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Mathematical Analysis of an Occult Hepatitis B Transmission Model Incorporating Vertical Transmission and Vaccination

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Abstract

Hepatitis B virus (HBV) infection remains a major public health problem due to its potential to cause chronic liver disease and its continued transmission through infected blood and body fluids, particularly from infected mothers to their newborns. Despite the availability of effective vaccines, incomplete vaccination, vertical transmission, and occult Hepatitis B infection continue to contribute to the persistence of the disease. This study develops and analyzes a deterministic mathematical model for the transmission dynamics of HBV incorporating vaccination, vertical transmission, and occult infection. The model extends existing HBV models by accounting for occult infection as a hidden source of disease persistence and by representing vertical transmission in relation to the proportion of chronic carriers in the population. The basic reproduction number was derived using the next-generation matrix method, while the disease-free and endemic equilibrium points and the local stability of the disease-free equilibrium were investi-

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gated. The results show that the disease-free equilibrium is locally asymptotically stable when the basic reproduction number is less than one and unstable when it is greater than one. Using the baseline parameter values, the analysis yielded $R_0 = 2.6026$, indicating that HBV can persist under the baseline conditions. Numerical simulations over a 10-year period showed that increased vaccination coverage and improved recovery reduce infection, while vertical transmission and occult infection contribute to disease persistence. Reducing vertical transmission alone was not sufficient to bring R_0 below unity, whereas the combined intervention produced the greatest reduction in the infected populations and reduced R_0 well below one. These findings highlight the importance of an integrated HBV control strategy involving sustained vaccination, prevention of mother-to-child transmission, reduction of effective transmission, improved management of infected individuals, and consideration of occult infection.

1 Introduction

Hepatitis is a disease characterized by inflammation of the liver and may result from viral infections, excessive alcohol consumption, autoimmune disorders, or exposure to toxic substances. Viral hepatitis is the most common form and remains a major public health concern because of its widespread occurrence and serious health consequences. The major types of viral hepatitis are Hepatitis A, B, C, D, and E, which differ in their modes of transmission and disease progression (WHO, 2024). While Hepatitis A and E are mainly transmitted through contaminated food and water, Hepatitis B, C, and D are primarily spread through infected blood and body fluids. Among these viruses, Hepatitis B virus (HBV) remains one of the leading causes of chronic liver disease worldwide. HBV is a partially double-stranded DNA virus belonging to the *Hepadnaviridae* family and primarily infects liver cells. It can cause both acute and chronic infections, with chronic infection increasing the risk of liver cirrhosis, liver failure, and hepatocellular carcinoma (Schweitzer et al., 2015). According to the World Health Organization, about 296 million people were living with chronic Hepatitis B infection in 2019, with approximately 1.5 million new infections occurring each year (WHO, 2024). The burden of the disease is particularly high in sub-Saharan Africa and East Asia, where the prevalence remains above 5% in many communities (Schweitzer et al., 2015; Polaris Observatory Collaborators, 2018). HBV is transmitted through contact with infected blood and body fluids, including mother-to-child transmission during pregnancy, delivery, or shortly after birth. This route of transmission is particularly important because infants infected at birth have a high risk of developing chronic infection, creating a long-term reservoir that sustains disease transmission (Hyun et al., 2017). Vaccination remains the most effective preventive measure against HBV, especially when the birth-dose vaccine is administered promptly. However, incomplete vaccination coverage and delayed vaccination continue to limit disease control in many developing countries (WHO, 2024).

Another important challenge in HBV control is occult Hepatitis B infection (OBI), a hid-

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den form of infection in which HBV DNA persists in the liver and may also be detected in the blood despite the absence of detectable hepatitis B surface antigen (HBsAg). This makes OBI particularly concerning because infected individuals may test negative during routine HBsAg screening while still harbouring the virus. Such undetected infections may contribute to HBV transmission, especially through blood transfusion and organ transplantation, and may reactivate when the immune system becomes suppressed (Makvandi, 2016; Obeagu et al., 2017; Rai et al., 2022). Therefore, OBI represents an important hidden reservoir that complicates the detection, control, and possible elimination of HBV. This gap motivates the development of a mathematical model that incorporates occult infection alongside vaccination and vertical transmission to provide a more realistic description of HBV dynamics. Motivated by this gap, this study develops and analyzes a deterministic mathematical model for the transmission dynamics of Hepatitis B that incorporates vaccination, vertical transmission, and occult infection. The model provides a more realistic representation of HBV transmission by accounting for the hidden reservoir created by occult infection. The basic reproduction number is derived using the next-generation matrix approach, the equilibrium states and their stability are investigated, and numerical simulations are performed to examine the effects of vaccination, vertical transmission, and occult infection on the spread of Hepatitis B.

2 Model Formulation

The total population at time t, denoted by $N(t)$, is divided into seven epidemiological compartments: susceptible individuals $S(t)$, vaccinated individuals $V(t)$, exposed individuals $E(t)$, acutely infected individuals $I(t)$, chronic carriers $C(t)$, occult infected individuals $O(t)$, and recovered individuals $R(t)$. Thus, the total population is given by

$$N(t) = S(t) + V(t) + E(t) + I(t) + C(t) + O(t) + R(t). \tag{1}$$

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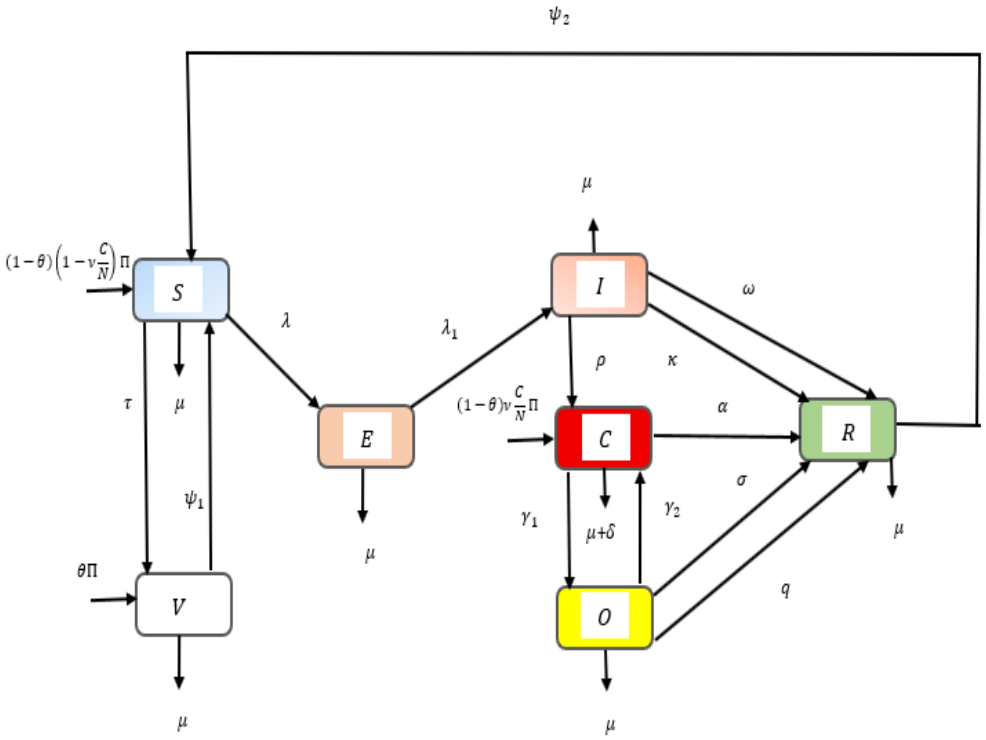

Figure 1: Epidemiological diagram of Model

The transmission dynamics of the model are governed by the parameters presented in Table 1. These parameters describe recruitment, vaccination, horizontal and vertical transmission, disease progression, recovery, loss of immunity, and mortality within the population.

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Table 1. Description of Model Parameters

Parameter	Description	Parameter	Description
Π	Recruitment rate into the population	ρ	Progression rate from acute infection to chronic carrier state
θ	Proportion of recruited individuals vaccinated at birth	κ	Recovery rate of acutely infected individuals
ν	Vertical transmission parameter from infected mothers to newborns	ω	Additional removal rate from the acutely infected class
β	Effective transmission rate of HBV	α	Recovery rate of chronic carriers
ϵ	Modification parameter for infectiousness of acutely infected individuals	γ_1	Progression rate from chronic carrier to occult infection
η	Modification parameter for infectiousness of occult infected individuals	γ_2	Reversion rate from occult infection to chronic carrier state
τ	Vaccination rate of susceptible individuals	q	Recovery rate of occult infected individuals
ψ_1	Rate of loss of vaccine-induced immunity	δ	Disease-induced death rate of chronic carriers
ψ_2	Rate of loss of immunity among recovered individuals	σ	Disease-related removal rate from the occult infected class
λ_1	Progression rate from exposed to acute infection	μ	Natural death rate in all compartments
λ	Force of infection of HBV		

The interactions described above define the flow of individuals between compartments and form the basis for the system of nonlinear differential equations that govern the dynamics of the model.

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The model consists of the following system of nonlinear ordinary differential equations:

$$\left.\begin{aligned}
\frac{dS}{dt} &= (1-\theta)\left(1-\nu\frac{C}{N}\right)\Pi + \psi_1 V + \psi_2 R - (\lambda+\tau+\mu)S,\\
\frac{dV}{dt} &= \theta\Pi + \tau S - (\psi_1+\mu)V,\\
\frac{dE}{dt} &= \lambda S - (\lambda_1+\mu)E,\\
\frac{dI}{dt} &= \lambda_1 E - (\rho+\kappa+\omega+\mu)I,\\
\frac{dC}{dt} &= (1-\theta)\nu\frac{C}{N}\Pi + \rho I + \gamma_2 O - (\alpha+\gamma_1+\delta+\mu)C,\\
\frac{dO}{dt} &= \gamma_1 C - (q+\gamma_2+\sigma+\mu)O,\\
\frac{dR}{dt} &= \kappa I + \alpha C + qO - (\psi_2+\mu)R.
\end{aligned}\right\} \tag{2}$$

The force of infection is given by

$$\lambda = \beta\left(\frac{\epsilon I + C + \eta O}{N}\right). \tag{3}$$

With initial conditions $S(0) \geq 0,\ V(0) \geq 0,\ E(0) \geq 0,\ I(0) \geq 0,\ C(0) \geq 0,\ O(0) \geq 0,\ R(0) \geq 0,\ (S(0), V(0), E(0), I(0), C(0), O(0), R(0)) \in \mathbb{R}^7_+$, where the force of infection λ is given in Equation (3).

3 Basic Properties of Model

The positivity of solution and invariant region of dynamical system (2) is handy, given the fact that, the model describes human population. Hence, the study claims the following results.

Positivity of Solutions

Because the model represents population sizes, all state variables must remain non–negative for all $t \geq 0$. This property ensures that the solutions are biologically feasible.

Theorem 3.1 (Positivity of Solutions)**.** *Suppose that*

$$(S(0), V(0), E(0), I(0), C(0), O(0), R(0)) \in \mathbb{R}^7_+.$$

Then, the solutions of system (2) remain non-negative for all $t \geq 0$.

Proof. From the first equation of system (2),

$$\frac{dS}{dt} \geq -(\lambda+\tau+\mu)S.$$

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Dividing by $S(t)$ and integrating over the interval $[0,t]$ yields

$$S(t) \geq S(0)\exp\left[-\int_0^t (\lambda+\tau+\mu)\,ds\right] \geq 0.$$

Applying the same argument to the remaining equations gives $V(t) \geq V(0)e^{-(\psi_1+\mu)t} \geq 0$, $E(t) \geq E(0)e^{-(\lambda_1+\mu)t} \geq 0$, $I(t) \geq I(0)e^{-(\rho+\kappa+\omega+\mu)t} \geq 0$, $C(t) \geq C(0)e^{-(\alpha+\gamma_1+\delta+\mu)t} \geq 0$, $O(t) \geq O(0)e^{-(q+\gamma_2+\sigma+\mu)t} \geq 0$, $R(t) \geq R(0)e^{-(\psi_2+\mu)t} \geq 0$. Hence, all state variables remain non-negative for all $t \geq 0$. Therefore, the feasible region $\mathbb{R}^7_+$ is positively invariant, and the model is mathematically and biologically well posed.

Boundedness of Solutions

The following theorem establishes that the solutions of the model remain in a biologically feasible region.

Theorem 3.2 (Invariant Region)**.** *The feasible region*

$$\Omega = \left\{(S,V,E,I,C,O,R) \in \mathbb{R}^7_+ : N(t) \leq \frac{\Pi}{\mu}\right\}$$

is positively invariant for system (2). Consequently, all solutions with non-negative initial conditions remain bounded for all $t \geq 0$.

Proof. Let

$$N(t) = S(t)+V(t)+E(t)+I(t)+C(t)+O(t)+R(t).$$

Differentiating $N(t)$ with respect to t and substituting the equations of system (2) yields

$$\frac{dN}{dt} = \Pi - \mu N - \omega I - \delta C - \sigma O. \tag{4}$$

Since $I \geq 0$, $C \geq 0$, $O \geq 0$, it follows from (4) that

$$\frac{dN}{dt} \leq \Pi - \mu N. \tag{5}$$

Consider the comparison equation

$$\frac{dy}{dt} = \Pi - \mu y,$$

whose solution is

$$y(t) = y(0)e^{-\mu t} + \frac{\Pi}{\mu}\left(1-e^{-\mu t}\right).$$

Applying the Comparison Theorem to (5) gives

$$N(t) \leq N(0)e^{-\mu t} + \frac{\Pi}{\mu}\left(1-e^{-\mu t}\right).$$

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Hence,

$$\limsup_{t\to\infty} N(t) \leq \frac{\Pi}{\mu},$$

which implies that

$$N(t) \leq \frac{\Pi}{\mu}.$$

Therefore,

$$\Omega = \left\{(S, V, E, I, C, O, R) \in \mathbb{R}^7_+ : N(t) \leq \frac{\Pi}{\mu}\right\}$$

is positively invariant. Thus, all solutions of system (2) remain bounded in the feasible region Ω.

4 The model analysis

Equilibrium Points

The equilibrium points of system (3.2) are obtained by setting

$$\frac{dS}{dt} = \frac{dV}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dC}{dt} = \frac{dO}{dt} = \frac{dR}{dt} = 0,$$

where

$$\lambda^* = \frac{\beta\left(\epsilon I^* + C^* + \eta O^*\right)}{N^*}, \qquad N^* = S^* + V^* + E^* + I^* + C^* + O^* + R^*.$$

Theorem 4.1 (Disease-Free Equilibrium)**.** *The model admits the disease-free equilibrium*

$$E_0 = \left(\frac{\Pi\left[\psi_1 + (1-\theta)\mu\right]}{\mu(\tau + \psi_1 + \mu)},\ \frac{\Pi(\tau + \theta\mu)}{\mu(\tau + \psi_1 + \mu)},\ 0,\ 0,\ 0,\ 0,\ 0,\ 0\right).$$

Proof. At the disease-free equilibrium, there is no infection in the population. Hence,

$$E^* = I^* = C^* = O^* = R^* = 0,$$

which implies that $\lambda^* = 0$. Solving the remaining equilibrium equations yields

$$S_0 = \frac{\Pi\left[\psi_1 + (1-\theta)\mu\right]}{\mu(\tau + \psi_1 + \mu)}, \qquad V_0 = \frac{\Pi(\tau + \theta\mu)}{\mu(\tau + \psi_1 + \mu)}.$$

Therefore, the disease-free equilibrium is

$$E_0 = \left(S_0, V_0, 0, 0, 0, 0, 0\right).$$

Theorem 4.2 (Endemic Equilibrium)**.** *If the disease persists in the population* ($\lambda^* > 0$)*, then the model admits the endemic equilibrium*

$$E_1 = (S^*, V^*, E^*, I^*, C^*, O^*, R^*),$$

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where

$$S^* = \frac{N^*}{\beta A\left[\epsilon + \frac{\rho}{D^*}\left(1 + \frac{\eta\gamma_1}{q+\gamma_2+\sigma+\mu}\right)\right]},$$

with

$$A = \frac{\lambda_1}{(\lambda_1+\mu)(\rho+\kappa+\omega+\mu)},$$

and

$$D^* = (\alpha+\gamma_1+\delta+\mu) - \frac{(1-\theta)\nu\Pi}{N^*} - \frac{\gamma_1\gamma_2}{q+\gamma_2+\sigma+\mu}.$$

The remaining equilibrium components are determined successively from S^* as

$$V^* = \frac{\theta\Pi + \tau S^*}{\psi_1+\mu},$$

$$E^* = \frac{\lambda^* S^*}{\lambda_1+\mu},$$

$$I^* = \frac{\lambda_1\lambda^* S^*}{(\lambda_1+\mu)(\rho+\kappa+\omega+\mu)},$$

$$C^* = \frac{\rho I^*}{D^*},$$

$$O^* = \frac{\gamma_1 C^*}{q+\gamma_2+\sigma+\mu},$$

and

$$R^* = \frac{\kappa I^* + \alpha C^* + qO^*}{\psi_2+\mu}.$$

Here, N^* is determined from the equilibrium population relation

$$N^* = S^* + V^* + E^* + I^* + C^* + O^* + R^*,$$

together with

$$\lambda^* = \beta\left(\frac{\epsilon I^* + C^* + \eta O^*}{N^*}\right).$$

Proof. For the endemic equilibrium, $\lambda^* > 0$. Setting the right-hand sides of the model equations equal to zero gives

$$V^* = \frac{\theta\Pi + \tau S^*}{\psi_1+\mu},$$

$$E^* = \frac{\lambda^* S^*}{\lambda_1+\mu},$$

and

$$I^* = \frac{\lambda_1\lambda^* S^*}{(\lambda_1+\mu)(\rho+\kappa+\omega+\mu)}.$$

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From the occult infected equilibrium equation,

$$O^* = \frac{\gamma_1 C^*}{q + \gamma_2 + \sigma + \mu}.$$

Substituting this expression into the chronic carrier equilibrium equation gives

$$C^* = \frac{\rho I^*}{(\alpha + \gamma_1 + \delta + \mu) - \dfrac{(1-\theta)\nu\Pi}{N^*} - \dfrac{\gamma_1\gamma_2}{q + \gamma_2 + \sigma + \mu}}.$$

Defining

$$D^* = (\alpha + \gamma_1 + \delta + \mu) - \frac{(1-\theta)\nu\Pi}{N^*} - \frac{\gamma_1\gamma_2}{q + \gamma_2 + \sigma + \mu},$$

we obtain

$$C^* = \frac{\rho I^*}{D^*}.$$

Also,

$$R^* = \frac{\kappa I^* + \alpha C^* + qO^*}{\psi_2 + \mu}.$$

To obtain S^*, the force of infection at the endemic equilibrium is

$$\lambda^* = \beta \left(\frac{\epsilon I^* + C^* + \eta O^*}{N^*} \right).$$

Using

$$I^* = A\lambda^* S^*, \qquad A = \frac{\lambda_1}{(\lambda_1 + \mu)(\rho + \kappa + \omega + \mu)},$$

together with

$$C^* = \frac{\rho A \lambda^* S^*}{D^*},$$

and

$$O^* = \frac{\gamma_1 \rho A \lambda^* S^*}{(q + \gamma_2 + \sigma + \mu) D^*},$$

the force of infection becomes

$$\lambda^* = \frac{\beta A \lambda^* S^*}{N^*} \left[\epsilon + \frac{\rho}{D^*} \left(1 + \frac{\eta\gamma_1}{q + \gamma_2 + \sigma + \mu} \right) \right].$$

Since $\lambda^* > 0$, dividing through by λ^* gives

$$1 = \frac{\beta A S^*}{N^*} \left[\epsilon + \frac{\rho}{D^*} \left(1 + \frac{\eta\gamma_1}{q + \gamma_2 + \sigma + \mu} \right) \right].$$

Hence,

$$S^* = \frac{N^*}{\beta A \left[\epsilon + \dfrac{\rho}{D^*} \left(1 + \dfrac{\eta\gamma_1}{q + \gamma_2 + \sigma + \mu} \right) \right]}.$$

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Therefore, once the positive endemic solution satisfying the force of infection and total population relations is determined, S^* is obtained from the expression above, and the remaining equilibrium components are determined successively from S^*. Hence, the endemic equilibrium is

$$E_1 = (S^*, V^*, E^*, I^*, C^*, O^*, R^*).$$

Basic Reproduction Number

The basic reproduction number of the model is obtained using the next-generation matrix method of Diekmann et al. (1990).

Theorem 4.3 (Basic Reproduction Number)**.** *The basic reproduction number of system (3.2) is given by*

$$R_0 = \rho(FV^{-1}),$$

where $\rho(\cdot)$ denotes the spectral radius of the next-generation matrix.

Proof. The infected compartments are

$$X = (E, I, C, O)^T,$$

and the infected subsystem is written as

$$\frac{dX}{dt} = \mathcal{F}(X) - \mathcal{V}(X).$$

Evaluating the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ at the disease-free equilibrium gives

$$F = \begin{pmatrix} 0 & \frac{\beta\epsilon S_0}{N_0} & \frac{\beta S_0}{N_0} & \frac{\beta\eta S_0}{N_0} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{(1-\theta)\nu\Pi}{N_0} & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} \lambda_1 + \mu & 0 & 0 & 0 \\ -\lambda_1 & \rho + \kappa + \omega + \mu & 0 & 0 \\ 0 & -\rho & \alpha + \gamma_1 + \delta + \mu & -\gamma_2 \\ 0 & 0 & -\gamma_1 & q + \gamma_2 + \sigma + \mu \end{pmatrix}.$$

At the disease-free equilibrium,

$$N_0 = S_0 + V_0 = \frac{\Pi}{\mu}.$$

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Hence,

$$\frac{(1-\theta)\nu\Pi}{N_0} = (1-\theta)\nu\mu.$$

The next-generation matrix is

$$K = FV^{-1}.$$

Hence, the basic reproduction number is obtained as the dominant eigenvalue of K, namely

$$R_0 = \frac{1}{2}\left[k_{11} + k_{33} + \sqrt{(k_{11}-k_{33})^2 + 4k_{13}k_{31}}\right],$$

where

$$k_{11} = \frac{\beta S_0 \lambda_1}{N_0(\lambda_1+\mu)(\rho+\kappa+\omega+\mu)}\left[\epsilon + \frac{\rho\{q+\gamma_2+\sigma+\mu+\eta\gamma_1\}}{\Delta}\right],$$

$$k_{13} = \frac{\beta S_0\{q+\gamma_2+\sigma+\mu+\eta\gamma_1\}}{N_0\Delta},$$

$$k_{31} = \frac{(1-\theta)\nu\Pi\rho\lambda_1(q+\gamma_2+\sigma+\mu)}{N_0(\lambda_1+\mu)(\rho+\kappa+\omega+\mu)\Delta},$$

$$k_{33} = \frac{(1-\theta)\nu\Pi(q+\gamma_2+\sigma+\mu)}{N_0\Delta},$$

with

$$\Delta = (\alpha+\gamma_1+\delta+\mu)(q+\gamma_2+\sigma+\mu) - \gamma_1\gamma_2.$$

Since

$$N_0 = \frac{\Pi}{\mu},$$

the vertical-transmission components may equivalently be written as

$$k_{31} = \frac{(1-\theta)\nu\mu\rho\lambda_1(q+\gamma_2+\sigma+\mu)}{(\lambda_1+\mu)(\rho+\kappa+\omega+\mu)\Delta},$$

and

$$k_{33} = \frac{(1-\theta)\nu\mu(q+\gamma_2+\sigma+\mu)}{\Delta}.$$

Therefore,

$$R_0 = \frac{1}{2}\left[k_{11} + k_{33} + \sqrt{(k_{11}-k_{33})^2 + 4k_{13}k_{31}}\right].$$

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Thus, R_0 represents the average number of secondary infections generated by a typical infectious individual introduced into a population at the disease-free equilibrium.

Local Stability of the Disease-Free Equilibrium

The local stability of the disease-free equilibrium is established by linearizing the model around the disease-free state and examining the eigenvalues of the corresponding Jacobian matrix.

For simplicity, let

$$\begin{aligned} &a_1 = \tau + \mu, && a_2 = \psi_1 + \mu, && a_3 = \lambda_1 + \mu, \\ &a_4 = \rho + \kappa + \omega + \mu, && a_5 = \alpha + \gamma_1 + \delta + \mu, && a_6 = q + \gamma_2 + \sigma + \mu, \\ &a_7 = \psi_2 + \mu. \end{aligned}$$

Also, define

$$b = \frac{\beta S_0}{N_0}, \qquad h = \frac{(1-\theta)\nu\Pi}{N_0}.$$

Theorem 4.4. *(Castillo-Chavez et al., 2002)*

The disease-free equilibrium

$$E_0 = \left(\frac{\Pi\left[\psi_1 + (1-\theta)\mu\right]}{\mu(\tau + \psi_1 + \mu)}, \frac{\Pi(\tau + \theta\mu)}{\mu(\tau + \psi_1 + \mu)}, 0, 0, 0, 0, 0 \right)$$

is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Proof.

The Jacobian matrix of the system evaluated at the disease-free equilibrium E_0 is

$$J(E_0) = \begin{pmatrix} -a_1 & \psi_1 & 0 & -\epsilon b & -(b+h) & -\eta b & \psi_2 \\ \tau & -a_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -a_3 & \epsilon b & b & \eta b & 0 \\ 0 & 0 & \lambda_1 & -a_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & \rho & h - a_5 & \gamma_2 & 0 \\ 0 & 0 & 0 & 0 & \gamma_1 & -a_6 & 0 \\ 0 & 0 & 0 & \kappa & \alpha & q & -a_7 \end{pmatrix},$$

where

$$S_0 = \frac{\Pi\left[\psi_1 + (1-\theta)\mu\right]}{\mu(\tau + \psi_1 + \mu)},$$

and

$$N_0 = S_0 + V_0 = \frac{\Pi}{\mu}.$$

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Thus,

$$b = \frac{\beta S_0}{N_0}, \qquad h = \frac{(1-\theta)\nu\Pi}{N_0} = (1-\theta)\nu\mu.$$

The characteristic equation of the full Jacobian can be separated into the demographic part and the infected subsystem. The eigenvalue corresponding to the recovered class is

$$\xi_1 = -a_7 = -(\psi_2 + \mu) < 0.$$

The eigenvalues associated with the susceptible and vaccinated classes are obtained from

$$\begin{vmatrix} \xi + a_1 & -\psi_1 \\ -\tau & \xi + a_2 \end{vmatrix} = 0.$$

Hence,

$$(\xi + a_1)(\xi + a_2) - \tau\psi_1 = 0,$$

or equivalently,

$$\xi^2 + (a_1 + a_2)\xi + (a_1 a_2 - \tau\psi_1) = 0.$$

Therefore,

$$\xi_{2,3} = \frac{-(a_1 + a_2) \pm \sqrt{(a_1 + a_2)^2 - 4(a_1 a_2 - \tau\psi_1)}}{2}.$$

Since

$$a_1 + a_2 = \tau + \psi_1 + 2\mu > 0,$$

and

$$a_1 a_2 - \tau\psi_1 = (\tau + \mu)(\psi_1 + \mu) - \tau\psi_1 = \mu(\tau + \psi_1 + \mu) > 0,$$

it follows that

$$\mathrm{Re}(\xi_2) < 0, \qquad \mathrm{Re}(\xi_3) < 0.$$

The remaining four eigenvalues are obtained from the infected subsystem

$$J_I = \begin{pmatrix} -a_3 & \epsilon b & b & \eta b \\ \lambda_1 & -a_4 & 0 & 0 \\ 0 & \rho & h - a_5 & \gamma_2 \\ 0 & 0 & \gamma_1 & -a_6 \end{pmatrix}.$$

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The characteristic equation of J_I is

$$\det(\xi I - J_I) = 0.$$

Let

$$D(\xi) = (\xi + a_5 - h)(\xi + a_6) - \gamma_1\gamma_2.$$

Then,

$$\begin{aligned} P(\xi) = (\xi + a_3)(\xi + a_4)D(\xi) - \epsilon b\lambda_1 D(\xi) \\ - b\lambda_1\rho(\xi + a_6) - \eta b\lambda_1\rho\gamma_1 = 0. \end{aligned}$$

This gives the four eigenvalues

$$\xi_4, \xi_5, \xi_6, \xi_7$$

of the infected subsystem.

The infected subsystem can also be written as

$$J_I = F - V,$$

where F and V are the matrices used in the computation of the basic reproduction number. By the theorem of van den Driessche and Watmough (2002), the matrix $F - V$ is stable if and only if

$$\rho(FV^{-1}) < 1.$$

Since

$$R_0 = \rho(FV^{-1}),$$

it follows that when

$$R_0 < 1,$$

the four eigenvalues of the infected subsystem satisfy

$$\mathrm{Re}(\xi_i) < 0, \qquad i = 4, 5, 6, 7.$$

Combining this result with

$$\xi_1 < 0, \qquad \mathrm{Re}(\xi_2) < 0, \qquad \mathrm{Re}(\xi_3) < 0,$$

all the eigenvalues of $J(E_0)$ have negative real parts whenever $R_0 < 1$. Hence, the disease-free equilibrium is locally asymptotically stable.

Conversely, if

$$R_0 > 1,$$

the matrix $F - V$ has at least one eigenvalue with positive real part. Therefore, the disease-free equilibrium becomes unstable.

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Hence, the disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

5 Results

5.1 Numerical Simulation

The numerical simulation of the model was carried out to show the behaviour of the different population classes over time. The system of equations was solved via MATLAB using the model equation. The parameter values presented in Table 3 and the initial conditions in Table 2 were used for the simulation. All transition and removal rates are expressed per year, and the simulations were carried out over a period of 10 years in order to observe the changes in the susceptible, vaccinated, exposed, acutely infected, chronic carrier, occult infected, and recovered populations.

The initial conditions were retained from the original simulation. The total initial population is

$$N(0) = 800 + 300 + 120 + 75 + 50 + 25 + 100 = 1470.$$

The acutely infected and chronic carrier populations together constitute

$$\frac{75 + 50}{1470} \times 100 = 8.50\%$$

of the initial population. This value is close to published estimates of Hepatitis B prevalence in Nigeria and therefore provides a reasonable epidemiological basis for the initial disease burden used in the simulation.

Table 2: Initial conditions used for numerical simulations

Variable	**Initial Value**	**Description**
$S(0)$	800	Initial susceptible population
$V(0)$	300	Initial vaccinated population
$E(0)$	120	Initial exposed population
$I(0)$	75	Initial acutely infected population
$C(0)$	50	Initial chronic carrier population
$O(0)$	25	Initial occult infected population
$R(0)$	100	Initial recovered population

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Table 3: Parameter values used for numerical simulations

Parameter Set I				Parameter Set II			
Par.	**Value**	**Description**	**Source**	**Par.**	**Value**	**Description**	**Source**
Π	20.58	Recruitment rate (year^{-1})	Calibrated as $\mu N(0)$	ρ	0.15	Acute-to-chronic progression (year^{-1})	Wodajo et al. (2023)
β	15.00	Effective transmission rate (year^{-1})	Calibrated to endemic baseline	κ	3.50	Acute recovery rate (year^{-1})	CDC (2025)
θ	0.17	Birth vaccination proportion	WHO	ω	0.02	Additional acute removal rate (year^{-1})	Assumed
ν	0.065	Vertical transmission probability	Riches et al. (2025)	α	0.015	Chronic clearance rate (year^{-1})	Clinical evidence
ϵ	0.40	Acute infectivity modifier	Assumed	γ_1	0.08	Chronic-to-occult progression (year^{-1})	Goyal and Chauhan (2018)
η	0.10	Occult infectivity modifier	Conservative assumption	γ_2	0.04	Occult-to-chronic reversion (year^{-1})	Goyal and Chauhan (2018)
τ	0.05	Susceptible vaccination rate (year^{-1})	Calibrated	q	0.12	Occult recovery rate (year^{-1})	Goyal and Chauhan (2018)
ψ_1	0.02	Vaccine immunity waning (year^{-1})	Norouzirad et al. (2016)	δ	0.03	Disease-induced death rate (year^{-1})	Wodajo et al. (2023)
ψ_2	0.01	Loss of recovered immunity (year^{-1})	Wodajo et al. (2023)	σ	0.00	Additional occult removal rate (year^{-1})	Set to zero
λ_1	4.00	Exposed-to-acute progression (year^{-1})	CDC	μ	0.014	Natural death rate (year^{-1})	Wodajo et al. (2023)

The recruitment rate was adjusted to

$$\Pi = \mu N(0) = (0.014)(1470) = 20.58$$

individuals per year. This prevents an unrealistic increase in population size that would occur if the previous value $\Pi = 1000$ were combined with an initial population of only 1470. The chronic recovery rate was reduced from 0.10 to 0.015 per year to reflect the low annual spontaneous clearance of chronic Hepatitis B. The additional occult removal parameter was set to $\sigma = 0$, since a separate mortality rate for occult infection is not sufficiently justified biologically. The occult infectivity modifier was reduced to $\eta = 0.10$ to represent the relatively low level of viraemia generally associated with occult infection.

Using the next-generation matrix and the baseline parameter values, the basic reproduction number is

$$R_0 = 2.6026.$$

the baseline represents an endemic transmission setting in which Hepatitis B can persist in the absence of sufficiently strong intervention.

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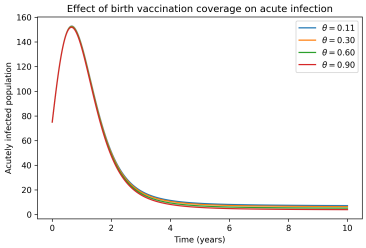
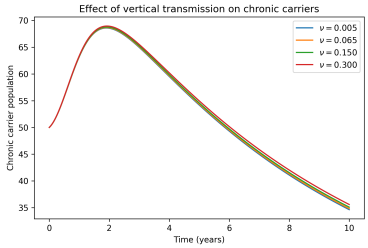
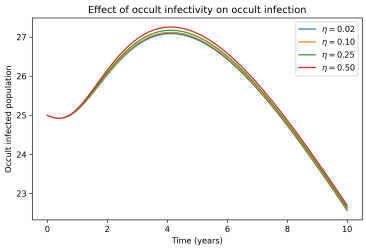


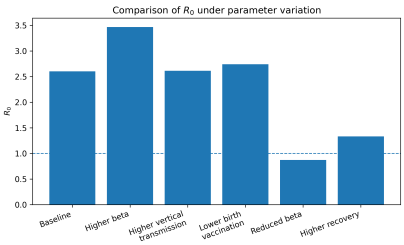
(a) Effect of vaccination coverage



(b) Effect of vertical transmission

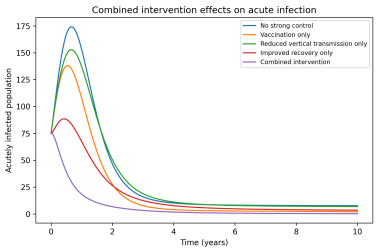


(c) Effect of occult infectivity

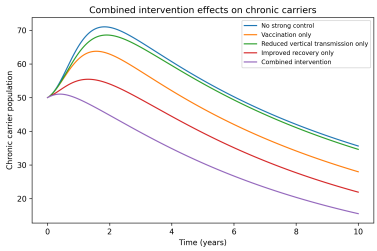


(d) Variation of R_0

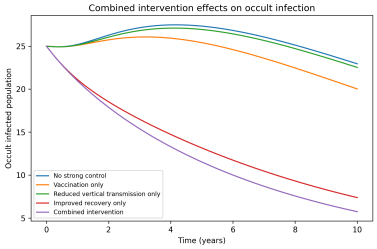
Figure 2: Effects of vaccination, vertical transmission, occult infectivity, and parameter variation on Hepatitis B transmission dynamics over a 10-year period.



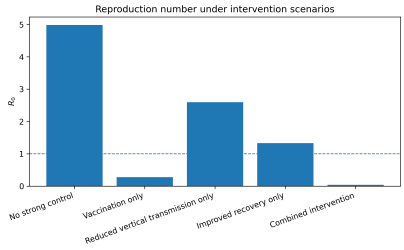
(a) Acute infection



(b) Chronic carriers



(c) Occult infection



(d) Comparison of R_0 under intervention strategies

Figure 3: Effects of individual and combined intervention strategies on Hepatitis B transmission dynamics over a 10-year period.

5.2 Discussion

Figure 2 illustrates the influence of selected epidemiological parameters on Hepatitis B transmission under the corrected model over the 10-year simulation period. As shown in Figure 2(a), increasing vaccination coverage reduces the acutely infected population. This occurs because higher vaccination coverage reduces the number of susceptible individuals available for infection and consequently limits horizontal transmission. Figure 2(b) shows that increasing the vertical transmission parameter increases the chronic carrier population. Although the vertical transmission term now depends on the proportion of chronic

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carriers, C/N, the result still confirms that mother-to-child transmission contributes to the persistence of chronic Hepatitis B in the population.

Figure 2(c) presents the effect of occult infectivity on the occult infected population. Higher values of the occult infectivity modifier produce a greater level of occult infection, indicating that hidden infections can contribute to continued HBV transmission even when their infectiousness is lower than that of the main infectious classes. Figure 2(d) shows the variation in the basic reproduction number under selected parameter changes. The corrected baseline reproduction number is $R_0 = 2.6026 > 1$, indicating that Hepatitis B can persist under the baseline epidemiological conditions. Increasing the transmission rate produces a further increase in R_0, while reduced transmission and improved recovery decrease the reproduction number. Changes in vertical transmission also influence R_0. These results show that reducing effective transmission and strengthening vaccination and recovery remain important for reducing HBV transmission.

Figure 3 further examines the effects of individual and combined intervention strategies. Figure 3(a) shows that acute infection remains higher when there is no strong control, while vaccination and improved recovery reduce the acutely infected population. The combined intervention produces the greatest and fastest reduction in acute infection over the simulation period. A similar pattern is observed in Figure 3(b), where chronic carriers persist at higher levels under weak control but decline considerably when several interventions are applied together. Figure 3(c) also shows that occult infection can be reduced through effective intervention, indicating that control strategies directed at the general transmission process can also reduce the hidden reservoir of HBV infection.

Figure 3(d) summarizes the intervention scenarios using the basic reproduction number. Under no strong control, $R_0 = 4.9832 > 1$, indicating a high possibility of sustained transmission. Vaccination alone reduces the reproduction number to $R_0 = 0.2771$, bringing transmission below the epidemic threshold. Reduction of vertical transmission alone gives $R_0 = 2.5982$, showing that controlling mother-to-child transmission by itself is not sufficient to interrupt transmission when horizontal transmission remains high. Improved recovery alone reduces the reproduction number to $R_0 = 1.3305$, but the value remains above unity. The combined intervention produces the greatest reduction, with $R_0 = 0.0377$, which is well below one.

The numerical results therefore show a more realistic distinction between the contribution of vertical transmission and other routes of HBV transmission. While prevention of mother-to-child transmission remains important, controlling vertical transmission alone may not eliminate the disease when effective contact transmission remains high. The strongest control is obtained when high vaccination coverage, reduced effective transmission, reduced vertical transmission, and improved recovery are implemented together. This confirms that an integrated intervention strategy is more effective for controlling Hepatitis B than relying on a single control measure.

6 Conclusion

A mathematical model for the transmission dynamics of Hepatitis B incorporating vaccination, vertical transmission, and occult infection was developed and analyzed. Vertical

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transmission was represented using the prevalence of chronic carriers, C/N, to provide a more biologically appropriate description of mother-to-child transmission. The model was shown to be mathematically and epidemiologically well posed, and the basic reproduction number was derived using the next-generation matrix approach. Stability analysis established that the disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$ and unstable when $R_0 > 1$.

Using the parameter values, the baseline reproduction number was obtained as $R_0 = 2.6026$, indicating that Hepatitis B can persist under the baseline conditions. Numerical simulations over a 10-year period showed that increased effective transmission promotes disease persistence, while vaccination and improved recovery reduce infection. Reducing vertical transmission alone was not sufficient to bring R_0 below unity, showing that mother-to-child transmission is important but is not the only factor sustaining HBV transmission. The simulations also showed that occult infection can contribute to the persistence of infection and should therefore be considered in Hepatitis B control strategies. Overall, the combined intervention produced the greatest reduction in the infected populations and reduced R_0 well below unity. The study therefore indicates that an integrated strategy involving increased vaccination coverage, prevention of mother-to-child transmission, reduction of effective transmission, and improved management of infected individuals provides a more effective approach for controlling Hepatitis B than relying on a single intervention.

Availability of data and material

Not Applicable.

Competing Interest

No competing interest is hereby declared by the authors.

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Authors' contributions

The development and simulation of the model, analysis, discussion of results and typesetting were implemented by all the authors and was approved for final manuscript.

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