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Mathematical Analysis of Hepatitis B with Control Strategies and Emphasis on Double-Dose Vaccination

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ABSTRACT
Hepatitis B is a major global public health concern, causing both acute and chronic liver disease and contributing to significant morbidity and mortality worldwide. Effective vaccination strategies are critical to controlling its spread. This study investigated the spread patterns of Hepatitis B and evaluated the effectiveness of double-dose vaccination in minimizing transmission. A mathematical model was developed to represent the transmission dynamics of Hepatitis B in the human population, incorporating both acute and chronic infection stages. The basic reproduction number (R_0) was calculated to assess the potential for disease outbreak and persistence, and stability analysis was conducted to determine conditions under which the infection could be eradicated or maintained. A sensitivity analysis identified key parameters influencing R_0, such as transmission rate, vaccination coverage, and progression to chronic infection. Numerical simulations were performed to validate theoretical results and demonstrate the impact of implementing a double-dose vaccination regimen. The findings indicated that increasing vaccination coverage through a double-dose strategy significantly reduces the prevalence of Hepatitis B, providing valuable insights for public health strategies and policy development aimed at disease control.

Keywords:
Hepatitis B,
Double-dose
Vaccination,
Basic reproduction
Number,
Sensitivity analysis,
Numerical simulation

INTRODUCTION

Hepatitis B virus (HBV) infection continues to be a significant global health challenge, with chronic infection affecting hundreds of millions of people worldwide and contributing to severe liver disease, including cirrhosis and hepatocellular carcinoma (Rajbhandari et al., 2025; WHO, 2025). Recent studies report that the global burden of chronic HBV remains high despite widespread vaccination efforts and antiviral treatments (Al-Busafi & Alwassief, 2024). According to the World Health Organization, gaps in screening, vaccination coverage, and linkage to care contribute to persistent transmission and disease progression in many regions, particularly in low-resource settings (WHO, 2025). Regional research also underscores substantial variability in HBV prevalence and risk factors across countries and communities, further accentuating the need for locally tailored public health strategies. Transmission of HBV occurs through exposure to infected blood and bodily fluids, including through perinatal, sexual, and percutaneous routes (Olakunde et al., 2025; WHO, 2025). In high endemic regions such as sub-Saharan Africa, perinatal transmission remains a leading source of new chronic infections, particularly in settings with low birth-dose vaccine coverage (Wondmeneh & Mekonnen, 2024).

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Empirical evidence from African populations confirms these transmission patterns; for example, systematic reviews show elevated HBV seroprevalence among pregnant women across the continent, indicating ongoing perinatal and horizontal transmission risks in these groups (Wondmeneh & Mekonnen, 2024). Behavioral, health-system, and sociodemographic factors, including low awareness and limited access to preventive care, further compound these transmission risks in many communities.

Epidemiological research from Nigeria, one of the countries with the highest estimated HBV burden in West Africa, demonstrates persistent prevalence and diverse molecular characteristics of circulating virus strains. A recent cross-sectional screening study in Abuja found HBV infection among 4.3% of adult participants, with higher rates in specific age and sex groups, highlighting population-specific patterns of risk and the need for targeted vaccination and care programs (Akabuike et al., 2024). National-level analyses of molecular epidemiology in Enugu and Nasarawa States have identified multiple HBV genotypes and drug-resistant variants, underscoring the complexity of viral dynamics and implications for treatment strategies (Egbe et al., 2025). These findings align with broader systematic reviews documenting high chronic HBV prevalence and related risk factors in varied Nigerian populations, including key subgroups like incarcerated individuals (Adepoju et al., 2025). Prevention remains central to HBV control, with universal immunization and enhanced screening being pivotal components of global elimination strategies. Despite evidence demonstrating the effectiveness of HBV vaccination in reducing incidence, vaccination coverage varies significantly across regions and populations (Al-Busafi & Alwassief, 2024; Tuuryare et al., 2025). Research in Somalia, for example, highlights disparities in adult HBV vaccine uptake and associated factors, pointing to gaps in public health messaging and access to preventive services (Tuuryare et al., 2025). Likewise, studies of vaccination practices among healthcare workers in Nigeria reveal psychosocial and digital predictors affecting uptake, suggesting that tailored interventions are needed to improve vaccine acceptance in high-risk groups (Aremu et al., 2025).

Advances in HBV treatment and care continue to evolve, with current antiviral regimens effectively suppressing viral replication and improving clinical outcomes (Rajbhandari et al., 2025). However, challenges remain regarding long-term treatment adherence, monitoring for drug resistance, and ensuring equitable access to care in low-resource settings. Moreover, integrating HBV testing and treatment into existing maternal health services has been explored in Ghana to improve prevention of mother-to-child transmission, demonstrating feasibility and potential impact (Koray et al., 2025). Collectively, these empirical studies emphasize the multidimensional nature of HBV control spanning epidemiology, prevention, clinical care, and health systems strengthening and reinforce the need for sustained, evidence-driven policies and programs.

Turab *et al.* (2024) developed a **fractional HBV transmission model** using a Caputo derivative to better capture the complex kinetics of infection stages, including acute and chronic carriers. They derived the basic reproduction number and equilibrium points, and applied an **Artificial Neural Network (ANN)** to classify training, testing, and validation datasets for model behaviour. The study demonstrated that fractional-order modelling could reveal nuanced epidemic dynamics and offered graphical comparisons of different non-integer orders, suggesting potential utility for informing control strategies that incorporate vaccination effects. El-Shenawy *et al.* (2025) introduced a **nonlinear HBV model** and performed numerical simulation with the **Dickson collocation technique,** transforming the ordinary differential equations into a nonlinear algebraic system for efficient computation. Their analysis revealed that the Dickson approach had higher precision and better convergence rates compared to traditional ANN methods. The authors concluded that this numerical method could accurately simulate complex virus–host interactions and provide insights into long-term dynamics, advancing the toolkit for epidemiological HBV modelling.

Mirgichan *et al.* (2025) constructed a **deterministic compartmental model** capturing HBV transmission dynamics by including **passive immunity,** vaccination at birth, screening, and treatment strategies. They derived the basic reproduction number and conducted numerical simulations using demographic and vaccination data to assess control impacts. Their findings underscored that passive immunity and comprehensive vaccination significantly influenced disease transmission patterns, offering quantitative support for integrated public health interventions aimed at reducing endemic HBV prevalence. Zarin *et al.* (2024) developed a **reaction–diffusion SVEICHR model** incorporating spatial diffusion to dissect Hepatitis B spread across heterogeneous environments. They calculated the basic reproduction number through a next-generation matrix and conducted equilibrium stability analysis. Simulations implemented in MATLAB showed how spatial movement and diffusion influenced disease dynamics and evaluated vaccination and hospitalization as control strategies. This work extended traditional compartmental modelling by adding **spatial heterogeneity,** which allowed a more realistic depiction of HBV transmission over continuous landscapes.

The research aimed to investigate the spread patterns of Hepatitis B and assess the effectiveness of treatment and vaccination strategies in minimizing disease transmission. By constructing a mathematical framework, the study sought to provide a comprehensive

understanding of the mechanisms underlying Hepatitis B transmission and to identify critical factors that influence its spread. To achieve this, the study pursued several specific objectives. First, a mathematical model was developed to depict the transmission dynamics of Hepatitis B. Second, the basic reproduction number (R_0) of the model was computed to quantify the potential for disease outbreak and persistence. Third, the stability of the model was analyzed to determine the conditions under which the disease could be eradicated or persist in the population. Fourth, a sensitivity analysis was conducted to evaluate the impact of key parameters on the basic reproduction number, identifying which factors most strongly affected disease transmission. Finally, numerical simulations of the model were performed to validate the theoretical findings and provide visual and quantitative confirmation of the model's behavior under various scenarios.

MATERIALS AND METHODS

Model Formulation.

The human population at time t, denoted by $N(t)$ is sub-divided into Six(6) mutually exclusive compartments of Susceptible humans $S(t)$, First dose of vaccinated humans $V_1(t)$, Second dose of vaccinated humans $V_2(t)$, Acute stage infectious humans (first six months after infection) $A(t)$, Chronic stage infectious humans $C(t)$ and Recovered humans $R(t)$. The recruitment rate of humans into the susceptible population is denoted by Λ_s, the recruitment rate of the first dose vaccinated humans into the population is at the rate of Λ_v, and the recruitment rate of the chronic stage infectious humans into the population is at the rate of Λ_c. We denote ξ as the effective contact rate of the susceptible and infectious humans, k as the degree of infectiousness of the acute stage individuals in comparison to the chronic individuals with HPV B. Humans die naturally at the rate of μ and the chronic stage HPV B individuals die due to the disease at the rate of ψ. We denote φ_1 as the rate of vaccination of the susceptible humans and φ_2 as the rate at which the first dose of vaccine fails. The progression rate from acute stage to chronic stage of HPV B due to manifestation of clinical symptoms is denoted by δ. The natural recovery rate of acute stage of HPV B is σ, the rate of recovery in the chronic stage class due to treatment is θ and the recovery rate of the second dose vaccinated humans is ε. We denote the rate of the second dose of the HPV B vaccine by α. The total human population is denoted as:

$$N(t) = S(t) + V_1(t) + V_2(t) + A(t) + C(t) + R(t)$$

In formulating the mathematical model for Hepatitis B transmission, several assumptions were made to simplify and accurately represent the dynamics of the disease. It was assumed that individuals in the acute stage of Hepatitis B infection could progress to a chronic infection if they did not receive appropriate treatment, reflecting the clinical reality of disease progression. Additionally, a proportion of individuals in the acute stage were considered capable of natural recovery, thereby exiting the infectious class without medical intervention. Finally, it was assumed that the rate of vaccine failure following the second dose was extremely negligible, implying that individuals who completed the recommended vaccination schedule were effectively protected against infection. These assumptions provided the foundation for constructing a tractable and realistic model to explore the transmission and control of Hepatitis B.

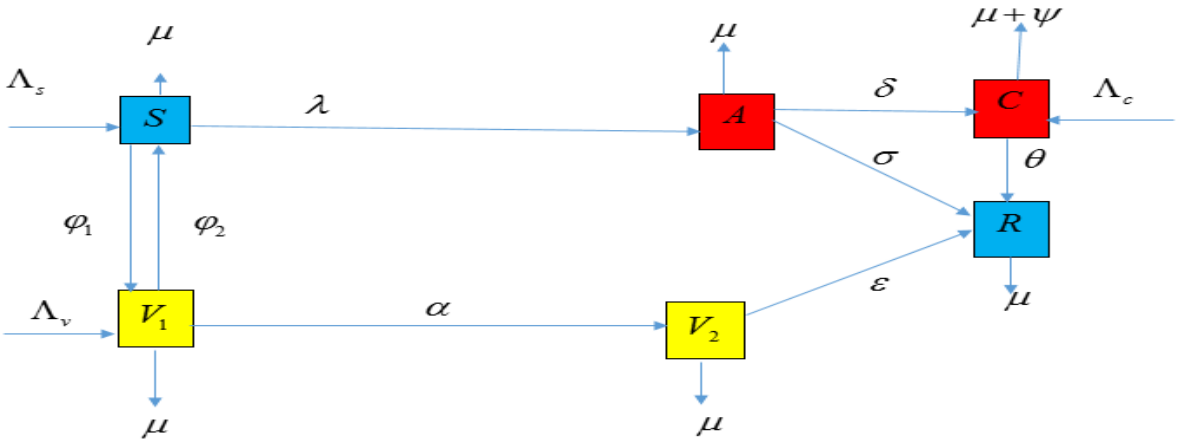

Fig. 1: Schematic diagram of the Hepatitis B model

Table 1: Variable and Parameters description

Variable	Description
S	Susceptible Humans
V_1	First dose vaccinated humans
V_2	Second dose vaccinated humans
A	Acute infectious humans (First six months after infection)
C	Chronic stage infectious humans
R	Recovered Humans
PARAMETER DESCRIPTION	
Λ_s	Recruitment rate of susceptible unvaccinated humans
Λ_v	Recruitment rate of first dose vaccinated humans
Λ_c	Recruitment rate of chronic infectious humans
φ_1	Rate of vaccination of susceptible humans
φ_2	Rate of first dosage vaccine failure
ξ	Contact rate of susceptible and infectious humans
μ	Natural death rate of humans
k	Degree of infectiousness of acute individual in comparison with chronic individuals with Hepatitis B
α	Rate of second dose of vaccine
ε	Recovery rate of second dose vaccinated humans
σ	Natural recovery rate of acute infectious humans
θ	Recovery rate of chronic infectious humans due to treatment
δ	Progression rate from acute to chronic infectious humans by developing symptoms
ψ	Disease induced death of the chronic infectious humans

Model Equations

Based on the model outlined above, the transmission dynamics of Hepatitis B in the population are described by the following system of differential equations:

$$\frac{dS}{dt} = \Lambda_s + \varphi_2 V_1 - (\lambda + \mu + \varphi_1) S,$$

$$\frac{dV_1}{dt} = \Lambda_v + \varphi_1 S - (\alpha + \mu + \varphi_2) V_1,$$

$$\frac{dV_2}{dt} = \alpha V_1 - (\varepsilon + \mu) V_2,$$

$$\frac{dA}{dt} = \lambda S - (\delta + \sigma + \mu) A,$$

$$\frac{dC}{dt} = \Lambda_c + \delta A - (\theta + \mu + \psi) \mathrm{C},$$

$$\frac{dR}{dt} = \sigma A + \theta C + \varepsilon V_2 - \mu R. \quad (1)$$

The force of infection of the Hepatitis B model in (1) is given as:

$$\lambda = \frac{\xi(kA + C)}{N},$$

RESULTS AND DISCUSSION

Invariant region of the Hepatitis B model

The solutions of the proposed Hepatitis B model are feasible for all t > 0, if they enter the invariant region D, which is given by:

$$D = \begin{Bmatrix} (S, V_1, V_2, A, C, R) : S > 0, V_1 > 0, \\ V_2 > 0, A > 0, C > 0, R > 0, \\ N < \frac{\Lambda_s}{\mu} < \frac{\Lambda_v}{\mu} < \frac{\Lambda_c}{\mu} \end{Bmatrix}$$

Proof

The total population of the humans in the Hepatitis B model is given as

$$N(t) = S(t) + V_1(t) + V_2(t) + A(t) + C(t) + R(t)$$

The sum of the differential equations is

$$N'(t) = S'(t) + V_1'(t) + V_2'(t) + A'(t) + C'(t) + R'(t)$$

On evaluating the algebraic terms, we obtain

$$N'(t) = \Lambda_h - (S + V_1 + V_2 + A + C + R)\mu - \psi C$$

$$N'(t) = \Lambda_h - \mu N - \psi C$$

$$\frac{dN}{dt} \le \Lambda_s - \mu N$$

Solving the differential equation using the integrating factor method, we obtained

$$N(t) \le \frac{\Lambda_s}{\mu} + \left(N(0) - \frac{\Lambda}{\mu} \right) e^{-\mu t}$$

Applying Birkhoff and Rota's theorem on the inequality, we obtain

$$0 \le N \le \frac{\Lambda_s}{\mu} \text{ as } t \to \infty$$

Thus, D is a positively invariant set under the flow described by the model (1) so that no solution path leaves through the boundary of region D.Thus, in this region, the Hepatitis B model can be considered as being epidemiologically and mathematically well posed.

Positivity of solution of the Hepatitis B model

It is necessary to prove that all state variable of the Hepatitis B model in are nonnegative for all time (t), for the model to be epidemiologically and mathematically well posed in a feasible region D given by:

$$D = \begin{Bmatrix} (S, V_1, V_2, A, C, R) \in R_+^6 : \\ (S + V_1 + V_2 + A + C + R) \le N \end{Bmatrix}$$

This is done by considering,

$$\{(S, V_1, V_2, A, C, R) \in R_+^6\}$$

Lemma 1:

Let the initial data for the model (1) be $(S, V_1, V_2, A, C, R) > 0$. Then the solutions (S, V_1, V_2, A, C, R) of the model (1) are positive for all time $t > 0$

Proof

Let $t = \sup \begin{Bmatrix} t > 0 : S > 0, V_1 > 0, V_2 > 0, \\ A > 0, \mathrm{C} > 0, R > 0 \in [0, t] \end{Bmatrix}$. Thus $t > 0.$

We have from the first equation that

$$\frac{dS}{dt} = \Lambda_s + \varphi_2 V_1 - (\lambda + \mu + \varphi_1) S,$$

$$\frac{dS}{dt} \ge -(\lambda + \mu + \varphi) S$$

This can also be written as

$$\int \frac{dS}{S} \ge -\int (\lambda + \mu + \varphi) dt$$

We obtained:

$$\ln S \ge -(\lambda + \mu + \varphi) t + C$$

$$S(t) \ge C e^{-(\lambda + \mu + \varphi) t}$$

Applying the initial condition; when $t = 0,\ S(0) = C$

Therefore, $S(t) \ge S(0) e^{-(\lambda + \mu + \varphi) t} \ge 0$ since $(\lambda_h + \mu_h) > 0$

Similarly, it can be shown that $V_1, V_2, A, C, R > 0$

Asymptotic stability of the disease-free equilibrium of the Hepatitis B model

The steady state where there is no infection (or absence of the disease), a point where $A=C=R=0$ is called the disease-free equilibrium point (DFE) of the Hepatitis B model which is given as:

$$\eta_0=\left\{S^*,V_1^*,V_2^*,A^*,C^*,R^*\right\}=$$

$$\left\{\frac{\Lambda_s}{\psi_1+\mu},\frac{\Lambda_v}{\alpha+\mu},\frac{\Lambda_v\alpha}{(\alpha+\mu)P_2},0,0,0\right\}$$

Basic Reproduction Number of the Hepatitis B Model

The basic reproduction number of Hepatitis B infected individuals denoted by R_0 is defined as the average number of secondary infections produced by a single Hepatitis B infectious individual introduced in a wholly susceptible population during his or her entire infectious period. We calculate the basic reproduction number by using the next generation operator method on the dynamical system (1).

Hence, it follows that

$R_0=\rho\left(FV^{-1}\right)$ where ρ is the dominant eigenvalue of FV^{-1}

$$F=\begin{bmatrix}\dfrac{\xi k\Lambda_s(\alpha+\mu)P_2}{(\psi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2} & \dfrac{\xi\Lambda_s(\alpha+\mu)P_2}{(\psi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2}\\ 0 & 0\end{bmatrix}$$

$$V=\begin{bmatrix}P_3 & 0\\ -\delta & P_4\end{bmatrix},\ V^{-1}=\begin{bmatrix}\dfrac{1}{P_3} & 0\\ \dfrac{\delta}{P_3P_4} & \dfrac{1}{P_4}\end{bmatrix},$$

$$FV^{-1}=\begin{bmatrix}\dfrac{\xi\Lambda_s(\alpha+\mu)P_2\left(kP_4+\delta\right)}{P_3\left(\left(\psi_1+\mu\right)\left(P_2+\alpha\right)\Lambda_v+\Lambda_s(\alpha+\mu)P_2\right)P_4} & \dfrac{\xi\Lambda_s(\alpha+\mu)P_2}{P_4\left(\left(\psi_1+\mu\right)\left(P_2+\alpha\right)\Lambda_v+\Lambda_s(\alpha+\mu)P_2\right)}\\ 0 & 0\end{bmatrix}$$

Therefore, the greatest eigenvalue of FV^{-1} the basic reproduction number of the Hepatitis B only model is

$$R_0=\frac{\xi\Lambda_sP_2\left(P_4\alpha k+P_4k\mu+\alpha\delta+\delta\mu\right)}{P_3P_4\left(P_2\alpha\Lambda_s+P_2\mu\Lambda_s+P_2\mu\Lambda_v+P_2\Lambda_v\varphi_1+\alpha\mu\Lambda_v+\alpha\Lambda_v\varphi_1\right)}$$

,

$$R_0=\frac{\xi\Lambda_s(\alpha+\mu)P_2\left(P_4k+\delta\right)}{P_3\left(\left(\psi_1+\mu\right)\left(P_2+\alpha\right)\Lambda_v+\Lambda_s(\alpha+\mu)P_2\right)P_4}$$

.

Local Asymptotic Stability of the DFE of the Hepatitis B Model

Theorem 1

The disease-free equilibrium point of the Hepatitis B only is locally asymptotically stable (LAS) if $R_0<1$, *and unstable if* $R_0>1$.

Proof

Using Jacobian matrix to prove the local stability of the disease free equilibrium point

$$J\left(\eta_0\right)=\begin{bmatrix}-(\varphi_1+\mu) & \varphi_2 & 0 & -\dfrac{\xi k\Lambda_s(\alpha+\mu)P_2}{(\varphi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2} & -\dfrac{\xi\Lambda_s(\alpha+\mu)P_2}{(\varphi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2} & 0\\ \varphi_1 & -P_1 & 0 & 0 & 0 & 0\\ 0 & \alpha & -P_2 & 0 & 0 & 0\\ 0 & 0 & 0 & \dfrac{\left(-P_3(\mu+\varphi_1)\Lambda_v+\Lambda_s(\mu+\alpha)(k\xi-P_3)\right)P_2-P_3\alpha\Lambda_v(\mu+\varphi_1)}{\left((\mu+\varphi_1)\Lambda_v+\Lambda_s(\mu+\alpha)\right)P_2+\alpha\Lambda_v(\mu+\varphi_1)} & \dfrac{\xi\Lambda_s(\alpha+\mu)P_2}{(\varphi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2} & 0\\ 0 & 0 & 0 & \delta & -P_4 & 0\\ 0 & 0 & \varepsilon & \sigma & 0 & -\mu\end{bmatrix}$$

Since the diagonal third and sixth columns consist of only the diagonal elements which makes $-\mu,-P_2$, we can reduce $J(\eta_0)$ to

$$J_1(\eta_0)=\begin{bmatrix} -(\varphi+\mu) & \varphi_2 & -\dfrac{\xi k\Lambda_s(\alpha+\mu)P_2}{(\varphi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2} & -\dfrac{\xi\Lambda_s(\alpha+\mu)P_2}{(\varphi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2} \\ \varphi & -P_1 & 0 & 0 \\ 0 & 0 & \dfrac{\left(-P_3(\mu+\varphi_1)\Lambda_v+\Lambda_s(\mu+\alpha)(k\xi-P_3)\right)P_2-P_3\alpha\Lambda_v(\mu+\varphi_1)}{\left((\mu+\varphi_1)\Lambda_v+\Lambda_s(\mu+\alpha)\right)P_2+\alpha\Lambda_v(\mu+\varphi_1)} & \dfrac{\xi\Lambda_s(\alpha+\mu)P_2}{(\varphi_1+\mu)(P_2+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)P_2} \\ 0 & 0 & \delta & -P_4 \end{bmatrix}$$

The characteristics polynomial of $J_1(\eta_0)$ is

$$\lambda^4+A\lambda^3+B\lambda^2+C\lambda+D=0$$

Where,

$$A=(\varphi_1+\mu+P_1)\left(\left(-\psi_1-\mu\right)\Lambda_v-\Lambda_s(\alpha+\mu)\right)P_2-\alpha\Lambda_v(\varphi_1+\mu)\Big)$$
$$+\left(-(P_3+P_4)(\varphi_1+\mu)\Lambda_v+\Lambda_s(\alpha+\mu)(k\xi-P_3-P_4)\right)P_2-(\Lambda_v\alpha P_3+\Lambda_v\alpha P_4)(\varphi_1+\mu)$$

$$B=(P_1\mu-\varphi_1(\varphi_2-P_1))\left(\left(\left(-\psi_1-\mu\right)\Lambda_v-\Lambda_s(\alpha+\mu)\right)P_2-\alpha\Lambda_v(\varphi_1+\mu)\right)$$
$$+(\varphi_1+\mu+P_1)\left(\left(-(P_3+P_4)(\psi_1+\mu)\Lambda_v+\Lambda_s(\alpha+\mu)(k\xi-P_3-P_4)\right)P_2-(\Lambda_v\alpha P_3+\Lambda_v\alpha P_4)(\varphi_1+\mu)\right)$$
$$+\left(-P_3P_4(\varphi_1+\mu)\Lambda_v+\left((k\xi-P_3)P_4+\xi\delta\right)(\alpha+\mu)\Lambda_s\right)P_2-\Lambda_v\alpha P_4P_3(\varphi_1+\mu)$$

$$C=(P_1\mu-\varphi_1(\varphi_2-P_1))\left(\left(-(P_3+P_4)(\varphi_1+\mu)\Lambda_v+\Lambda_s(\alpha+\mu)(k\xi-P_3-P_4)\right)P_2-(\Lambda_v\alpha P_3+\Lambda_v\alpha P_4)(\varphi_1+\mu)\right)$$
$$+(\varphi_1+\mu+P_1)\left(\left(-P_3P_4(\varphi_1+\mu)\Lambda_v+\left((k\xi-P_3)P_4+\xi\delta\right)(\alpha+\mu)\Lambda_s\right)P_2-\Lambda_v\alpha P_4P_3(\varphi_1+\mu)\right)$$

$$D=(P_1\mu-\varphi_1(\varphi_2-P_1))\left(\left(-P_3P_4(\psi_1+\mu)\Lambda_v+\left((k\xi-P_3)P_4+\xi\delta\right)(\alpha+\mu)\Lambda_s\right)P_2-\Lambda_v\alpha P_4P_3(\psi_1+\mu)\right)$$
$$+(1-R_0)$$

Applying Routh-Hurwitz criterion to the Characteristics polynomial, we have that

$$(1-R_0)>0$$
$$\Rightarrow R_0<1$$

Thus the DFE point of the Hepatitis B only model is locally asymptotically stable.

Global Asymptotic Stability of the Disease free equilibrium point of the Hepatitis B Model.

To investigate the global stability of the disease-free equilibrium, we use the technique implemented by Castillo-Chavez and song.

To do this, we write the equation in the uninfected class as

$$\frac{dX}{dt}=F(X,Z)$$

And we re-write the equation in the infected class as

$$\frac{dZ}{dt}=G(X,Z)$$

Where $X=(S,V_1,V_2,R)\in ¡^4_+$ denotes the uninfected population and

$Z=(A,C)\in ¡^2_+$ denotes the infected population

$\eta_0=(X^*,0)$ represent the disease free equilibrium of the system, and it globally asymptotically stable if it satisfies the following conditions:

$H_1:\dfrac{dX}{dt}=F(X^*,0),X^*$ is globally asymptotically stable

$$H_2:\frac{dZ}{dt}=D_ZG(X^*,0)Z-\hat{G}(X,Z)$$

$\hat{G}(X,Z) \geq 0$ for all $(X,Z) \in D$ and where $D_Z G(X^*,0)$ is an M- matrix (i.e the diagonal elements are no-negative and it is also the Jacobian of $\hat{G}(X,Z) \geq 0$ evaluated at $(X^*,0)$.

If the system satisfies the above condition, then the theorem below holds.

Theorem 2

The equilibrium point $\eta_0 = (X^*,0)$. *is globally asymptotically stable if* $R_0 \leq 1$

$$F(X,Z) = \begin{bmatrix} \Lambda_s + \varphi_2 V_1 - (\lambda + \mu + \varphi_1)S, \\ \Lambda_v + \varphi_1 S - (\alpha + \mu + \varphi_2)V_1, \\ \alpha V_1 - (\varepsilon + \mu)V_2, \\ \sigma A + \theta C + \varepsilon V_2 - \mu R. \end{bmatrix}$$

$$G(X,Z) = \begin{bmatrix} \lambda S - (\delta + \sigma + \mu)A \\ \Lambda_c + \delta A - (\theta + \mu + \psi)C \end{bmatrix}$$

At disease free equilibrium,

H_1:

$$\frac{dS}{dt} = \Lambda_h - (\varphi + \mu)S$$

$$\frac{dV_1}{dt} = \Lambda_v - (\alpha + \mu)S$$

$$\frac{dV_2}{dt} = \alpha V_1^* - P_2 V_2^*$$

$$\frac{dR}{dt} = 0$$

H_2:

$$D_z G(X^*,0)Z = \begin{bmatrix} \frac{\xi k \Lambda_s (\alpha + \mu)P_2}{(\varphi_1 + \mu)(P_2 + \alpha)\Lambda_v + \Lambda_s(\alpha + \mu)P_2} - (\delta + \sigma + \mu)A \\ \Lambda_c + \delta A - (\theta + \mu + \psi)C \end{bmatrix}$$

$$\hat{G}(X,Z) = D_Z G(X^*,0)Z - G(X,Z)$$

$$\hat{G}(X,Z) = \begin{bmatrix} \frac{\xi k \Lambda_s (\alpha + \mu)P_2}{(\varphi_1 + \mu)(P_2 + \alpha)\Lambda_v + \Lambda_s(\alpha + \mu)P_2}\left(1 - \frac{S}{N}\right) \\ 0 \end{bmatrix}$$

Clearly, $1 \geq \frac{S}{N}$ this implies that $\hat{G}(X,Z) \geq 0$.

Therefore the disease-free equilibrium of the Hepatitis B only model is globally asymptotically stable.

Endemic Equilibrium Point of the Hepatitis B Model

The endemic equilibrium point is the steady state where there is persistence or prevalence of a disease in the population.

Theorem 3

The endemic equilibrium point of the Hepatitis B model in (1) is stable if $R_0 > 1$ *and unstable if* $R_0 < 1$.

Proof

To obtain the endemic equilibrium we set the RHS of the differential equations in (1) to zero and solve for the state variables.

Thus, at the endemic equilibrium point,

$$\frac{dS}{dt} = \frac{dV_1}{dt} = \frac{dV_2}{dt} = \frac{dA}{dt} = \frac{dC}{dt} = \frac{dR}{dt} = 0.$$

Let $\eta^{**} = (S^{**}, V_1^{**}, V_2^{**}, A^{**}, C^{**}, R^{**})$ be the endemic equilibrium point.

We have that,

$$S^{**} = \frac{P_1\Lambda_s + \Lambda_v\varphi_2}{(\lambda^{**} + \mu + \varphi_1)P_1 - \varphi_2\varphi_1}$$

$$V_1^{**} = \frac{(\Lambda_s + \Lambda_v)\varphi_1 + \Lambda_v(\lambda^{**} + \mu)}{(P_1 - \varphi_2)\varphi_1 + P_1(\lambda^{**} + \mu)}$$

$$V_2^{**} = \frac{\left((\Lambda_s + \Lambda_v)\varphi_1 + \Lambda_v(\lambda^{**} + \mu)\right)\alpha}{P_2\left((P_1 - \varphi_2)\varphi_1 + P_1(\lambda^{**} + \mu)\right)}$$

$$A^{**} = \frac{\lambda^{**}(P_1\Lambda_s + \Lambda_v\varphi_2)}{P_3\left((\lambda^{**} + \mu + \varphi_1)P_1 - \varphi_2\varphi_1\right)}$$

$$\left(\Lambda_c(\lambda^{**} + \mu + \varphi_1)P_3 + \delta\lambda^{**}\Lambda_s\right)P_1$$

$$C^{**} = \frac{-\varphi_2\left(P_3\Lambda_c\varphi_1 - \delta\lambda^{**}\Lambda_v\right)}{P_3\left((\lambda^{**} + \mu + \varphi_1)P_1 - \varphi_2\varphi_1\right)P_4}$$

$$\begin{pmatrix} \theta\left((\lambda^{**} + \mu + \varphi_1)P_1 - \varphi_2\varphi_1\right)\Lambda_c P_3 + \\ \lambda^{**}(P_4\sigma + \delta\theta)(P_1\Lambda_s + \Lambda_v\varphi_2) \end{pmatrix} P_2$$

$$R^{**} = \frac{+P_4\left(\Lambda_v\lambda^{**} + (\Lambda_s + \Lambda_v)\varphi_1 + \Lambda_v\mu\right)P_3\varepsilon\alpha}{P_4\left((\lambda^{**} + \mu + \varphi_1)P_1 - \varphi_2\varphi_1\right)P_2\mu P_3}$$

Substituting them into the force of infection for the Hepatitis B disease transmission in the rodents' population, $\lambda^{**} = \frac{\xi\left(kA^{**} + C^{**}\right)}{N^{**}}$, we obtained

$$A_1\lambda_h^{**2} + A_2\lambda_h^{**} + A_3 = 0 \qquad (2)$$

$$A_2 = \left(\left(\left(\left(\varphi_1 + \varphi_2 + \mu\right)\Lambda_v + \Lambda_s\left(\varphi_1 + P_1\right)\right)P_4 - \left(\left(\varphi_2 - P_1\right)\varphi_1 - P_1(\mu - \xi)\right)\Lambda_c\right)P_3 - \xi\left(P_1\Lambda_s + \Lambda_v\varphi_2\right)\left(P_4k + \delta\right)\right)P_2$$
$$+P_4\alpha\left(\left(\mu + \varphi_1\right)\Lambda_v + \Lambda_s\varphi_1\right)P_3$$

$$A_3 = -P_1P_2P_3\mu\xi\Lambda_c - P_1P_2P_3\xi\Lambda_c\varphi_1 + P_2P_3\xi\Lambda_c\varphi_1\varphi_2\left(1 - R_0\right)$$

The phenomenon of backward bifurcation, which is characterized by co-existence of a stable disease-free equilibrium point and a stable endemic equilibrium when the associated reproduction number of Hepatitis B model is less than unity. Biologically, the implication of backward bifurcation is that the necessary condition for the effective control of Hepatitis B in the population when the reproduction number is less than unity is no longer sufficient. In backward bifurcation, multiple endemic equilibria must exist. Thus, this implies that there are three cases which are to be considered of the Hepatitis B endemic polynomial depending on the sign of A_2 and A_3 since A_1 is always positive. That is;

1) If $A_2 < 0$ and $A_3 = 0$ or $A_2^2 - 4A_1A_3 = 0$, then eq. (2) has a unique endemic equilibrium point (one positive root) and no possibility of backward bifurcation.
2) If $A_2 < 0$, $A_3 > 0$ and $A_2^2 - 4A_1A_3 > 0$, then eq. (2) has two endemic equilibria (two possible roots), and therefore it is possible for backward bifurcation to occur.
3) Otherwise, there is none.

Where,

$$A_1 = \left(\left(\left(\varphi_2 + P_3\right)P_4 + \delta\varphi_2\right)\Lambda_v + P_1\left(P_3\Lambda_c + P_4\Lambda_s + \delta\Lambda_s\right)\right)P_2 + P_3P_4\alpha\Lambda_v$$

However it is important to note that A_3 is always positive if $R_0 < 1$ and negative if $R_0^Y > 1$.

Sensitivity Analysis of the Hepatitis B Model

Sensitivity analysis is carried out to determine the parameters that enhances the spread as well as control of an infection in a population.

The sensitivity index of the reproduction number of the Hepatitis B model with respect to any parameter say p is given by:

$$\Im_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}.$$

Given that

$$R_0 = \frac{\xi\Lambda_s\left(\alpha + \mu\right)P_2\left(P_4k + \delta\right)}{P_3\left(\left(\psi_1 + \mu\right)\left(P_2 + \alpha\right)\Lambda_v + \Lambda_s\left(\alpha + \mu\right)P_2\right)P_4},$$

$$R_0 = \frac{\xi\Lambda_s\left(\alpha + \mu\right)\left(\varepsilon + \mu\right)\left(\left(\theta + \mu + \psi\right)k + \delta\right)}{\left(\delta + \sigma + \mu\right)\left(\left(\psi_1 + \mu\right)\left(\varepsilon + \mu + \alpha\right)\Lambda_v + \Lambda_s\left(\alpha + \mu\right)\left(\varepsilon + \mu\right)\right)\left(\theta + \mu + \psi\right)}$$

$$\Im_\xi^{R_0} = 1.0000$$

$$\Im_{\Lambda_s}^{R_0} = \frac{\left(\psi_1 + \mu\right)\left(\epsilon + \mu + \alpha\right)\Lambda_v}{\left(\Lambda_s + \Lambda_v\right)\mu^2 + \left(\left(\psi_1 + \alpha + \epsilon\right)\Lambda_v + \Lambda_s\left(\alpha + \epsilon\right)\right)\mu + \psi_1\left(\alpha + \epsilon\right)\Lambda_v + \Lambda_s\alpha\epsilon} = 0.4737$$

$$\Im_\alpha^{R_0} = -\frac{\Lambda_v\epsilon\left(\psi_1 + \mu\right)\alpha}{\left(\left(\Lambda_s + \Lambda_v\right)\mu^2 + \left(\left(\psi_1 + \alpha + \epsilon\right)\Lambda_v + \Lambda_s\left(\alpha + \epsilon\right)\right)\mu + \psi_1\left(\alpha + \epsilon\right)\Lambda_v + \Lambda_s\alpha\epsilon\right)\left(\alpha + \mu\right)} = -0.0504$$

$$\Im_\mu^{R_0} = -\frac{\xi\Lambda_s\left(\mu + \alpha\right)\left(\epsilon + \mu\right)\left(\left(\theta + \mu + \psi\right)k + \delta\right)}{\left(\delta + \sigma + \mu\right)^2\left(\left(\mu + \psi_1\right)\left(\epsilon + \mu + \alpha\right)\Lambda_v + \Lambda_s\left(\mu + \alpha\right)\left(\epsilon + \mu\right)\right)\left(\theta + \mu + \psi\right)} -$$
$$\frac{\xi\Lambda_s\left(\mu + \alpha\right)\left(\epsilon + \mu\right)\left(\left(\theta + \mu + \psi\right)k + \delta\right)\left(\left(\epsilon + \mu + \alpha\right)\Lambda_v + \left(\mu + \psi_1\right)\Lambda_v + \Lambda_s\left(\epsilon + \mu\right) + \Lambda_s\left(\mu + \alpha\right)\right)}{\left(\delta + \sigma + \mu\right)\left(\left(\mu + \psi_1\right)\left(\epsilon + \mu + \alpha\right)\Lambda_v + \Lambda_s\left(\mu + \alpha\right)\left(\epsilon + \mu\right)\right)^2\left(\theta + \mu + \psi\right)} = -0.1370$$

$$\Im_{\varepsilon}^{R_0} = -\frac{\Lambda_v \epsilon\left(\psi_1+\mu\right)\alpha}{(\epsilon+\mu)\left(\left(\Lambda_s+\Lambda_v\right)\mu^2+\left(\left(\psi_1+\alpha+\epsilon\right)\Lambda_v+\Lambda_s(\alpha+\epsilon)\right)\mu+\psi_1(\alpha+\epsilon)\Lambda_v+\Lambda_s\alpha\epsilon\right)} = -0.0579$$

$$\Im_{\theta}^{R_0} = -\frac{\delta\theta}{\left((\theta+\mu+\psi)k+\delta\right)(\theta+\mu+\psi)} = -0.0051$$

$$\Im_{\psi}^{R_0} = -\frac{\delta\psi}{\left((\theta+\mu+\psi)k+\delta\right)(\theta+\mu+\psi)} = -0.1039$$

$$\Im_{k}^{R_0} = \frac{(\theta+\mu+\psi)k}{(\theta+\mu+\psi)k+\delta} = 0.7037,$$

$$\Im_{\delta}^{R_0} = -\frac{\left((\theta+\mu+\psi)k-\mu-\sigma\right)\delta}{(\delta+\sigma+\mu)\left((\theta+\mu+\psi)k+\delta\right)} = -0.0690,$$

$$\Im_{\sigma}^{R_0} = -\frac{\sigma}{\delta+\sigma+\mu} = -0.0909,$$

$$\Im_{\Lambda_v}^{R_0} = -\frac{(\psi_1+\mu)(\epsilon+\mu+\alpha)\Lambda_v}{(\psi_1+\mu)(\epsilon+\mu+\alpha)\Lambda_v+\Lambda_s(\alpha+\mu)(\epsilon+\mu)} = -0.4737$$

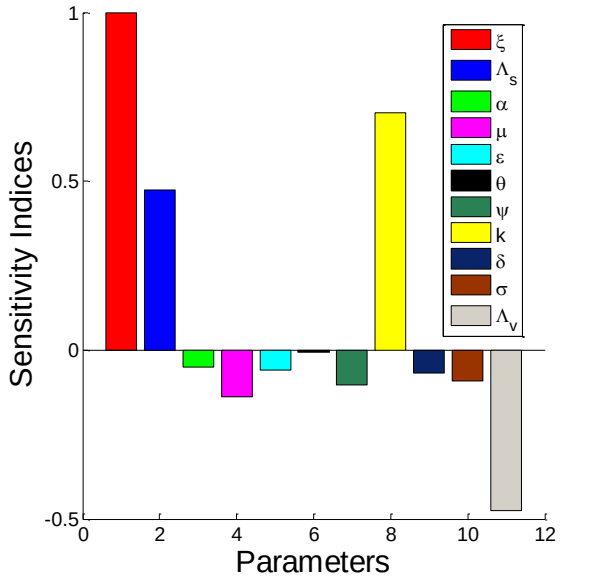

Figure 2 Bar chat of Hepatitis B sensitivity Indices

The results of the sensitivity analysis revealed the influence of different model parameters on the spread of Hepatitis B within the human population. Parameters that exhibited positive sensitivity indices were found to promote the transmission of the virus, indicating that increases in these parameters lead to a higher basic reproduction number (R_0) and, consequently, a greater prevalence of infection (Agbata et al, 2025). These parameters typically include factors such as the transmission rate, contact rate between susceptible and infected individuals, or the progression rate from acute to chronic infection, all of which directly enhance the potential for disease spread. Conversely, parameters with negative sensitivity indices were observed to suppress the transmission of Hepatitis B, meaning that increases in these factors result in a lower (R_0) and reduced prevalence of infection. Such parameters often encompass vaccination coverage, recovery rate of acutely infected individuals, or effectiveness of treatment interventions, all of which act to mitigate the spread of the virus. Understanding the relative sensitivity of these parameters provides critical insight into which aspects of disease dynamics are most impactful and can inform the design of targeted public health strategies to control and reduce the burden of Hepatitis B.

Numerical Simulations

Table 2. Parameter Values and Sources

Parameter	Value	Sources
Λ_s	0.29	Assumed
Λ_v	0.2	Assumed
Λ_c	0.11	Assumed
φ_1	0.52	Atnaw et al. (2023)
φ_2	0.04	Shepard et al. (2006)
ξ	0.24	Atnaw et al. (2023)
μ	0.9	Atnaw et al. (2023)
k	0.5	Pang and Zhou (2010)
α	0.72	Atnaw et al. (2023)
ε	0.51	Assumed
σ	0.12	Atnaw et al. (2023)
θ	0.025	Zou et al. (2010)
δ	0.3	Atnaw et al. (2023)
ψ	0.5	Atnaw et al. (2023)

In this section, we present the graphical representations of the numerical simulations of our Hepatitis B model.

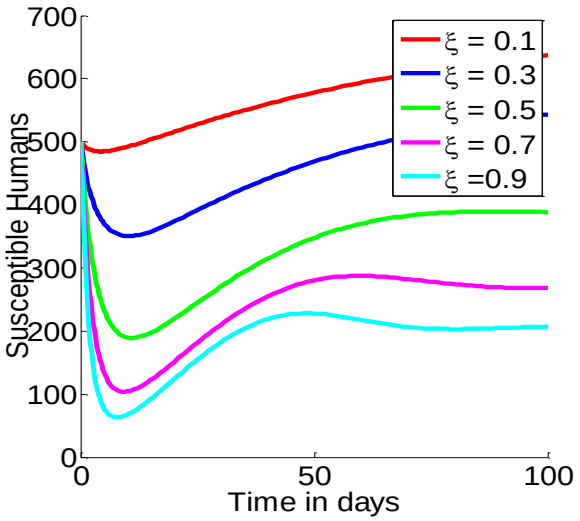

Figure 3a. Effect of varying ξ on susceptible Humans

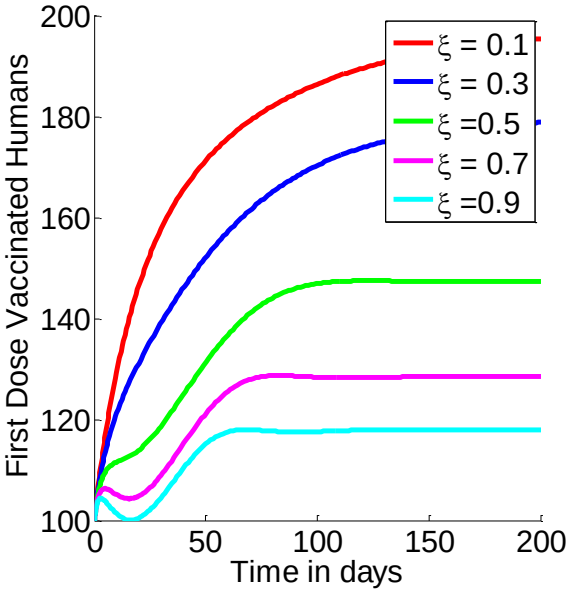

Figure 3b. Effect of varying ξ on V_1 humans

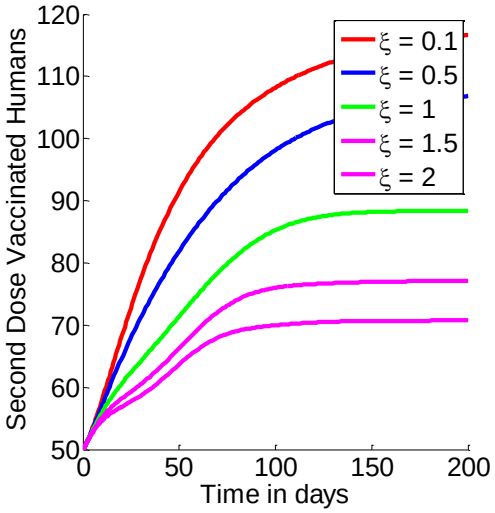

Figure 4a. Effect of varying ξ on V_2 Humans

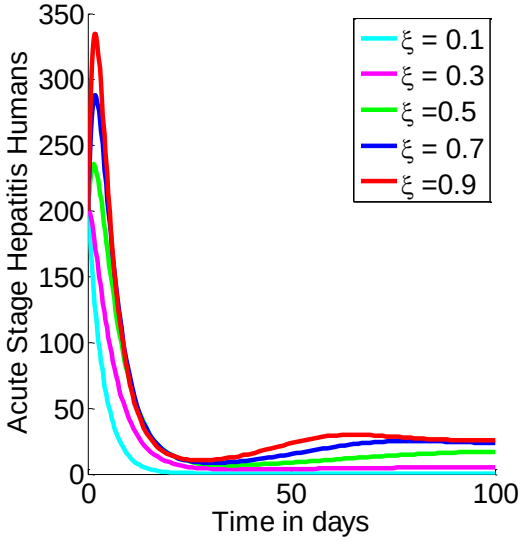

Figure 4b. Effect of varying ξ on Acute stage Hepatitis Humans

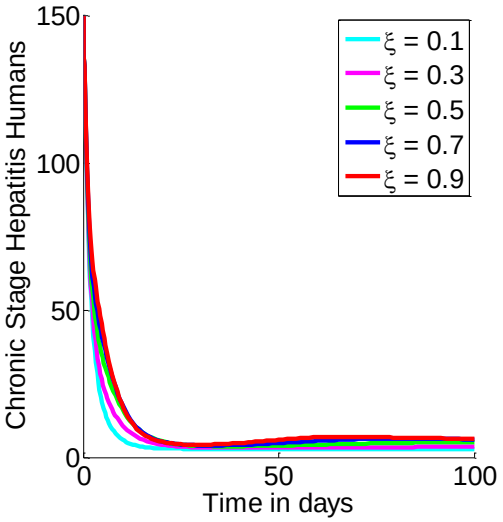

Figure 5a. Effect of varying β_b on Treated Humans

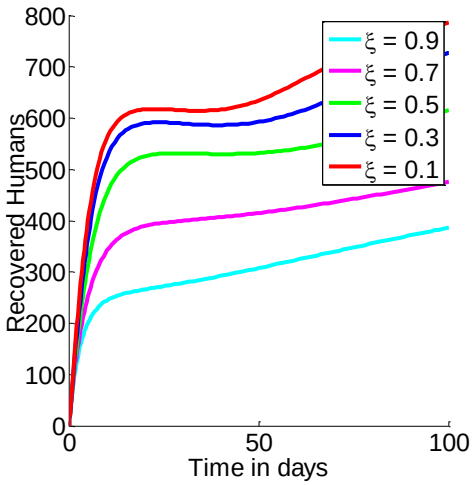

Figure 5b. Effect of varying β_b on Recovered Humans

As ξ increases, the force of infection λ also increases, resulting in a higher rate at which susceptible individuals contract the infection as seen in figure **3a**. This leads to a more rapid decline in the susceptible population over time with higher values of ξ. In contrast, for lower values of ξ, the decrease in S is more gradual, indicating a slower spread of the infection. In scenarios with very high ξ, S declines sharply, reflecting a rapid transmission rate. The depletion of susceptible individuals is a critical indicator of the infection's spread through the population, and high ξ values exacerbate this decline significantly. The V_1 compartment includes individuals who have received the first dose of the vaccine. Initially, V_1 increases as vaccination efforts ($\varphi_1 S$) are implemented as seen in figure **3b**. However, as ξ increases, the infection pressure accelerates the transition from V_1 to either the V_2 compartment or the infection compartments, reducing the duration individuals stay in V_1. Consequently, for higher ξ, V_1 reaches a peak earlier and starts to decline sooner compared to lower ξ values. This dynamic reflects the balance between vaccination and infection pressure in the population.

The V_2 compartment rises as individuals from V_1 receive their second dose of the vaccine as observed from figure **4a**. With higher ξ, there is an increased urgency to move from V_1 to V_2 due to the heightened infection risk. This results in a quicker rise and potentially a higher peak in V_2. However, this also depends on the capacity to vaccinate individuals promptly. Higher values of ξ may cause a strain on vaccination efforts, influencing the V_2 compartment dynamics significantly. The A compartment, representing individuals with acute hepatitis B infection, exhibits a notable increase as ξ rises. The rate at which susceptible individuals become acutely infected (λS) is directly influenced by ξ. Thus, with higher ξ, the A compartment shows a steeper and more pronounced rise, leading to higher peaks of acute infection. Lower ξ values result in a slower increase and a lower peak of acute infections, indicating a more controlled spread of the disease. This compartment is crucial in understanding the immediate burden of disease and the pressure on healthcare resources. Chronic infections, represented by the C compartment, accumulate as a proportion of acutely infected individuals (δA) transition into this state. With increasing ξ, the influx into C from A becomes more pronounced, leading to a higher accumulation of chronic cases over time. The difference in the C compartment between various ξ values becomes more significant, reflecting the impact of increased infection pressure on long-term disease outcomes. The chronic infection compartment is essential for understanding the long-term health impacts and the need for ongoing medical care and monitoring.

The R compartment includes individuals who recover from acute infection (σA) or chronic infection (θC), as well as those who clear the infection through

vaccination effects. Higher ξ values lead to faster and larger accumulation in R due to higher transition rates from A and C as the infection spreads more rapidly. This results in more recoveries in the population, which can be seen as both a positive outcome of effective immune responses and a consequence of the higher overall burden of disease from figure **5b**. The recovery rates reflect the overall dynamics of disease resolution and the effectiveness of vaccination and natural immunity.

CONCLUSION

In this study, we developed a mathematical model to investigate the transmission dynamics of Hepatitis B, explicitly incorporating treatment as a control measure. Using the model, we calculated the basic reproduction number (R_0), which provided insight into the potential for disease outbreak and persistence within the population. The analysis revealed that the system exhibited local asymptotic stability when $R_0 < 1$, global asymptotic stability under specific parameter conditions, and maintained a stable endemic equilibrium when $R_0 > 1$. To understand the influence of individual parameters on disease dynamics, we performed a sensitivity analysis. Parameters with positive sensitivity indices, such as the transmission rate and progression to chronic infection, were found to enhance the spread of Hepatitis B, while parameters with negative sensitivity indices, including vaccination coverage and recovery rate, suppressed transmission, thereby highlighting key factors for targeted intervention. Additionally, numerical simulations were conducted by varying the contact rate ((\beta)), representing interactions between susceptible and infected individuals. The simulations demonstrated that reducing contacts between susceptible and infected populations, combined with increasing the rate of treatment, significantly decreased the prevalence and overall burden of Hepatitis B. These findings underscore the critical role of treatment strategies and behavioral interventions, such as reducing exposure risk, in controlling Hepatitis B transmission in human populations. To reduce the burden of Hepatitis B, it was recommended that expanding vaccination coverage with timely administration of both doses, minimizing contact rates through public awareness and safe practices, and strengthening healthcare services for acute and chronic cases, including timely diagnosis and effective treatment. For future research, incorporating time-dependent optimal control strategies, developing age-structured models, and applying fractional-order approaches could provide deeper insights into transmission dynamics and enhance strategies for controlling the disease.

Conflict of Interest

The authors declare there is no conflict of interest regarding publication of this article

REFERENCE

Adepoju, V. A., Abdulrahim, A., Udah, D. C., Jamil, S., Olaniyi, B. O., Gulumbe, B. H., Adebisi, Y. A., & Adnani, Q. E. S. (2025). *Prevalence and risk factors of hepatitis B and C among prison inmates in Nigeria: A systematic review and meta-analysis.* **BMC Public Health, 25**, Article 25614. https://doi.org/10.1186/s12889-025-25614-8

Agbata, B. C., Asante-Mensa, F., Abah, E., Kwabi, P. A., Amoah-Mensah, J., Shior, M. M., Meseda, P. K., Topman, N. N., & Obeng-Denteh, W. (2025). *Semi-analytical approach to a fractional-order model for the dynamics and control of typhoid fever*. *Journal of Basic and Applied Sciences Research, 3*(3), 215–226. https://doi.org/10.4314/jobasr.v3i3.23

Agbata, B. C., Dervishi, R., Asante-Mensa, F., Kwabi, P. A., Odeh, J. O., Amoah-Mensah, J., Meseda, P. K., & Obeng-Denteh, W. (2025). *Mathematical modelling of measles disease with double dose vaccination*. *Journal of Basic and Applied Sciences Research, 3*(3), 199–214. https://doi.org/10.4314/jobasr.v3i3.22

Akabuike, O. M., Aworh, M. K., Uzoebo, N. L., et al. (2024). *Evaluating hepatitis B screening, prevalence, vaccination coverage, and linkage to care in Abuja, Nigeria: Insights from a cross-sectional study. BMC Public Health, 24*, Article 3475. https://doi.org/10.1186/s12889-024-21017-3

Al-Busafi, S. A., & Alwassief, A. (2024). *Global perspectives on the hepatitis B vaccination: Challenges, achievements, and the road to elimination by 2030. Vaccines, 12*(3), 288. https://doi.org/10.3390/vaccines12030288

Atnaw, M., Abonyo, O., & Theuri, D. (2023). *Mathematical model of hepatitis B disease with optimal control and cost-effectiveness analysis*. *Computational and Mathematical Methods in Medicine, 2023*, Article 5215494. https://doi.org/10.1155/2023/5215494

Egbe, K. A., Ike, A. C., Akanbi, O. A., et al. (2025). *Molecular epidemiology of hepatitis B virus from blood samples obtained from persons in Enugu and Nasarawa States, Nigeria. BMC Infectious Diseases, 25*, Article 772. (Springer)

El-Shenawy, A., El-Gamel, M., & Abouelsaid, M. (2025). *Numerical simulation of a nonlinear hepatitis B virus mathematical model using the Dickson collocation*

technique. *Scientific Reports, 15*, 44651. https://doi.org/10.1038/s41598-025-31826-0 (Nature)

Koray, M. H., Adomah-Afari, A., Xu, D. R., et al. (2025). *Developing strategies for integrating Hepatitis B virus infection testing and treatment into routine PMTCT services in Ghana's Upper West Region: A rapid qualitative study*. *BMC Public Health, 25*, Article 3090. https://doi.org/10.1186/s12889-025-24285-9

Mirgichan, J. K., Ngari, C. G., Karanja, S., & Muriungi, R. (2025). *Mathematical modeling and simulation of hepatitis B transmission dynamics with passive immunity and control strategies*. *Heliyon*, *11*(2), e41744. https://doi.org/10.1016/j.heliyon.2025.e41744 (PubMed)

Rajbhandari, R., Nguyen, V. H., Knoble, A., Fricker, G., & Chung, R. T. (2025). *Advances in the management of hepatitis B. BMJ, 389*, e079579. https://doi.org/10.1136/bmj-2024-079579 (BMJ)

Shepard, C. W., Simard, E. P., Finelli, L., Fiore, A. E., & Bell, B. P. (2006). *Hepatitis B virus infection: Epidemiology and vaccination. Epidemiologic Reviews, 28*(1), 112–125. https://doi.org/10.1093/epirev/mxl008

Turab, A., Shafqat, R., Muhammad, S., Shuaib, M., & Khan, M. F. (2024). *Predictive modeling of hepatitis B viral dynamics: A Caputo derivative-based approach using artificial neural networks*. *Scientific Reports, 14*, 21853. https://doi.org/10.1038/s41598-024-70788-7 (Nature)

Tuuryare, S. A. H., Timothy, K. K., & Mohamud, M. H. (2025). *Hepatitis B vaccination coverage and associated factors among adults in Somalia: A cross-sectional study*. *BMC Public Health, 25*, Article 3917-0. https://doi.org/10.1186/s12889-025-25171-0 (SpringerLink)

Wondmeneh, T. G., & Mekonnen, A. T. (2024). *Epidemiology of hepatitis B virus infection among pregnant women in Africa: A systematic review and meta-analysis. BMC Infectious Diseases, 24*, Article 921. World Health Organization. (2025). *Hepatitis B fact sheet.* https://www.who.int/news-room/fact-sheets/detail/hepatitis-b (MDPI)

Zarin, R., Wannasingha Humphries, U., & Saleewong, T. (2024). *Advanced mathematical modeling of hepatitis B transmission dynamics with and without diffusion effect using real data from Thailand*. *European Physical Journal Plus, 139*, 385. https://doi.org/10.1140/epjp/s13360-024-05154-7 (epjplus.epj.org)

Zou, L., Zhang, W., & Ruan, S. (2010). *Modeling the transmission dynamics and control of hepatitis B virus in China*. *Journal of Theoretical Biology, 262*(2), 330–338. https://doi.org/10.1016/j.jtbi.2009.09.026