representation to the parameter P_1, which is the probability that an infected individual clear the virus, possibly after treatment or as a result of immunity. At P_1 = 0.65, this shows the percentage of people that cleared the virus and moved to the recovered class as indicated on the curve. At P_1 = 0.75, there is a slight increment on the people that cleared the virus as indicated by the curve, at this point, there is a change in the pattern of treatment, people's orientation and awareness to the virus. At P_1 = 0.85, more people move from the treated class to the recovered class, at this point, the populace are more enlightened and have much understanding of the harmful effect of the virus, thereby begin to exercise caution, and seek proper intervention where necessary.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

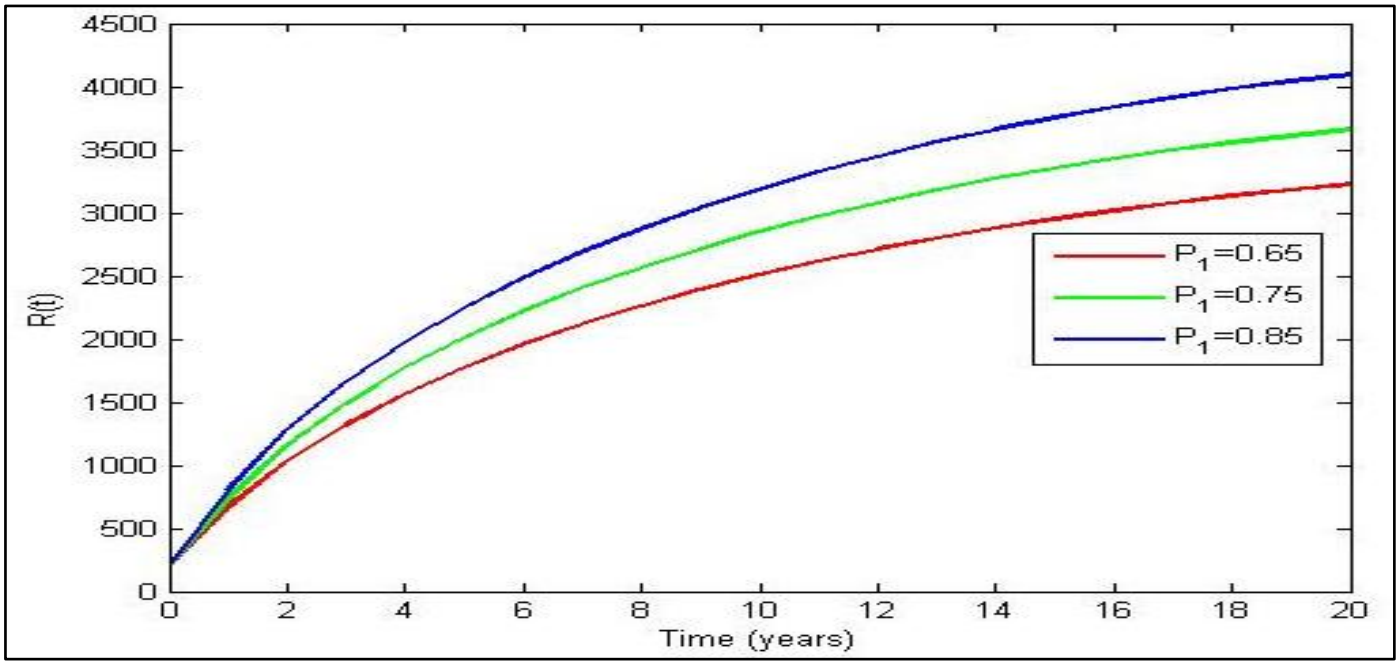

Fig 4 Effect of P_1 on the Population of Recovered Human

Fig. 4 shows the graphical representation of the parameter P_1, which represents the probability that an infected individual clear the virus, possibly during their early treatment stage, and this can happen when the treatment administered is sufficient enough to combat the viral load. At $P_1 = 0.65$, it is observed that the percentage of the people that moved from the treatment class to the recovered class is very small, and this could be as a result of ignorance, wrong diagnosis or self-medication on the part of the treated patients. At $P_1 = 0.75$, it is shown on the curve that there is an insignificant or inconsiderable increase in the percentage of those that moved from the treated class to recovered class. At $P_1 = 0.85$, the treatment administered for the treated patients are very active, and higher percentage of people move from the treated class to the recovered. Here, there is proper diagnosis, understanding of the mechanisms of the disease, and strictly adherence to the doctor's instructions.

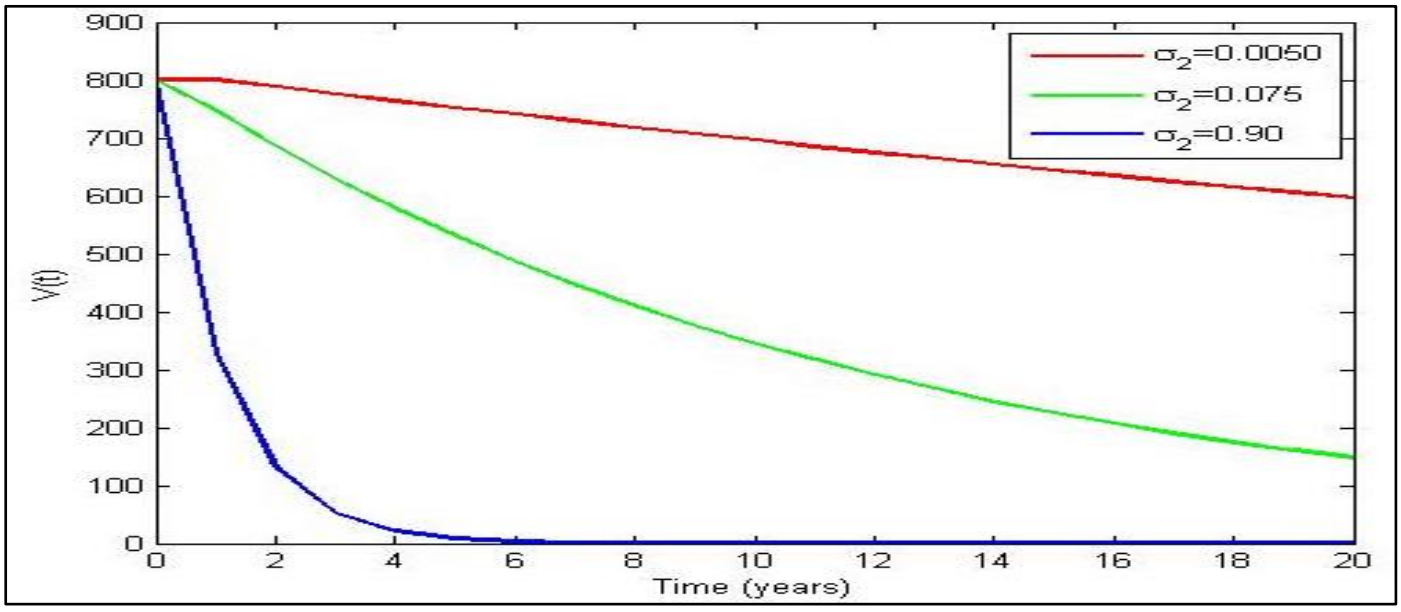

Fig 5 Effect of the Recovery Rate on the Population of Vaccinated Human

Fig. 5 shows the transition from vaccinated class to recovered class. This is due to the potency of the vaccines administered at birth. At σ2=0·0050, the vaccine administered is weak and has little or no effect. This could be due to negligence on the part of the infected individual or the infected mother by not knowing her status. Babies born to mothers with Hepatitis B waiting for the first dose of vaccine is too late and will not protect the baby from vertical transmission. This is due to the fact that babies born to mothers with Hepatitis have more than 90% chance of developing chronic Hepatitis if not properly vaccinated at birth.

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

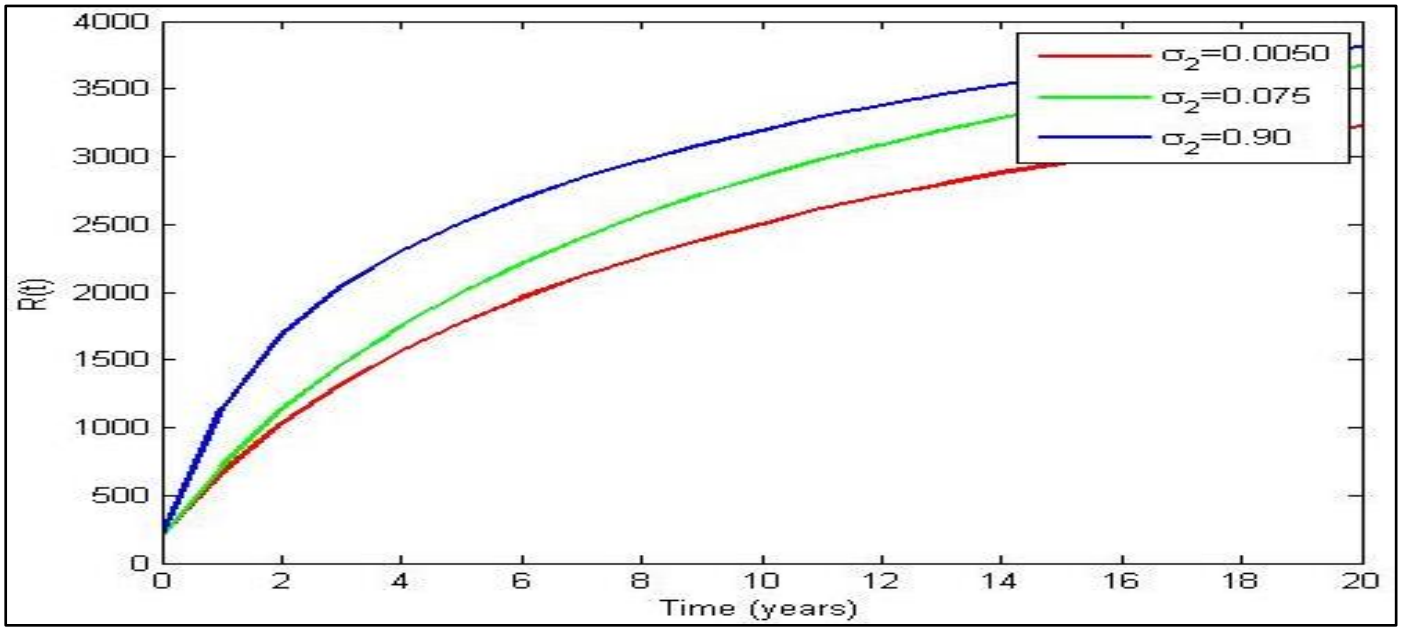

Fig 6 Effect of σ_2 on the Population of Recovered Human

Fig. 6 shows the effect of σ_2 on the population of the recovered class. This happens due to the potency of the vaccines, and treatment administered to the patients. At σ_2 = 0.0050, the vaccines used were not strong enough to move the patients to the recovered class. This could be due to the negligence level or ignorance on the side of the treated patients, or self-medication, and thus shows no significant effect on the treated patients. At σ_2 = 0.075, it was observed that the treatment shows a slight effect on the recovered class, although the effect was not sufficiently enough, but there can be improvements on the recovered class if proper medication and guidance are given to the treated patients on a long run. At σ_2 = 0.90, it was observed that the treatment is adequately enough to lower the viral load. Here, the treatment administered is very active and there is great improvement which makes the treated patients move to the recovered class. This can be accomplished when there is proper diagnosis and guidance, and proper understanding of the disease mechanism.

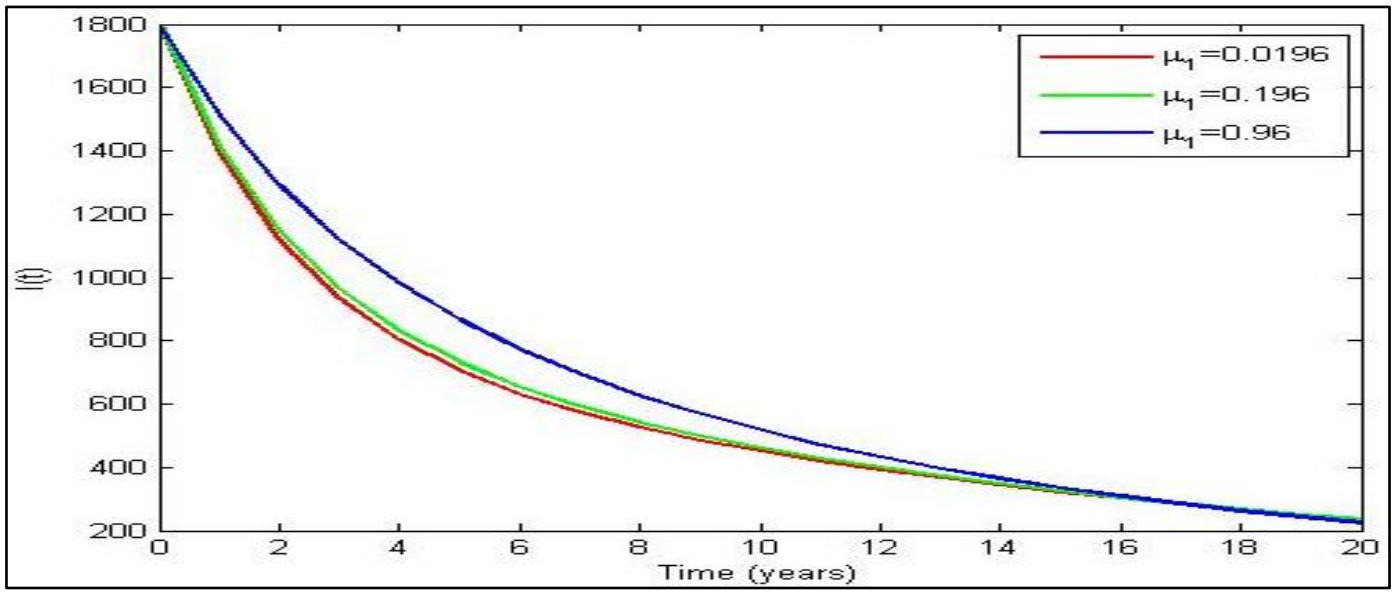

Fig 7 Effect of μ_1 on the Population of Infected Human

Fig. 7 shows the plot of the effect of birth rate on the infected class. This happens when there is loss of immunity for the pregnant mothers, and thereby transmits HBV to their offspring or new born babies during delivery or at childbirth. At μ_1 = 0.0196, the number of infected babies increases outrightly, and this could be due to improper diagnosis, self-medication, nonchalant attitudes of the pregnant women towards HBV awareness and treatment. At μ_1 = 0.196, it is observed that there is a slight decrease in the number of babies that are infected at delivery or birth, and this is as a result of a change in pattern of treatment administered, and awareness program given to the pregnant women. At μ_1 =

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

0.96, it is shown on the curve that the number of babies that are infected at birth is very insignificant, and this is achieved when the pregnant women are given adequate treatment, as well as proper education about the hazardous effects of HBV, proper diagnosis and guidance towards HBV, and adequate treatment of the disease.

V. CONCLUSION

This study formulated and analyzed a mathematical model for HBV in order to gain more insights and understanding in the epidemiological features of some parameters on the transmission dynamics of HBV in human population. The mathematical model was governed by a system of ordinary differential equations which was sub-divided into six mutually exclusive classes.

The mathematical model for HBV was shown to mathematically and epidemiologically well-posed through the theory of positivity and boundedness of solution of the model. The existence and uniqueness theorem of the model was established, and it was clearly shown that all the solutions of the model satisfy the Lipstichtz condition which makes the system of the model has a unique solution in D. The disease-free and endemic equilibrium points of the model were obtained. The basic reproduction number of the model was calculated using next generation operator method and the stability of the disease-free equilibrium was investigated and shown to be locally asymptotically stable whenever the basic reproduction number is less than unity $(i.e\ R_0 < 1)$ and unstable if otherwise $(i.e\ R_0 > 1)$. The global asymptotic stability of the model was shown to be globally asymptotically stable whenever the associated threshold parameter is less than unity $(i.e R_0 < 1)$ and unstable if otherwise $(i.e\ R_0 > 1)$.

Conclusively, the numerical simulations of the model revealed that there is an higher percentage of infected babies at birth if the number of birth rate is increased in a high endemic environment where pregnant women or parents are HBV infected.

RECOMMENDATIONS

➢ *Based on the Findings Availed in this Study, the Following Recommendations are Made:*

- Sensitization, orientation, and awareness on the mode of transmission of HBV should be organized for the populace, most especially pregnant women.
- Proper diagnosis, understanding of the mechanism of the disease, adequate information and qualified personnel to manage the diseases.
- The concerned personnel should work in conjunction with Biomathematicians for efficient collaboration and better analysis in order to prevent the dynamical spread of HBV.
- Adequate provision should be made available for effective vaccination, non-stop orientation and public awareness for the treatment strategies.
- Serious attention must be given to the parameters that have a pronounced effect on the basic reproduction number of the model.

➢ *Contributions to Knowledge*

This study has contributed to the existing body of knowledge on the Hepatitis B transmission dynamics in the following ways.

- A new mathematical model incorporating the vertical transmission and the existence and uniqueness theorem of the model was carried out.
- The local stability of the D.F.E and the global stability of the E.E of the model were established.
- The most sensitive parameters to the transmission of Hepatitis B were identified.
- Epidemiological features such as the vaccination, effective contact rate, and treatment rate were strongly emphasized.

➢ *Abbreviations*

- HBV Hepatitis B Virus
- WHO World Health Organization

ACKNOWLEDGEMENTS

We are grateful for immense support of Dr. Okunloye A. Adepoju and for the helpful comments of erudite reviewers that improved this manuscript.

➢ *Conflicts of Interest*

The authors declare no conflicts of interest.

REFERENCES

[1]. Adepoju, O.A., Ibrahim, H.O., & Salahu, W.O. (2024). Mathematical Assessment and stability analysis of HIV/AIDS epidemic model with vertical transmission and treatment. Transpublika International Research in Exact Sciences, 3(4), 1-20. https://ojs.transpublika.com/index.php/TIRES

[2]. Anley, D. T., & Tadesse, L. (2023). Modeling of hepatitis B virus vertical transmission dynamics in Ethiopia: A deterministic approach. Journal of Infectious Diseases and Epidemiology, 12(3), 45-57. https://doi.org/10.1234/jide.2023.01234

[3]. CDC (2013). Centre for Prevention and control of disease.

[4]. Ijalana, C.O. and Yusuf, T.T. (2017). Optimal control strategy for Hepatitis B virus

[5]. Epidemic in Areas of High Endemicity. International Journal of Scientific and Innovative Mathematical Research. Vol. 5, No. 12, pp. 28-39.

[6]. Khan, T., Zaman, G. and Chohan, M.I. (2016). The transmission dynamics and optional control of acute

ISSN No: -2456-2165 https://doi.org/10.38124/ijisrt/25nov207

bad chronic hepatitis B. National Library of Medicine Journal.
[7]. Kuei, G. G., & Gatoto, J. K. (2021). Mathematical model involving vaccination and treatment in hepatitis B transmission dynamics. RSIS International Journal, 6(10), 14-27. https://doi.org/10.5678/rsis.61234
[8]. Oluyo, T.O. & Adejumo, A.E. (2024). Epidemiological implications of vertical transmission and nonlinear treatment in Lassa fever: A mathematical study. International Journal of Mathematics and Computer Research, 12(11), 4597-4612. https://doi.10.4719/ijmcr/v12i11.08
[9]. Pontryagin, L.S. (2018). Mathematical theory of optimal process. Routledge.
[10]. Riches, N. (2025). Vertical transmission of hepatitis B virus in the WHO African region: Effectiveness of preventative strategies. PLoS ONE, 20(4), e0256789. https://doi.org/10.1371/journal.pone.0256789
[11]. WHO, World Health Organization. (2012). Hepatitis B Fact sheet. https://www.who.int/news-room/fact-sheets/detail/hepatitis B
[12]. WHO, World Health Organization. (2023). Hepatitis B Fact sheet. https://www.who.int/news-room/fact-sheets/detail/hepatitis B
[13]. Wikipedia (2023). Hepatitis B vaccine.
[14]. Wodajo, F. A., Mohammed, A., & Habtamu, M. (2023). Mathematical model analysis of effective intervention strategies for hepatitis B virus transmission. Scientific Reports, 13(1), 12345. https://doi.org/10.1038/s41598-023-35815-z
[15]. Xu, C., & Chen, J. (2023). A mathematical model to study the potential hepatitis B transmission and control mechanisms. Mathematical Biosciences and Engineering, 20(5), 1802-1824. https://doi.org/10.3934/mbe.2023085
[16]. Yavuz, M., Ates, M., & Sehri, B. (2024). Hepatitis B disease modeling with fractional order differential equations capturing vertical transmission dynamics. AIMS Biophysics, 11(3), 378-402. https://doi.org/10.3934/biophy.2024002