

The Transmission Dynamics of Hepatitis B Virus Disease with Impact of Awareness and Vaccination

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ABSTRACT

Hepatitis B is a viral infection that targets the liver, caused by the Hepatitis B virus (HBV). It is one of the most common blood-borne infection, it occurs when the virus gets to the liver through the bloodstream, multiplies and releases many new viruses into the bloodstream, it inhibits the life-preserving functions of the liver, thereby increasing the risk of a serious liver disorder. Hepatitis B infection has two main stages; the acute phase and the chronic phase. In this study, we focused on these stages by developing and analyzing a new mathematical model that explains how the disease spreads within a population. We also explored different ways to control the virus, including the use of vaccines and raising awareness about infection, which can help reduce transmission and improve prevention efforts. The existence and uniqueness of the model's solution was ascertained, using the Lipchitz condition, the boundedness of the solutions and positivity of the variables and parameters of the model were established, the later by comparison theorem. Effective basic Reproduction number of the model was obtained by next generation matrix method. The local and global stabilities of the system of equations of the model were obtained. Numerical simulations of the dynamics of the model were carried out and interpreted. It was discovered that, the importance of vaccination and Awareness of the disease (through testing), help in better management of the silent killer disease (HBV).

Keywords: Hepatitis B virus, awareness, vaccination, basic reproductive number and stability.

1.0 INTRODUCTION

The word "Hepatitis" generally means inflammation of the liver (Mehta & Reddivari, 2022; Malik *et al.*, 2022), while Hepatitis B is a disease that targets the liver caused by Hepatitis B Virus (HBV). Hepatitis B occurs when the virus gets to the liver through the bloodstream, multiplies in the liver, and releases many new viruses back into the bloodstream (Zuckerman, 1996). Once this happen, the life-preserving functions of the liver are inhibited, thereby increasing the risk of developing severe liver disorder such as: liver cirrhosis, hepatic decompensation or hepatocellular carcinoma (Harkisoen *et al.*, 2012; Shi *et al.*, 2016; Tu *et al.*, 2020; Malik *et al.*, 2022; Jose-Abrego *et al.*, 2022). The two major phases of HBV infection are acute and chronic due to the inapparent nature of HBV, many infected individuals remain unaware of their HBV infection status (Meriki *et al.*, 2022). Thus, fails to seek treatment and counselling before the infection gets advanced. This may be the major reason why 68% of persons living with chronic Hepatitis B virus (HBV) infection remain undiagnosed or unaware of their infection (Krist *et al.*, 2020).

Globally, the population of individuals living with chronic HBV infection was estimated to be approximately 296 million in 2020 (Makuza *et al.*, 2022) and approximately 1.5 million people become newly infected each year (WHO, 2022). In 2016, the World Health Organization (WHO) introduced a global health sector strategy aimed at eliminating viral hepatitis as a public health threat. The plan targets a 90% reduction in new infections and a 65% reduction in hepatitis-related deaths by 2030, in alignment with the Sustainable Development Goals (SDGs) (Liu *et al.*, 2021; Makuza *et al.*, 2022). The key priorities of this strategy include reducing the transmission of HBV through the expansion of screening programs, effective resource mobilization, and the development of national strategies to support countries in prevention, diagnosis, and treatment efforts. The risk of acquiring hepatitis B was drastically reduced by high hygiene standards, an HBV public education campaign, a screening campaign (for humans and blood products) and the introduction of a prophylactic vaccine (Datfar *et al.*, 2021).

2.0 MATERIAL

Abdulrahman et al. (2013) developed and thoroughly analyzed an age-structured mathematical model describing the transmission dynamics of the Hepatitis B virus (HBV) in a population with vital dynamics, taking into account vertical transmission and sexual maturity. Their findings indicated that factors such as the birth rate, death/removal rate, probability of HBV transmission per sexual contact, and the average number of sexual contacts are highly sensitive parameters influencing the spread of HBV. The study further emphasized that vaccination, consistent condom use, and reducing the number of sexual partners are effective strategies for controlling HBV transmission. In a related study, Khan et al. (2019) proposed a mathematical model for HBV dynamics that incorporated different infection phases, as well as hospitalization, migration, and vaccination parameters. The authors examined the stability properties of the model to assess the influence of these parameters and validated their analytical results through numerical simulations, confirming the model's reliability and applicability. Similarly, Alrabaiah et al. (2020) formulated and analyzed a six-compartmental model for HBV transmission dynamics. Their analysis revealed that when the basic reproduction number (R_0) is below one, the disease-free equilibrium remains asymptotically stable, whereas for R_0 greater than one, the endemic equilibrium becomes uniformly persistent. The authors

further supported their theoretical findings with numerical simulations, demonstrating the effectiveness of each proposed control strategy in limiting HBV spread.

3.0 FORMULATION OF THE MODEL

We formulate a mathematical model to describe the spread and the control of Hepatitis B Virus (HBV) in the human population. The total population size at time t to be denoted by $N(t)$ and is subsequently divided into six subgroup corresponding to different epidemiological status: Susceptible individuals $S(t)$ and $A(t)$ acute infected individuals, Unaware ($C_u(t)$) and aware through screening ($C_s(t)$) chronic infected individuals, Recovered individuals ($R(t)$), and vaccinated individuals $V(t)$. Hence,

$$N(t) = S(t) + A(t) + C_u(t) + C_s(t) + R(t) + V(t) \tag{1}$$

Figure 1 below represents schematically the epidemiologically of Hepatitis B Virus (HBV) mathematical model. The different stages of the disease were shown by different circle and the arrows represent the way individual progress from a stage to next.

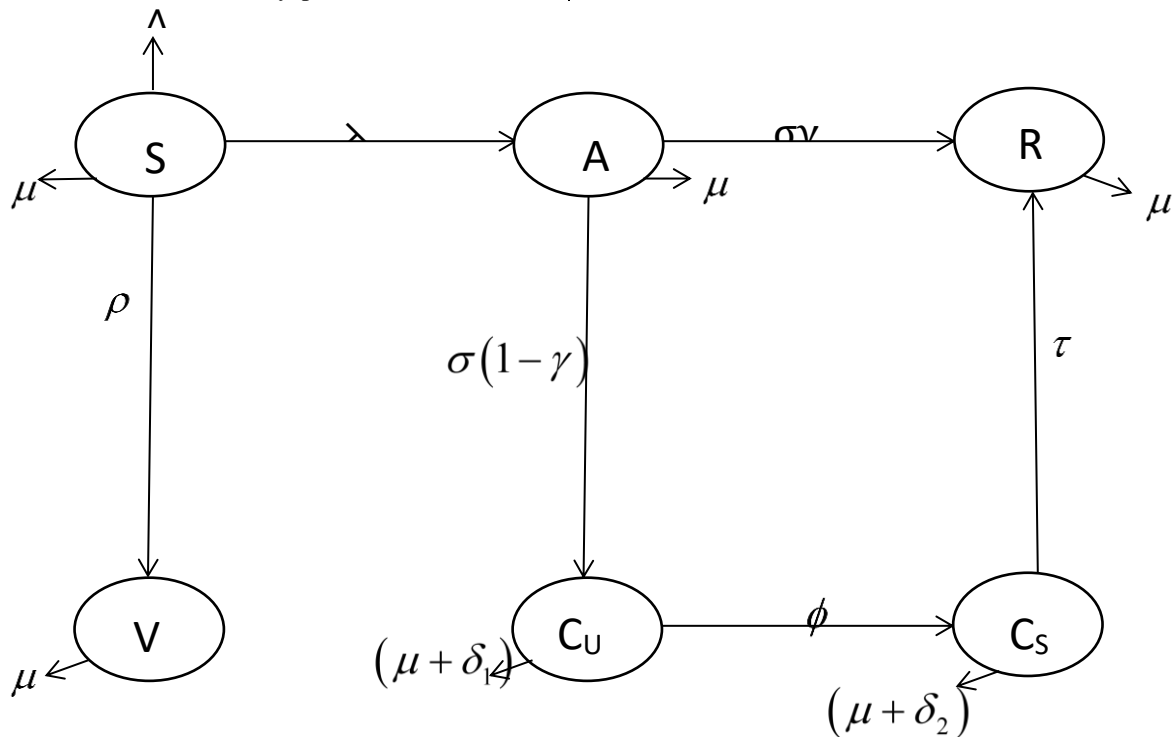


Figure 1: Schematic Flow Diagram of Hepatitis B Virus (HBV) Mathematical Model.**Model Assumptions:**

- At time t , the susceptible individuals $S(t)$ enter into population at constant recruitment rate Λ , All individuals in the population experience a natural mortality rate μ , HBV chronically infected individuals have additional death rate due to HBV infection $\delta_{1 \& 2}$, it is Assumed that C_s cannot transmit the disease since they are aware of their Hepatitis B Virus (HBV) status, and lastly the susceptible individual may acquire HBV through effective contact rate with infected individual in A and C_u , is given by β the force of infection

$$\text{associated with HBV is denoted by } \lambda = \frac{\beta(A + \eta C_U)}{N}$$

- η is Modification parameter associated with reduced infectiousness of C_u , γ Natural recovery of acute infected class after six month and if they are not able to clear the disease after six month they will progress to C_u , and τ is rate is the of treated infected individuals with full immunity.

The following system equation are formed from the above assumption.

$$\begin{aligned} \frac{ds}{dt} &= \Lambda - \lambda s - (\rho + \mu)s \\ \frac{dA}{dt} &= \lambda s - (\gamma\sigma + \mu)s \\ \frac{dC_U}{dt} &= \sigma(1 - \gamma)A - (\phi + \mu + \gamma_1)C_U \\ \frac{dC_s}{dt} &= \phi C_s - (\tau + \mu + \gamma_2)C_s \\ \frac{dR}{dt} &= \sigma\gamma A + \tau c_s - \mu R \\ \frac{dv}{dt} &= \rho s - \mu v \end{aligned} \tag{3}$$

Boundedness of Solution

Consider the region

$$D_1 = \{S, A, C_u, C_s, R, V, \in \mathbb{R}_+^7 / S > 0, A \geq 0, C_u \geq 0, C_s \geq 0, R \geq 0, V > 0, N > 0\} \tag{3.2}$$

It can be shown that the set D_1 is positively invariant and serves as a global attractor of all positive solutions of the system defined in equation (3).

Lemma 1: The region D_1 is positively invariant for the system (3)

Proof: The rate of change of the total human population is given as



$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dA}{dt} + \frac{dC_u}{dt} + \frac{dC_s}{dt} + \frac{dR}{dt} + \frac{dV}{dt}$$

$$\frac{dN}{dt} = \Lambda - \mu N - \delta_1 C_u - \delta_2 C_s$$

By standard comparison theorem,

$$\frac{dN}{dt} \leq \Lambda - \mu N$$

So we have

$$\frac{dN}{dt} + \mu N \leq \Lambda$$

Solving using the integrating factor method

$$N(t)e^{\mu t} = \int \Lambda e^{\mu t} dt + C_1$$

$$\text{at } t = 0, C_1 = N(0) - \frac{\Lambda}{\mu}$$

then

$$N(t) \leq \frac{\Lambda}{\mu} \quad \text{therefore,} \quad N(t) \leq \frac{\Lambda}{\mu}$$

Therefore, D_1 is identified as a positively invariant and attracting set, ensuring that all solution paths remain within D_1 and no trajectory crosses its boundary.

Positivity of Solution

Lemma 2: let the initial data for the model

$$\Omega = \left\{ \begin{array}{l} S, A, C_u, C_s, R, V \in \mathbb{R}_+^7 / S > 0, A \geq 0, C_u \geq 0, C_s \geq 0, R \geq 0, V \geq 0, \text{ for} \\ 0 < N \leq \frac{\Lambda}{\mu} \end{array} \right\} \quad (4)$$

Then the solution

$$S(t), A(t), C_u(t), C_s(t), R(t), V(t) \quad (5)$$

With positive initial data will remain positive for all time $t > 0$.

Proof:

$$\text{Let } t_1 = \text{Sup} \{ t > 0 : S(0) > 0, A(0) > 0, C_u(0) > 0, C_s(0) > 0, R(0) > 0, V(0) > 0 \} > 0 \quad (6)$$

$$\frac{dS}{dt} = \Lambda - (\lambda + k_1)S \quad (7)$$

To solve the ODE using the integrating factor method

$$I.F = \exp \left[\{k_1 t\} + \int_0^t \lambda(\tau) d\tau \right] \quad (8)$$

$$S(t) \exp \left[\{k_1 t\} + \int_0^t \lambda(\tau) d\tau \right] = \int \Lambda \exp \left[\{k_1 t\} + \int_0^t \lambda(\tau) d\tau \right] dt + s(0) \quad (9)$$

$$S(t) \exp \left[\left\{ k_1 t \right\} + \int_0^t \lambda(\tau) d\tau \right] = \int_0^y \exp \left[\left\{ k_1 t \right\} + \int_0^t \lambda(\tau) d\tau \right] dy + S(0) \tag{10}$$

$$S(t) = S(0) \exp \left[\left\{ -k_1 t \right\} - \int_0^t \lambda(\tau) d\tau \right] + \exp \left[\left\{ -k_1 t \right\} - \int_0^t \lambda(\tau) d\tau \right] \int_0^y \Lambda \exp \left[\left\{ k_1 t \right\} + \int_0^t \lambda(\tau) d\tau \right] dy > 0 \tag{11}$$

Similarly using the integrating factor method, we obtain

$$\frac{dA}{dt} \geq -k_2 A$$

Similarly, same goes with the remaining equation

It follows that the system equation (3) and the model to be epidemiologically well-posed.

Equilibrium point state E⁰

$$\frac{ds}{dt} = \frac{dA}{dt} = \frac{dC_u}{dt} = \frac{dC_s}{dt} = \frac{dR}{dt} = \frac{dv}{dt} = 0 \tag{12}$$

At any arbitrary equilibrium state, let

$$\{S^*, A^*, C_u^*, C_s^*, R^*, V^*, N^*, \lambda^*\} = \{S, A, C_u, C_s, R, V, N, \lambda\} \tag{13}$$

Thus, substituting into (4) we obtained the disease-free equilibrium state given by

$$(S, A, C_u, C_s, R, V) = \left(\frac{\Lambda}{k_1}, 0, 0, 0, 0, \frac{\rho \Lambda}{\mu k_1} \right) \tag{14}$$

Basic Reproductive Number

A more widely accepted and biologically meaningful method for determining the basic reproduction number is the next-generation operator approach, first described by Diekmann and Heesterbeek (2000) and later formalized by Van de Driessche and Watmough (2002). Using this technique we derived the effective basic reproduction number, R_0 for the system which is defined as the spectral radius (ρ) of the next generation matrix.

$$R_0 = \left[\frac{\partial f_i(x_0)}{\partial(x_j)} \right] \left[\frac{\partial V_i(x_0)}{\partial x_i} \right]^{-1}$$

where $F = \frac{\partial f_i(x_0)}{\partial x_j}$, and $V = \frac{\partial V_i(x_0)}{\partial x_j}$

$$F = \begin{pmatrix} \beta & \beta\eta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad \text{and} \quad V = \begin{pmatrix} k_2 & 0 & 0 \\ -\sigma(1-\gamma) & k_3 & 0 \\ 0 & -\phi & k_4 \end{pmatrix}$$

Simplifying, FV^{-1}

$$R_0 = \frac{\beta(k_3 + \eta\sigma(1-\gamma))}{k_2k_3} \tag{15}$$

Local Stability of the Disease Free-Equilibrium

Theorem 1: The Disease Free-Equilibrium (DFE) of the system equation (3) is locally asymptotically stable if $R_C < 1$ and Unstable if $R_C > 1$

Proof: We used the Jacobian stability technique to determine the local stability of the system. Consider the Jacobian matrix of equation (3) evaluated at disease free equilibrium (DFE), given by

$$J = \begin{pmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & -k_2 & 0 & 0 & 0 & 0 \\ 0 & \sigma(1-\gamma) & -k_3 & 0 & 0 & 0 \\ 0 & 0 & \phi & k_4 & 0 & 0 \\ 0 & \sigma\gamma & 0 & \tau & -\mu & 0 \\ \rho & 0 & 0 & 0 & 0 & -\mu \end{pmatrix}$$

Thus, the characteristics equation of the row transformed Jacobian matrix is given by

$$\begin{vmatrix} -k_1 - \lambda_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & -k_2 - \lambda_2 & 0 & 0 & 0 & 0 \\ 0 & \sigma(1-\gamma) & -k_3 - \lambda_3 & 0 & 0 & 0 \\ 0 & 0 & \phi & -k_4 - \lambda_4 & 0 & 0 \\ 0 & \sigma\gamma & 0 & \tau & -\mu - \lambda_5 & 0 \\ \rho & 0 & 0 & 0 & 0 & -\mu - \lambda_6 \end{vmatrix} = 0$$

And therefore, the eigenvalues are

$$\begin{aligned} \lambda_1 &= -k_1 < 0 \\ \lambda_2 &= -k_2 < 0 \\ \lambda_3 &= -k_3 < 0 \\ \lambda_4 &= -k_4 < 0 \\ \lambda_5 &= -\mu < 0 \\ \lambda_6 &= -\mu < 0 \end{aligned}$$

Since all the eigenvalues are less than unity then Hence the theorem is proved.

2.5 Global Stability

Theorem 2: The Disease Free Equilibrium (DFE) of equation (3), is globally asymptotically stable (GAS) in Ω if $R_C < 1$.

Proof: The global stability of the Disease Free Equilibrium (DFE) of mathematical model equation (3), we need to follow the approach in Castillo Chavez et al. (2002) that the conditions must be mate for $R_C < 1$. Let $X = (S, V, R)$, $Y = (A, C_u, C_s)$ and separate model equation (3) into:

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

$$F = \begin{pmatrix} \beta & \beta\eta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} k_2 & 0 & 0 \\ -\sigma(1-\gamma) & k_3 & 0 \\ 0 & -\phi & k_4 \end{pmatrix}$$

So that

$$J = \left(1 - \frac{S}{N}\right) \begin{pmatrix} \beta & \beta\eta & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

J is a non-negative matrix since $S(t) \leq N(t) \leq \frac{\Lambda}{\mu}$ in the invariant set. Hence it follows that

$$\frac{dX}{dt} \leq (F - V)X \tag{16}$$

Using the fact that all eigenvalues of the matrix $F - V$ all have negative real parts (see local stability result) it follows that the linearized differential inequality system (3.5) is stable whenever $R_0 < 1$. Consequently, $(A, C_u, C_s) \rightarrow (0, 0, 0)$ as $t \rightarrow \infty$. Thus, by the comparison theorem, $(A, C_u, C_s) \rightarrow (0, 0, 0)$ as $t \rightarrow \infty$. Substituting $A = C_u = C_s = 0$ in (3.1)-(3.6) gives $S(t) \rightarrow S^0$ as $t \rightarrow \infty$. Thus, $(S(t), A(t), C_u(t), C_s(t), V(t), R(t)) \rightarrow (S^0, 0, 0, 0, 0, V^0)$ as $t \rightarrow \infty$ for $R_0 < 1$. Thus, the systems of equation is globally asymptotically stable if $R_0 < 1$. The conclusion follows from the comparison theorem.

4. RESULTS AND DISCUSSION

The numerical simulations of the model are designed to examine the effect of vaccination, awareness creation, recovery rate, and other epidemiological factors on the transmission dynamics of the Hepatitis B Virus (HBV). The simulations validate the analytical results—particularly the conditions under which the **Disease-Free Equilibrium (DFE)** and the **Endemic Equilibrium (EE)** are stable. The discussion of each figure below connects these numerical results to the biological interpretation of

HBV transmission in human populations.

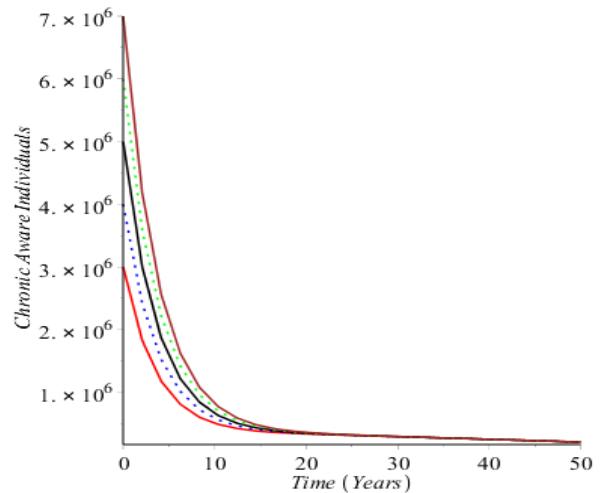


Figure 4.1: Global Asymptotic Stability of the Disease-Free Equilibrium (DFE)

This figure illustrates that when the **effective reproduction number ($Re < 1$)**, the system converges toward the DFE. Regardless of the initial number of chronically infected individuals, the infection eventually dies out over time. This validates the analytical finding that $Re < 1$ ensures local and global asymptotic stability of the DFE. Epidemiologically, this means that if public health interventions—such as vaccination and awareness—reduce the average number of secondary infections below unity, HBV transmission can be effectively interrupted and eradicated from the population.

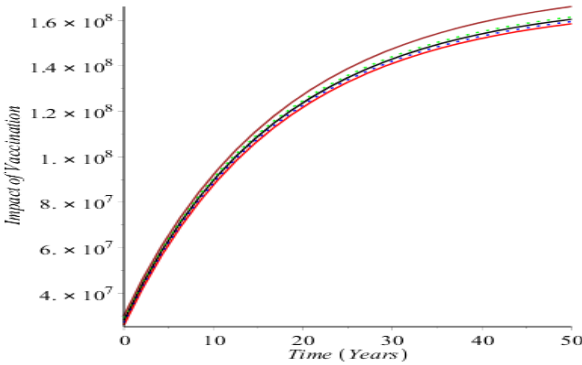


Figure 4.2: Dynamics of Chronic Unaware Individuals (C_u)

The plot shows a persistent increase in the number of chronically infected but unaware individuals. This reflects the real-world challenge where a large proportion of HBV carriers remain undiagnosed due to ignorance, stigma, or lack of testing facilities. Despite interventions, if awareness campaigns and screening programs are weak, the pool of undiagnosed infections remains high, sustaining silent transmission chains. Thus, Figure 4.2 emphasizes the importance of community-based testing and sensitization to reduce the chronic unaware population.

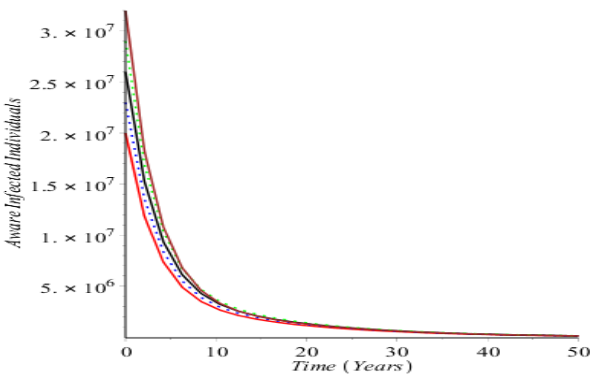


Figure 4.3: Reduction of Aware Infected Individuals (C_s)

When the effective reproduction number $Re < 1$, the population of aware infected individuals declines toward zero. Awareness programs encourage infected individuals to adopt preventive behaviors such as avoiding unprotected sex and sharing needles, which reduce secondary infections. This demonstrates that awareness campaigns are an effective non-pharmaceutical control strategy, complementing vaccination. The trend confirms that knowledge and self-management can substantially suppress HBV prevalence.

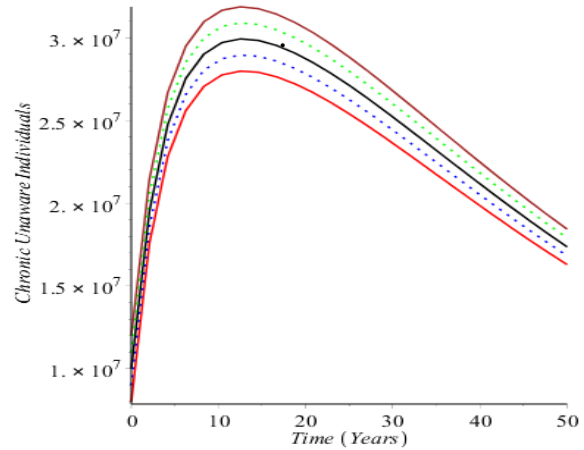


Figure 4.4: Impact of Vaccination Coverage on Disease Threat

This figure shows that increasing vaccination coverage significantly reduces HBV prevalence and increases the proportion of protected individuals. As vaccination rates rise, the susceptible class declines, leading to herd immunity that prevents further spread of the virus. This aligns with WHO recommendations for universal infant vaccination and targeted adult vaccination in high-risk groups. The numerical trend in Figure 4.4 confirms that vaccination is one of the most powerful interventions for HBV control.

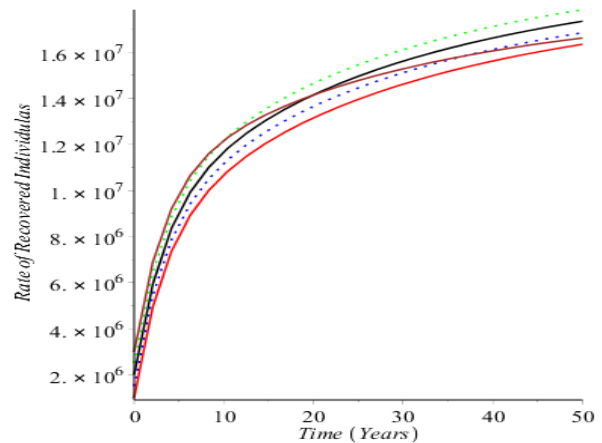


Figure 4.5: Effect of Recovery Rate on Disease Prevalence

An increase in the recovery rate (γ) of HBV-infected individuals leads to a faster decline in disease prevalence. This can result from effective medical treatment, early diagnosis, and adherence to therapy. The plot demonstrates that promoting access to antiviral medications and routine monitoring can help patients achieve viral suppression and functional cure. The numerical output corroborates theoretical expectations that improved recovery reduces the infectious pool and contributes to overall disease decline.

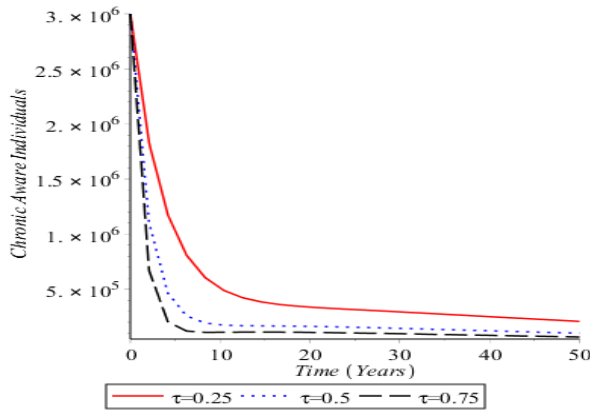


Figure 4.6: Influence of Treatment of Chronic Aware Individuals

The figure compares different treatment rates (25%, 50%, 75%) among aware chronic individuals. Higher treatment coverage drives the infection curves closer to zero, implying that increased access to therapy can lead to HBV eradication in the long term. This highlights the need for national programs to provide affordable treatment for chronic HBV carriers and integrate it into public health systems. The downward convergence of the plots confirms that therapeutic intervention is a key control pillar, alongside vaccination and awareness.

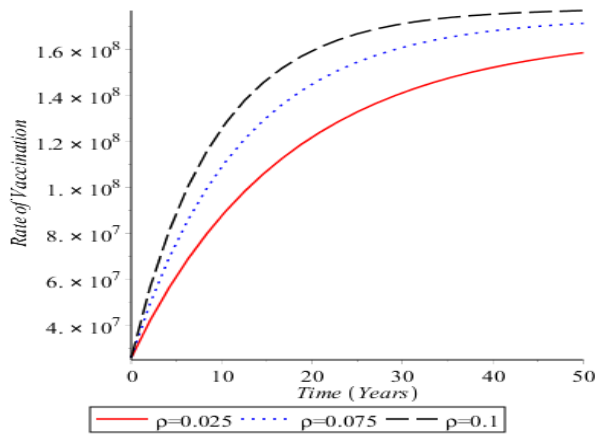


Figure 4.7: Effect of Vaccination Rate on Susceptible Population

As the rate of vaccination increases, the number of susceptible individuals moving into the vaccinated class also rises. The upward movement in the vaccinated group signifies that more individuals are protected from potential exposure. This demonstrates the preventive advantage of expanding vaccination campaigns, especially in endemic regions. The result supports the global goal of reaching at least 90% vaccination coverage among infants and high-risk populations to eliminate HBV transmission by 2030.

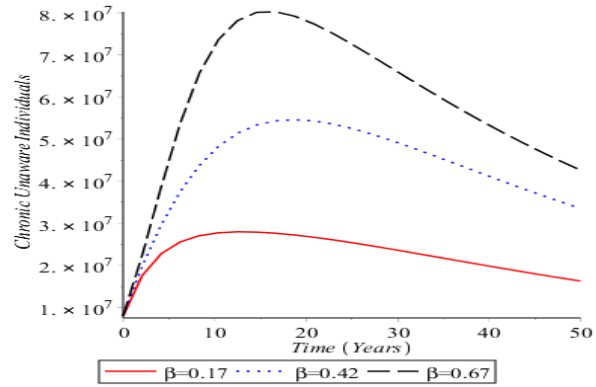


Figure 4.8: Impact of Contact Rate Between Chronic Unaware and Susceptible Individuals

The simulation reveals that increased effective contact between chronic unaware and susceptible individuals accelerates disease spread. This reinforces the epidemiological reality that unrecognized carriers drive HBV transmission in communities. Therefore, the figure underlines the critical role of targeted education, behavioral change communication, and regular screening to identify hidden infections and break transmission chains. Reducing the contact rate effectively lowers the basic reproduction number.

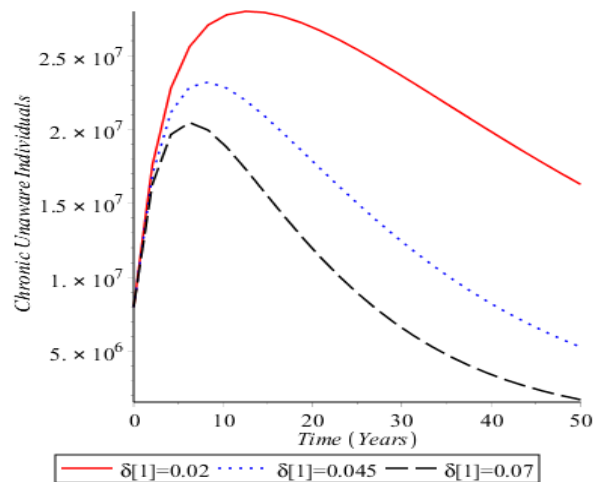


Figure 4.9: Effect of HBV-Induced Death Among Chronic Unaware Individuals

The decline in the infection curve due to HBV-induced death among chronic unaware individuals indicates that mortality reduces the number of potential transmitters. Although death is not a desirable control measure, this simulation emphasizes that the removal of infectious individuals (through death or effective treatment) reduces transmission pressure in the population. However, from a public health standpoint, prevention through vaccination and early detection is ethically superior to relying on infection-induced mortality.

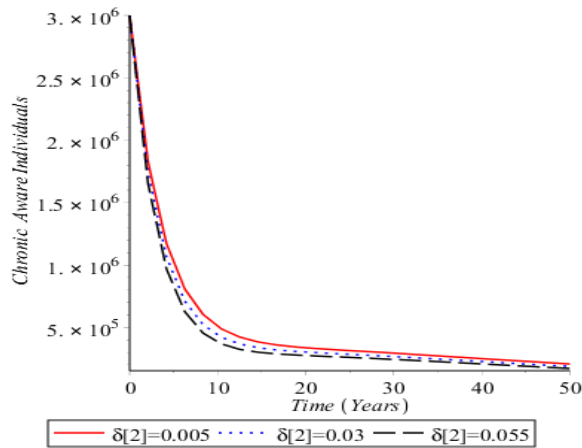


Figure 4.10: Impact of Death Among Chronic Aware Individuals

In this case, awareness and behavioral modification reduce exposure risk, while HBV-related mortality among aware individuals indirectly decreases transmission. The figure shows a gradual decline toward zero, indicating that awareness fosters responsible behavior that limits the spread. Moreover, fear of disease consequences may motivate others to seek vaccination or testing, indirectly promoting disease control. The numerical trend thus demonstrates the synergistic impact of awareness and behavioral adaptation on reducing HBV burden.

Overall Interpretation

Collectively, Figures 4.1 – 4.10 confirm the epidemiological soundness of the mathematical model. The simulations reveal that HBV transmission can be substantially curtailed when awareness creation, vaccination, and treatment programs are simultaneously implemented. The effective reproduction number remains the key threshold parameter:

- $R_e < 1$: Disease-free equilibrium, elimination possible.
- $R_e > 1$: Endemic equilibrium, sustained transmission.

The findings validate the theoretical results on the local and global stability of the equilibria and demonstrate that integrated control measures — combining vaccination, treatment, and public awareness — are essential to achieving HBV eradication goals in Nigeria and beyond.

5.0 CONCLUSION

This research work has been carried out using deterministic mathematical model for the Hepatitis B Virus (HBV) transmission dynamics in a population, the model shown control strategies that could be implemented to reduce the transmission of Hepatitis B Virus (HBV) in a society which include vaccinating the susceptible state as well as Unawareness and Awareness (testing) in the chronic state. The mathematical model has Disease Free Equilibrium (DFE) and endemic equilibrium, the basic reproductive number was obtained using next generation matrix, the local and global stability shown to be asymptotically stable. Applying all the control parameters in this model simultaneously leads to a smooth and unique way of curbing Hepatitis B Virus in a society.

REFERENCES

- Abdulrahman, S., Akinwande, N. I., Awojoyogbe, O. B., & Abubakar, U. Y. (2013). Sensitivity analysis of the parameters of a mathematical model of Hepatitis B virus transmission. *Universal Journal of Applied Mathematics*, 1(4): 230-241.
- Alarbiah, H. Safi, M.A, Dar Assi, M. H., Al-Hdaibat, B., Ullah, S., Khan, M. A, & Shah, S. A. A (2020). Optimal Control analysis of hepatitis B Virus with treatment and vaccination. *Results in physics* 1, 1-16
- Datfar, T., Doulberis, M., Papaefthymiou, A., Hines, I. N., & Manzini, G. (2021). Viral hepatitis and hepatocellular carcinoma: State of the art. *Pathogens*, 10(11): 1-19
- Harkisoen, S., Arends, J. E., van Erpecum, K. J., van den Hoek, A., & Hoepelman, A. I. (2012). Hepatitis B viral load and risk of HBV-related liver disease: from East to West. *Annals of hepatology*, 11(2): 164-171.
- Jose-Abrego, A., Rivera-Iñiguez, I., Torres-Reyes, L. A., & Roman, S. (2022). Anti-hepatitis b virus activity of food nutrients and potential mechanisms of action. *Annals of Hepatology*, 100766:1-7. <https://doi.org/10.1016/j.aohep.2022.100766>.
- Khan, T., Ahmad, S., & Zaman, G. (2019). Modeling and qualitative analysis of a hepatitis B epidemic model. *Chaos: An Interdisciplinary Journal of Nonlinear Science*, 29(10): 103-139
- Krist, A. H., Davidson, K. W., Mangione, C. M., Barry, M. J., Cabana, M., Caughey, A. B., & US Preventive Services Task Force. (2020). Screening for hepatitis B virus infection in adolescents and adults: US Preventive Services Task Force recommendation statement. *Jama*, 324(23): 2415-2422.
- Liu, J. F., Chen, T. Y., & Zhao, Y. R. (2021). Vertical transmission of hepatitis B virus: propositions and future directions. *Chinese Medical Journal*, 134(23): 2825-2831.



- Makuza, J. D., Jeong, D., Binka, M., Adu, P. A., Cua, G., Yu, A., ... & Janjua, N. Z. (2022). Impact of Hepatitis B Virus Infection, Non-alcoholic Fatty Liver Disease, and Hepatitis C Virus Co-infection on Liver-Related Death among People Tested for Hepatitis B Virus in British Columbia: Results from a Large Longitudinal Population-Based Cohort Study. *Viruses*, 14(11):1-13.
- Malik, G. F., Zakaria, N., Majeed, M. I., & Ismail, F. W. (2022). Viral hepatitis-the road traveled and the journey remaining. *Hepatic Medicine: Evidence and Research*, 14:13-26.
- Meriki, H. D., Tufon, K. A., Georges, T. D., Mokake, N. D. M., Gobina, R. M., Tony, N. J., ... & Anong, D. N. (2022). Epidemiological factors associated with HBV infection and uptake of testing in south west region of Cameroon: What can be done to scale up HBV testing in our setting? *PLOS Global Public Health*, 2(5):1-11.
- Mehta P, Reddivari AKR. Hepatitis 2022 Oct . In: StatPearls (Internet). Treasure Island (FL): StatPearls Publishing; 2023 Jan- PMID: 32119436
- Shi, Y. X., Huang, C. J., & Yang, Z. G. (2016). Impact of hepatitis B virus infection on hepatic metabolic signaling pathway. *World journal of gastroenterology*, 22(36): 8161–8167.
- Tu, T., Block, J. M., Wang, S., Cohen, C., & Douglas, M. W. (2020). The lived experience of chronic hepatitis B: a broader view of its impacts and why we need a cure. *Viruses*, 12(5):1-20.
- World Health Organization. (2022). Hepatitis B. Available online: <https://www.who.int/news-room/fact-sheets/detail/hepatitis-b> (accessed on 31 May 2022).
- Zuckerman A.J. (1996) Hepatitis Viruses. In: Baron, S, editor. *Medical Microbiology*. 4th. Edition, University of Texas Medical Branch at Galveston; Galveston TX.