Modeling the Effect of Vaccination and Treatment on the Transmission Dynamics of Hepatitis B Virus and HIV/AIDS Coinfection

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Hepatitis B and HIV/AIDS co-infections are common globally due to their similar mode of transmission. Since HIV infection modifies the course of HBV infection by increasing the rate of chronicity, prolonging HBV viremia, and increasing liver disease-associated deaths, individuals with co-infection of both diseases have a higher tendency of developing cirrhosis of the liver, higher levels of HBV DNA, reduced rate of clearance of the hepatitis B e antigen (HBeAg), and more likely to die than an individual with a single infection. This nature of HBV-HIV/AIDS co-infection motivated us to conduct this study. In this paper, we proposed and rigorously analyzed a deterministic mathematical model with the aim of investigating the effect of vaccination against hepatitis B virus and treatment for all infections on the transmission dynamics of HBV-HIV/AIDS co-infection in a population. We proved that the solutions of the sub-models and the co-infection model are positive and bounded. The models are studied qualitatively using the stability theory of differential equations, and the effective reproduction numbers of the models are derived using the next generation matrix method. Stability of the equilibria of the sub-models and the co-infection model is analyzed using Routh-Hurwitz criteria. The disease-free and endemic equilibria of the sub-models and the co-infection model are computed, and both local and global asymptotic stability conditions for those equilibria are discussed. We performed a sensitivity analysis to illustrate the influence of different parameters on the effective reproduction number of HBV-HIV/AIDS co-infection model, and we identified the most sensitive parameters are \( \omega_B \) and \( \omega_H \), which are the effective contact rates for HBV and HIV transmission, respectively. The numerical simulation of the model is done using MATLAB, and the findings from the simulations are discussed. It is observed that if the vaccination and treatment rates are increased, then the number of individuals susceptible to both infections and HBV-HIV/AIDS co-infection decreases and even falls to zero over time. Hence, it is concluded that vaccination against hepatitis B virus infection, treatment of hepatitis B and HIV/AIDS infections, and HBV-HIV/AIDS infection at the highest possible rate is very essential to control the spread of HBV-HIV/AIDS co-infection as an important public health problem.

1. Introduction

Globally, more in the developing countries, there are a number of deadly infectious diseases that are severely affecting the lifespan of the human population. Hepatitis B is one of such deadly diseases that is caused by hepatitis B virus (HBV). Currently, despite the use of a preventive vaccine for several decades as well as the use of effective and well-tolerated viral suppressive medications since 1998, approximately 250 million people are chronically infected with the virus and a lot of people die every year from hepatitis B-related liver failure and liver cancer [1, 2]. The virus is highly infectious, and the main routes of transmission are mother to neonate (vertical transmission), sexual contact with infectious partners, transplantation of organs from infected donors, exposure to improperly sterilized health care instruments, and the administration of contaminated blood products in poorer countries [1, 3]. The virus assaults liver cells and causes acute and chronic liver disease [4]. Acute hepatitis B infection is usually self-limiting; however, chronic
hepatitis B infection is life-long [5]. On the other hand, human immunodeficiency virus (HIV) is also a global public health problem affecting many people worldwide causing large morbidity and mortality [6, 7]. It is a retrovirus which weakens the body immunity by removing CD4+ T-cells, and if untreated, it continues to multiply in the host till it reaches the highest level leading to a very serious stage called AIDS [8, 9]. Everyone diagnosed with HIV should start ART regardless of his/her stage of infection or complications to lower the viral load, to improve the quality of life, and to lower the possibility of transmitting the virus to [10, 11]. HBV and HIV/AIDS co-infections are common globally due to their similar mode of transmission. Although there is a lack of accurate data for co-infection in many parts of the world, its spread is increasing in parts of West and South Africa [12]. The viruses have similar properties such as transmission, using a reverse transcriptase enzyme in replication, likelihood to develop chronic infection, and excessive chance of mutation in their genome, sometimes resulting in resistance to extensively used antiviral agents [13, 14]. Coinfection of HBV and HIV/AIDS occurs when individuals get infections concurrently or individuals who are positive for one of the strains are infected with the other strain within a brief period of time before an infection with the first strain has been established and an immune response has developed. It is common to note that HIV infection modifies the course of HBV infection by increasing the rate of chronicity, prolonging HBV viremia, and increasing liver disease-associated deaths [12]. Individuals with coinfection of both diseases have a higher tendency of developing cirrhosis of the liver, higher levels of HBV DNA, reduced rate of clearance of hepatitis B e antigen (HBeAg), and more likely to die than the ones with hepatitis B [15, 16]. Antiretroviral therapy (ART) is challenging when coinfection is present as HIV-infected individuals are usually less responsive to treatment for HBV and have a high risk of hepatotoxicity and drug interactions; however, the availability of ART with activity against HBV and HIV/AIDS coinfection, particularly tenofovir disoproxil fumarate (TDF) and tenofovir alafenamide fumarate (TAF), allows for simplification of treatment regimen and results in significant improvements in outcomes [12]. The study of infectious disease coepidemics is critical to understand how the diseases are related and how prevention and treatment efforts can be most effective. Thus, in the study of infectious diseases, mathematical models give us a fascinating insight into the very complicated infection dynamics and effective control measures. In recent years, most mathematical epidemic models are formulated based on a single disease, although a growing number of studies have considered coepidemics. Shen et al. [17] formulated a mathematical model for coronavirus infection with an optimal control analysis when vaccination is present. Using the suggested controls, they developed an optimal control model and derived mathematical results from it. Through optimal control, they obtained results showing that controls can be useful for minimizing infected individuals and improving population health. Kumar et al. [18] studied an efficient numerical scheme for a fractional model of HIV-1 infection of CD4+ T-cells with the effect of antiviral drug therapy. They solved the nonlinear mathematical models with fractional derivative by the aid of Legendre wavelet operational matrix method and noticed that the suggested schemes are more efficient and effective. Li et al. [19] developed a mathematical model to study the third wave of coronavirus infection in Pakistan through a new mathematical model and observed that the suggested parameters can decrease efficiently the infection cases of the third wave in Pakistan. Bonyah et al. [20] proposed a deterministic coinfection Zika-dengue model and analyzed the transmission of the diseases, which incorporates the prevention and treatment of the infectious nature of each disease. Their numerical optimal control analysis indicates that effective prevention and treatment of each disease will help in the effective control and eradication of the disease. They obtained results showing that the control of coinfection of dengue Zika requires that communities should combine both prevention and treatment associated with each disease at the same time. Okosun et al. [21] examined a coinfection of a cryptosporidiosis-HIV/AIDS deterministic model by incorporating time-dependent control prevention and treatments. They studied the model basic properties and presented the numerical results, which show that the best strategy to control coinfection is to combine all controls at the same time. The authors in [22] conducted a study on cholera-schistosomiasis coinfection dynamics. Based on their findings, they suggested to the public health department that the only possible cost-effective strategy for the elimination of schistosomiasis and cholera coinfection is the combination of both diseases’ preventive measures and the treatment of schistosomiasis. Mathematical models formulated to study the dynamics of HBV-HIV/AIDS coinfection are rare in the literature, although the coexistence between the two infections exists. In our review of the literature, we have got only two mathematical models of HBV-HIV/AIDS coinfection and we used them as the initial literature reviewed as follows: Nampala et al. [23] developed a mathematical model of hepatotoxicity and antiretroviral therapeutic effects in HIV/HBV coinfection. They used numerical simulations to study the therapeutic as well as toxic effect of the currently used HIV/HBV therapy and consequently derived an optimal combination for treating coinfection. In their model, they did not consider protection against all infections rather focused on hepatotoxicity and antiretroviral therapeutic effects in HIV/HBV coinfection. They obtained results showing that emtricitabine, tenofovir, and efavirenz are the optimal combination that maximises the therapeutic effect of therapy and minimizes the toxic response to medication in HBV/HIV coinfection. Bowong et al. [13] formulated and analyzed a realistic mathematical model for HBV-HIV coinfection, which incorporates the key epidemiological and biological features of each of the two diseases in a population. In their model, they did not consider treatment for all infections rather focused only on vaccination against HBV infection. The above two studies motivated us to develop our new model. Therefore, in this paper, we proposed and rigorously analyzed a deterministic mathematical model incorporating vaccination against hepatitis B virus and treatment for all infections on the
2. Model Assumptions and Formulation

The model subdivides the total population at time $t$ denoted by $N(t)$, into nine mutually exclusive compartments depending on their disease status: susceptible individuals to both diseases ($S(t)$), immune individuals following vaccination to HBV infection ($V(t)$), HBV-only infected class ($I_B(t)$), HIV-only infected class ($I_H(t)$), HBV-HIV/AIDS coinfected class ($I_{BH}(t)$), HBV-HIV/AIDS-treated class ($T_{BH}(t)$), HBV-treated class ($T_B(t)$), HIV-treated class ($T_H(t)$), and suppressed viral load class ($S_V(t)$). The integration among people is homogeneous and become infected with HIV and HBV by the force of infection $\lambda_H$ and $\lambda_B$, respectively. Individuals are recruited into the population (vaccinated class) at a constant rate $b$ with the proportion $\rho$ of which are immunized to protect them from HBV infection. The size of the vaccinated class decreases due to the natural death rate $\mu$, the transfer of HIV-infected individuals, and individuals whose vaccine efficacy wanes to $I_H$ and susceptible classes, respectively. The efficacy of the vaccine wanes out at a rate $q$ and the natural recovery rate of individuals from HBV infection in HBV-only infected class is $\sigma$. The susceptible class increases due to the recruitment of unvaccinated individuals, the transfer of naturally recovered individuals from HBV-only infected class, and the transfer of individuals whose vaccine efficacy wanes. Because of the natural mortality rate $\mu$ and the movement of HBV and HIV-infected individuals to HBV-only and HIV-only infected classes, respectively, the susceptible class decreases. Individuals in $S$ and $V$ compartments get HIV infection by the force of infection $\lambda_H$, whereas individuals in $S$ get HBV infection by the force of infection $\lambda_B$. Individuals in HBV-only infected class get HIV infection by force of infection $\lambda_H$ within a brief period of time before an infection with the first strain (HBV) has been established and an immune response has developed. Similarly, individuals in HIV-only infected class get HBV infection by force of infection $\lambda_B$ within a brief period of time before an infection with the first strain (HIV) has been established and an immune response has developed. Naturally recovered patients from HBV infection in HBV-HIV/AIDS-coinfected class enter in to HIV-only infected class at a rate $r$. Since the effective treatment reduces the viral load of the infected individuals in $T_B$, $T_H$, and $T_{BH}$ classes to the required undetectable level, individuals in these compartments progress to a suppressed viral load class at the progress rate $\phi$, $\theta$, and $\varepsilon$, respectively. If the vaccine efficacy not wanes, individuals who are vaccinated against HIV infection are susceptible to HIV-only. Due to the fact that there may be no immunity to loss life whether or not one is unwell or healthy, the natural death rate for individuals in different classes is the same. We take no consideration of disease-related deaths in all treated classes due to the effective treatment. HBV-infected individuals get treatment at chronic stage only. The immunity acquired naturally by HBV-infected individuals is lifelong. No individual gets infected with both diseases at once, rather he/she gets infected with one of the two diseases first and the second afterwards, but within a brief period of time before infection with the first strain is established. Individuals in HBV-only infected class who are able to clear the infection due to natural immunity enter into the susceptible class at rate $\sigma$, but they are not susceptible to HBV infection again since they develop antibodies and protected from HBV infection for lifelong. Vertical transmission for both HBV and HIV has not been considered here.

Using parameters and their description in Table 1 and the model assumptions and formulation given in Section 2, the schematic diagram for the transmission dynamics of HBV-HIV/AIDS coinfected is given by the following:

From the flow diagram of the model in Figure 1, the dynamical system of the model is as follows:

\[ \frac{dS}{dt} = (1-\rho)b + qV + \sigma I_B - (\lambda_H + \lambda_H + \mu)S, \]
\[ \frac{dV}{dt} = \rho b - (\lambda_H + q + \mu)V, \]
\[ \frac{dI_B}{dt} = \lambda_B S - (\sigma + \alpha + \lambda_H + \delta_B + \mu)I_B, \]
\[ \frac{dI_H}{dt} = \lambda_H (S + V) + r I_{BH} - (\pi\gamma + (1-\pi)\lambda_B + \delta_H + \mu)I_H, \]
\[ \frac{dI_{BH}}{dt} = (1-\pi)\lambda_B I_H + \lambda_H I_B - (r + \beta + \delta_{BH} + \mu)I_{BH}, \]
\[ \frac{dT_B}{dt} = \alpha I_B - (\phi + \mu)T_B, \]
\[ \frac{dT_H}{dt} = \pi\gamma I_H - (\theta + \mu)T_H, \]
\[ \frac{dT_{BH}}{dt} = \beta I_{BH} - (\varepsilon + \mu)T_{BH}, \]
\[ \frac{dS_V}{dt} = \varepsilon T_{BH} + \phi T_B + \theta T_H - \mu S_V, \]

where the force of infection associated with HBV infection is given by the following:

\[ \frac{dI_B}{dt} = (1-\pi)\lambda_B I_H + \lambda_H I_B - (r + \beta + \delta_{BH} + \mu)I_{BH}, \]
\[ \frac{dT_B}{dt} = \alpha I_B - (\phi + \mu)T_B, \]
\[ \frac{dT_H}{dt} = \pi\gamma I_H - (\theta + \mu)T_H, \]
\[ \frac{dT_{BH}}{dt} = \beta I_{BH} - (\varepsilon + \mu)T_{BH}, \]
\[ \frac{dS_V}{dt} = \varepsilon T_{BH} + \phi T_B + \theta T_H - \mu S_V, \]
\[ \lambda_B = \omega_B \frac{(I_B + I_{BH})}{N}, \quad \lambda_H = \omega_H \frac{(I_H + I_{BH})}{N}. \]

and the force of infection associated with HIV infection is given by the following:

Initially, \( S(0) > 0 \), \( V(0) \geq 0 \), \( I_B(0) \geq 0 \), \( I_H(0) \geq 0 \), \( I_{BH}(0) \geq 0 \), \( T_B(0) \geq 0 \), \( T_H(0) \geq 0 \), \( T_{BH}(0) \geq 0 \), and \( S_V(0) \geq 0 \).
2.1. Positivity of the Solutions. For (1) to be epidemiologically meaningful, it is important to prove that all its state variables are nonnegative for all time $t \geq 0$. In other words, the solutions of (1) with positive initial data remain positive for all time $t \geq 0$ because the model deals with the human population. This can be verified as follows:

**Theorem 1.** Let $\Omega = \{(S, V, I_B, I_H, I_{BH}, T_B, T_H, T_{BH}, S_Y) \in \mathbb{R}_+^9 : S(0) > 0, V(0) \geq 0, I_B(0) \geq 0, I_H(0) \geq 0, I_{BH}(0) \geq 0, T_B(0) \geq 0, T_H(0) \geq 0, T_{BH}(0) \geq 0, S_Y(0) \geq 0\}$, then the solutions of $\{S, V, I_B, I_H, I_{BH}, T_B, T_H, T_{BH}, S_Y\}$ are positive for $t \geq 0$.

**Proof.** Taking $ds/dt = (1 - \rho)b + qV + \alpha I_B - (\lambda_B + \lambda_H + \mu)S$ from (1) and making the left side of the equation zero, we have a solution:

$$S(t) = S(0)e^{-(\lambda_B + \lambda_H + \mu)t} + \frac{(1 - \rho)b + qV + \alpha I_B}{\lambda_B + \lambda_H + \mu} \left(1 - e^{-(\lambda_B + \lambda_H + \mu)t}\right).$$

(4)

Since $S(0) > 0$, $e^{-(\lambda_B + \lambda_H + \mu)t} > 0$ and $(1 - \rho)b + qV + \alpha I_B / \lambda_B + \lambda_H + \mu > 0$ for $t \geq 0$, $S(t) = S(0)e^{-(\lambda_B + \lambda_H + \mu)t} + (1 - \rho)b + qV + \alpha I_B / \lambda_B + \lambda_H + \mu > 0$.

Taking $dV/dt = \rho b - (\lambda_H + q + \mu) V$ from (1), we have a solution

$$V(t) = V(0)e^{-(\lambda_H + q + \mu)t} + \frac{\rho b}{\lambda_H + q + \mu} \left(1 - e^{-(\lambda_H + q + \mu)t}\right).$$

(5)

Since $V(0) > 0$, $e^{-(\lambda_H + q + \mu)t} > 0$ and $\rho b / \lambda_H + q + \mu > 0$ for $t \geq 0$, $V(t) = V(0)e^{-(\lambda_H + q + \mu)t} + \rho b / \lambda_H + q + \mu > 0$.

Taking $dI_B/dt = \lambda_B S - (\sigma + \alpha + \lambda_H + \delta_B + \mu) I_B$ from (1), we have a solution

$$I_B(t) = I_B(0)e^{-(\sigma + \alpha + \lambda_H + \delta_B + \mu)t} + \frac{\lambda_B S}{\sigma + \alpha + \lambda_H + \delta_B + \mu} \left(1 - e^{-(\sigma + \alpha + \lambda_H + \delta_B + \mu)t}\right).$$

(6)

Since $I_B(0) > 0$, $e^{-(\sigma + \alpha + \lambda_H + \delta_B + \mu)t} > 0$ and $\lambda_B S / (\sigma + \alpha + \lambda_H + \delta_B + \mu) > 0$ for $t \geq 0$, $I_B(t) > 0$.

Taking $dI_H/dt = \lambda_H S + \lambda_H V + r I_{BH} - (\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu) I_H$ from (2), we have a solution

$$I_H(t) = I_H(0)e^{-(\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu)t} + \frac{\lambda_H S + \lambda_H V + r I_{BH}}{\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu} \left(1 - e^{-(\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu)t}\right).$$

(7)

Since $I_H(0) > 0$, $e^{-(\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu)t} > 0$ and $\lambda_H S + \lambda_H V + r > 0$, $I_{BH}\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu > 0$.

$$I_H(t) = I_H(0)e^{-(\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu)t} + \frac{\lambda_H S + \lambda_H V + r I_{BH}}{\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu} \left(1 - e^{-(\pi_Y + (1 - \pi)\lambda_B + \delta_H + \mu)t}\right).$$

(8)

Taking $dI_{BH}/dt = (1 - \pi)\lambda_B I_H + \lambda_H I_B - (r + \beta + \delta_{BH} + \mu) I_{BH}$ from (2), we have a solution

$$I_{BH}(t) = I_{BH}(0)e^{-(r + \beta + \delta_{BH} + \mu)t} + \frac{(1 - \pi)\lambda_B I_H + \lambda_H I_B}{r + \beta + \delta_{BH} + \mu} \left(1 - e^{-(r + \beta + \delta_{BH} + \mu)t}\right).$$

(9)

Since $I_{BH}(0) > 0$, $e^{-(r + \beta + \delta_{BH} + \mu)t} > 0$ and $(1 - \pi)\lambda_B I_H + \lambda_H I_B > 0$ for $t \geq 0$, $I_{BH}(t) = I_{BH}(0)e^{-(r + \beta + \delta_{BH} + \mu)t} + \frac{(1 - \pi)\lambda_B I_H + \lambda_H I_B}{r + \beta + \delta_{BH} + \mu} \left(1 - e^{-(r + \beta + \delta_{BH} + \mu)t}\right) > 0$.

(10)

Taking $dT_B/dt = \alpha I_B - (\phi + \mu) T_B$ from (1), we have a solution

$$T_B(t) = T_B(0)e^{-\phi t} + \frac{\alpha I_B}{\phi + \mu} \left(1 - e^{-\phi t}\right).$$

(11)

Since $T_B(0) > 0$, $e^{-\phi t} > 0$ and $\alpha I_B / (\phi + \mu) > 0$ for $t \geq 0$, $T_B(t) = T_B(0)e^{-\phi t} + \alpha I_B / (\phi + \mu) > 0$.

Taking $dT_H/dt = \pi_Y I_H - (\theta + \mu) T_H$ from (1), we have a solution

$$T_H(t) = T_H(0)e^{-\theta t} + \frac{\pi_Y I_H}{\theta + \mu} \left(1 - e^{-\theta t}\right).$$

(12)

Since $T_H(0) > 0$, $e^{-\theta t} > 0$ and $\pi_Y I_H / (\theta + \mu) > 0$,

$$T_H(t) = T_H(0)e^{-\theta t} + \frac{\pi_Y I_H}{\theta + \mu} \left(1 - e^{-\theta t}\right) > 0.$$ (13)

Taking $dT_{BH}/dt = \beta I_{BH} - (\varepsilon + \mu) T_{BH}$ from (1), we have a solution

$$T_{BH}(t) = T_{BH}(0)e^{-\varepsilon t} + \frac{\beta I_{BH}}{\varepsilon + \mu} \left(1 - e^{-\varepsilon t}\right).$$

(14)

Since $T_{BH}(0) > 0$, $e^{-\varepsilon t} > 0$ and $\beta I_{BH} / (\varepsilon + \mu) > 0$,

$$T_{BH}(t) = T_{BH}(0)e^{-\varepsilon t} + \frac{\beta I_{BH}}{\varepsilon + \mu} \left(1 - e^{-\varepsilon t}\right) > 0.$$ (15)
Taking \( \frac{dS_V}{dt} = \varepsilon T_{BH} + \phi T_B + \theta T_H - \mu S_V \) from (1), we have a solution

\[
S_V(t) = S_V(0)e^{-\mu t} + \frac{\varepsilon T_{BH} + \phi T_B + \theta T_H}{\mu} (1 - e^{-\mu t}).
\]  

(16)

Since \( S_V(0) > 0, e^{-\mu t} > 0 \) and \( \varepsilon T_{BH} + \phi T_B + \theta T_H / \mu > 0 \),

\[
S_V(t) = S_V(0)e^{-\mu t} + \frac{\varepsilon T_{BH} + \phi T_B + \theta T_H}{\mu} (1 - e^{-\mu t}) > 0.
\]  

(17)

This completes the proof of the theorem. Therefore, the solutions of the model are positive. \( \Box \)

2.2. Boundedness of the Solution Region

**Theorem 2.** The solution trajectories of (1) evolve in a positive invariant region.

\[ \Omega = \left\{ (S, V, I_B, I_{BH}, T_B, T_H, T_{BH}, S_V) \in \mathbb{R}^9_{>0} : N \leq \frac{b}{\mu} \right\}. \]  

(18)

**Proof.** Let \( N(t) \) represent the whole population at time \( t \).

Thus,

\[
\begin{align*}
N &= S + V + I_B + I_{BH} + T_B + T_H + T_{BH} + S_V \\
&= \frac{dN}{dt} \\
&= \frac{ds}{dt} + \frac{dv}{dt} + \frac{dI_B}{dt} + \frac{dI_{BH}}{dt} + \frac{dT_B}{dt} + \frac{dT_H}{dt} + \frac{dT_{BH}}{dt} + \frac{dS_V}{dt} \\
&= b - \mu(S + V + I_B + I_{BH} + T_B + T_H + T_{BH} + S_V) \\
&\quad - [(I_B + T_B)\delta_B + (I_{BH} + T_H)\delta_H + (I_{BH} + T_B)\delta_{BH}] \\
&\quad - b - \mu N - [(I_B + T_B)\delta_B + (I_{BH} + T_H)\delta_H + (I_{BH} + T_B)\delta_{BH}].
\end{align*}
\]  

(19)

If \( N > b / \mu \), \( dN / dt < 0 \).

In the absence of mortality due to HBV infection, HIV/AIDS, and HBV-HIV/AIDS coinfection and comparing both sides of the equation using the standard comparison theorem, (19) becomes

\[
\frac{dN}{dt} \leq b - \mu N.
\]  

(20)

Integrating (20) on both sides, we get the following:

(1) \(-1 / \mu \ln (b - \mu N) \leq t + C \) where \( C \) is a constant
(2) \( b - \mu N \geq C_1 e^{-\mu t} \), where \( C_1 = e^{-\mu t} \) is a constant
(3) \( b - \mu N(0) \geq C_1 \iff N(0) \leq b / \mu \)

Now taking \( b - \mu N(0) = C_1 \), the inequality \( b - \mu N \geq C_1 e^{-\mu t} \) becomes the following:

\[
N(t) \leq N(0)e^{-\mu t} + \frac{b}{\mu} (1 - e^{-\mu t}).
\]  

(21)

As \( t \rightarrow \infty \), the population size \( N(t) \rightarrow b / \mu \) whenever \( N(0) \leq b / \mu \) as is follows:

(1) \( 0 \leq N(t) \leq b / \mu \)
(2) All feasible solutions of components of the dynamical system with initial conditions enter the region \( \Omega = \{ (S, V, I_B, I_{BH}, T_B, T_H, T_{BH}, S_V) \in \mathbb{R}^9_{>0} : N \leq b / \mu \} \) for all \( t \geq 0 \)

Thus, the region \( \Omega \) is positively invariant.

After checking the positivity of the solutions and the boundedness of the solution region of the full model, we considered the dynamics of the two submodels, namely, HBV-only and HIV/AIDS-only submodels. This would help us to lay down the foundation for the qualitative analysis of the full mode. \( \Box \)

3. Analysis of the Submodels

The analysis of the full model will be followed by analyzing the dynamics of the hepatitis B (HB) only and HIV-only submodels.

3.1. HB-Only Submodel. The HB-only model is obtained by setting \( I_{BH} = T_B = T_{BH} = S_V = 0 \). Thus, we have the following dynamical system:

\[
\begin{align*}
\frac{ds}{dt} &= (1 - \rho) b + qV - (\lambda_B + \mu) S, \\
\frac{dv}{dt} &= \rho b - (\varphi + \mu) V, \\
\frac{dI_B}{dt} &= \lambda_B S - (\sigma + \alpha + \delta_B + \mu) I_B, \\
\frac{dT_B}{dt} &= \alpha I_B - \mu T_B,
\end{align*}
\]  

(22)

where \( \lambda_B = \omega_B(I_B/N) \) is the force of infection.

The solutions of the model are positive, and the trajectories evolve in a positive invariant region

\[
\Omega_B = \left\{ (S, V, I_B, T_B) \in \mathbb{R}^3_{>0} : N \leq \frac{b}{\mu} \right\}.
\]  

(23)

3.1.1. Disease-Free Equilibrium Point (DFE) of HBV-Only Submodel. The DFE of the dynamical system in (22) represented by \( E^0_B \) is equal to \( E^0_B = \{(b(q + \mu(1 - \rho)))/\mu(q + \mu), \rho b/q + \mu, 0, 0\} \). It is obtained by making the right-hand side of (22) and the infectious and treatment class zero.

3.1.2. Effective and Basic Reproduction Numbers of the Model. The effective reproduction number of hepatitis B-infected individuals, denoted by \( R_e^B \), that of the dynamical system (22), is defined as the expected number of secondary cases produced by one typical infection joining in a
population made up of both susceptible and nonsusceptible hosts during its infectious period [24, 25]. It is obtained by taking the spectral radius of the matrix \( FV^{-1} = (\rho(FV^{-1})) \) as follows:

\[
\rho(FV^{-1}) = \frac{\omega_B(q + \mu(1 - \rho))}{(q + \mu)(\sigma + \delta_B + \mu)} \implies FV^{-1} = \frac{\omega_B(q + \mu(1 - \rho))}{(q + \mu)(\sigma + \delta_B + \mu)} \implies \rho(FV^{-1}) = \frac{\omega_B(q + \mu(1 - \rho))}{(q + \mu)(\sigma + \delta_B + \mu)}.
\]

On the other hand, the basic reproduction number of the dynamical system (22), denoted by \( RB \), is derived when initially the entire population is susceptible [27, 28]. That is, when there is no vaccination and treatment (\( \rho = \alpha = 0 \)). In model (22), \( \mathcal{F} = [\lambda_B S] \) and \( \mathcal{V} = [(\sigma + \alpha + \delta_B + \mu)I_B] \).

The associated Jacobian matrices of \( \mathcal{F} \) and \( \mathcal{V} \) evaluated at DFE point, respectively, are as follows:

\[
F = \frac{\omega_B(q + \mu(1 - \rho))}{(q + \mu)} \quad \text{and} \quad V = \sigma + \alpha + \delta_B + \mu \implies V^{-1} = \frac{1}{\sigma + \delta_B + \mu} \implies \rho(FV^{-1}) = \frac{\omega_B(q + \mu(1 - \rho))}{(q + \mu)(\sigma + \delta_B + \mu)}.
\]

3.1.3 Stability of the Disease-Free Equilibrium (DFE) of HB-Only Submodel

**Theorem 3.** The disease-free equilibrium \( E^0_B = (b(q + \mu(1 - \rho))/\mu(q + \mu), \rho B(q + \mu, 0, 0) \) of (22) is locally asymptotically stable if the effective reproduction number \( R^B_{\text{eff}} < 1 \) and is unstable otherwise.

**Proof.** The Jacobian matrix of (22) at the DFE point \( E^0_B = (b(q + \mu(1 - \rho))/\mu(q + \mu), \rho B(q + \mu, 0, 0) \) is as follows:

\[
J(E^0_B) = \begin{bmatrix}
\frac{\partial f_1(E^0_B)}{\partial S} & \frac{\partial f_1(E^0_B)}{\partial V} & \frac{\partial f_1(E^0_B)}{\partial I^0_B} & \frac{\partial f_1(E^0_B)}{\partial T^0_B} \\
\frac{\partial f_2(E^0_B)}{\partial S} & \frac{\partial f_2(E^0_B)}{\partial V} & \frac{\partial f_2(E^0_B)}{\partial I^0_B} & \frac{\partial f_2(E^0_B)}{\partial T^0_B} \\
\frac{\partial f_3(E^0_B)}{\partial S} & \frac{\partial f_3(E^0_B)}{\partial V} & \frac{\partial f_3(E^0_B)}{\partial I^0_B} & \frac{\partial f_3(E^0_B)}{\partial T^0_B} \\
\frac{\partial f_4(E^0_B)}{\partial S} & \frac{\partial f_4(E^0_B)}{\partial V} & \frac{\partial f_4(E^0_B)}{\partial I^0_B} & \frac{\partial f_4(E^0_B)}{\partial T^0_B}
\end{bmatrix}
\]

The corresponding characteristic equation is as follows:

\[
\begin{vmatrix}
-(\mu + \lambda) & q & \sigma - \frac{\omega_B(q + \mu(1 - \rho))}{q + \mu} & 0 \\
0 & -(\sigma + \mu) & \frac{\omega_B(q + \mu(1 - \rho))}{q + \mu} & 0 \\
0 & 0 & \frac{\omega_B(q + \mu(1 - \rho))}{q + \mu} - (\sigma + \alpha + \delta_B + \mu) - \lambda & 0 \\
0 & 0 & 0 & -\mu - (\mu + \lambda)
\end{vmatrix} = 0.
\]

Thus, after some steps, the roots of the characteristic equation are \( \lambda_1 = \lambda_2 = -\mu, \lambda_3 = -(q + \mu) \) and

\[
\lambda_4 = \frac{-\alpha - \mu - \alpha \mu - \mu^2 - \sigma \alpha - \mu \sigma - \alpha \delta_B - \mu \delta_B + \alpha \omega_B + \mu \omega_B - \mu \rho \omega_B}{q + \mu}.
\]

But the fourth eigenvalue \( \lambda_4 \) can be simplified as \( (\sigma + \alpha + \delta_B + \mu)(R^B_{\text{eff}} - 1) \).

1. \( \lambda_4 \) is negative if \( R^B_{\text{eff}} < 1 \)

2. All the eigenvalues of the Jacobian matrix have negative real parts when \( R^B_{\text{eff}} < 1 \)
Hence, the disease-free equilibrium 
\[ E_0^B = \left( (b(q + \mu(1 - \rho))/\mu(q + \mu), (pb(q + \mu), 0, 0) \right) \] of (22) is locally asymptotically stable if the effective reproduction number \( R_{eff}^B < 1 \) and unstable otherwise.

**Theorem 4.** The disease-free equilibrium \( E_0^B = (b(q + \mu(1 - \rho))/\mu(q + \mu), pb(q + \mu, 0, 0) \) of (22) is globally asymptotically stable in the feasible region \( \Omega_B \) if the effective reproduction number \( R_{eff}^B < 1 \) and is unstable otherwise.

**Proof.** Consider the following LaSalle-Lyapunov candidate function:
\[ \mathcal{L}_B = b_1 I_B, \] (29)
where
\[ b_1 = \frac{1}{\sigma + \alpha + \delta_B + \mu}. \] (30)

\( \mathcal{L}_B \) is a continuous function for all \((S, V, I_B, T_B) \in \mathbb{R}^4_{>0}, \) and it has first-order partial derivative and has minimum at
\[ E_0^B = \left( (b(q + \mu(1 - \rho))/\mu(q + \mu), pb(q + \mu, q + \mu, 0, 0) \right). \] (31)

Its time derivative along the solution path yields the following:
\[ \dot{\mathcal{L}}_B = b_1 \dot{I}_B = b_1 \left( \frac{\omega B S}{N} - (\sigma + \alpha + \delta_B + \mu) \right) I_B \]
\[ = b_1 \left( \frac{\omega B S}{N} - (\sigma + \alpha + \delta_B + \mu) \right) I_B. \] (32)

Since the state variables of the model when the HBV is endemic in the population do not exceed the state variables of the model in a population free of HBV, at the disease-free equilibrium \( E_0^B = (b(q + \mu(1 - \rho))/\mu(q + \mu), pb(q + \mu, 0, 0) \) with
\[ R_{eff}^B = \frac{\omega B (q + \mu(1 - \rho))}{(q + \mu)(\sigma + \alpha + \delta_B + \mu)}, \] (33)

\( \mathcal{L}_B \) can be simplified as follows:
\[ \dot{\mathcal{L}}_B = b_1 \left( \frac{\omega B S}{N} - (\sigma + \alpha + \delta_B + \mu) \right) I_B \]
\[ = b_1 \left( \frac{\omega B (q + \mu(1 - \rho))}{q + \mu} \right) I_B \]
\[ = b_1 \left( \frac{R_{eff} B (\sigma + \alpha + \delta_B + \mu)}{(q + \mu)(q + \mu)} \right) I_B \]
\[ = b_1 \left( \frac{R_{eff} B (\sigma + \alpha + \delta_B + \mu)}{(q + \mu)(q + \mu)} \right) I_B \]
\[ = b_1 \left( \frac{R_{eff} B (\sigma + \alpha + \delta_B + \mu)}{(q + \mu)(q + \mu)} \right) I_B. \] (34)

This implies \( \dot{\mathcal{L}}_B \leq 0 \) when \( R_{eff}^B < 1. \) Furthermore, \( \dot{\mathcal{L}}_B = 0 \) if and only if \( I_B = 0. \) Thus, by LaSalle’s invariance principle [29], the largest invariant set in \( \Omega_B \) contained in \( \{ (S, V, I_B, T_B) \in \mathbb{R}^4_{>0} \} \) is reduced to the DFE. This proves the global asymptotic stability of the DFE \( E_0^B \) on \( \Omega_B \) if the effective reproduction number \( R_{eff}^B < 1 \) and unstable otherwise.

3.1.4. Existence and Stability of Endemic Equilibrium (EE) of HB-Only Submodel. Let an arbitrary equilibrium point of HB-only submodel (22) be denoted by \( E^*_{\text{HB}} = (S^*, V^*, I^*_{\text{HB}}, T^*_{\text{HB}}) \) and \( \lambda_{\text{HB}}^* = \omega_{\text{HB}}^*/N^* \) be the associated infection rate (“force of infection”) at the endemic equilibrium point. After some calculations, we get \( \lambda_{\text{HB}}^* = \mu B (\sigma + \alpha + \delta_B + \mu)/\lambda_{\text{HB}}^* \) if \( R_{eff}^B - 1 \) \( (\alpha/\mu)(q + \mu) + \mu B \). This implies \( \lambda_{\text{HB}}^* > 0 \) if \( R_{eff}^B > 1 \), and hence, an endemic equilibrium point \( E^*_{\text{HB}} = (S^*, V^*, I^*_{\text{HB}}, T^*_{\text{HB}}) \) of HB-only submodel (22) exists whenever \( R_{eff}^B > 1 \).

\[ S^* = \frac{b((\alpha + \mu)(q + \mu(1 - \rho)) + \mu \alpha B (\sigma + \alpha + \delta_B + \mu))}{\mu(q + \mu)(\alpha + \alpha + \delta_B + \mu) + (\alpha + \delta_B + \mu)(\alpha + \alpha + \delta_B + \mu)} \]
\[ V^* = \frac{\rho B}{q + \mu} \]
\[ I^*_{\text{HB}} = \frac{\mu B ((\alpha + \mu)(q + \mu(1 - \rho)) + \mu B (\alpha + \delta_B + \mu)(\alpha + \alpha + \delta_B + \mu))((\alpha + \alpha + \delta_B + \mu))}{(q + \mu)(\alpha + \alpha + \delta_B + \mu) - \omega_{\text{HB}} B} \]
\[ T^*_{\text{HB}} = \frac{\mu B ((\alpha + \mu)(q + \mu(1 - \rho)) + \mu B (\alpha + \delta_B + \mu)(\alpha + \alpha + \delta_B + \mu))((\alpha + \alpha + \delta_B + \mu))}{(q + \mu)(\alpha + \alpha + \delta_B + \mu) - \omega_{\text{HB}} B}. \] (35)
**Theorem 5.** The positive endemic equilibrium \( E^* = (S^*, V^*, I^* B, T^* B) \) of (22) is locally asymptotically stable if \( R^B_{eff} > 1 \).

**Proof.** The Jacobian matrix of (22) is as follows:

\[
J_B = \begin{pmatrix}
-(\omega_B I^*_B/N^* + \mu) & q & \sigma - \omega_B S^*/N^* & 0 \\
0 & -(q + \mu) & 0 & 0 \\
\omega_B I^*_B/N^* & 0 & \omega_B S^*/N^* - (\alpha + \sigma + \delta_B + \mu) & 0 \\
0 & 0 & \alpha & -\mu
\end{pmatrix}
\]  

(36)

The Jacobian matrix of (3)/(22) at the endemic equilibrium \( E_B = (S^*, V^*, I^*_B, T^*_B) \) is as follows:

\[
J_B(E^*_B) = \begin{pmatrix}
-(q + \mu)(\omega_B - (\alpha + \sigma + \delta_B + \mu)) + \mu(\alpha + \delta_B + \mu - \omega_B) & q & -\alpha - \delta_B + \mu & 0 \\
\rho(\alpha + \delta_B + \mu) & 0 & -(q + \mu) & 0 \\
(q + \mu)(\omega_B - (\alpha + \sigma + \delta_B + \mu)) - \omega_B \mu \rho & 0 & 0 & 0 \\
\rho(\alpha + \delta_B + \mu) & 0 & \alpha & -\mu
\end{pmatrix}
\]  

(37)

Let \( \lambda_i \) be the eigenvalues of the Jacobian matrix \( J_B(E^*_B) \) where \( i = 1, 2, 3, 4 \).

Then, the eigenvalues of the Jacobian matrix \( J_B(E^*_B) \) are \( \lambda_1 = -\mu < 0 \) or \( \lambda_2 = -(q + \mu) < 0 \) or \( k_2 \lambda^2 + k_1 \lambda + k_0 = 0 \) where

\[
k_1 = 1, k_2 = \frac{(q + \mu)(\alpha + \sigma + \delta_B + \mu)(R^B_{eff} - 1) + \mu(\alpha + \delta_B + \mu)}{\rho(\alpha + \delta_B + \mu)}
\]

(38)

To check the algebraic sign of the remaining two eigenvalues for the quadratic equation \( k_2 \lambda^2 + k_1 \lambda + k_0 = 0 \), we can apply Routh-Hurwitz stability criteria. Since \( k_2 > 1 > 0 \), both \( k_1 \) and \( k_2 \) should be positive. Clearly, \( k_1 \) and \( k_2 \) are positive if \( R^B_{eff} > 1 \).

This implies \( \lambda_3 < 0 \) and \( \lambda_4 < 0 \).

Hence, since all eigenvalues are negative, the endemic equilibrium \( E^*_B = (S^*, V^*, I^*_B, T^*_B) \) of (22) is locally asymptotically stable if \( R^B_{eff} > 1 \).

\( \square \)

**Theorem 6.** The endemic equilibrium \( E^*_B = (S^*, V^*, I^*_B, T^*_B) \) of (22) is globally asymptotically stable if \( R^B_{eff} > 1 \).

**Proof.** Let us take the Lyapunov function \( \mathcal{L}_B = b_1(S - S^*)^2 + b_2(I_B - I^*_B)^2 \), \( b_1 > 0, b_2 > 0 \).

The time derivative of \( \mathcal{L}_B(S, I_B) \) is

\[
\dot{\mathcal{L}}_B = \frac{2b_1}{S} (S - S^*) (S_0 - S^*) + \frac{2b_2}{I_B} (I_B - I^*_B) (I_{0B} - I^*_B)
\]

Using expressions at the endemic equilibrium \( (1 - \rho)b + \rho V + \sigma I_B = (\omega_B^B N^* + \mu)^* S^* \), \( \sigma + \alpha + \delta_B + \mu = \omega_B^S / N^* \), and \( N = N^* \),

\[
\dot{\mathcal{L}}_B = \frac{2b_1}{S} (S - S^*) \left( \omega_B^B S^* (S^* - S) - \omega_B^B S^* (S^* - S) \right)
\]

(39)
\[ + 2b_2 \frac{\omega_B I_B}{N} (I_B - I_B^*)(S - S^*) \]

\[ = -2b_1 \mu (S - S^*)(S - S^*) + \frac{2b_2 \omega_B}{N} S^* (I_B^* - I_B)(S - S^*) \]

\[ - \frac{2b_2 \omega_B}{N} I_B (S - S^*)(S - S^*) - 2b_2 \frac{\omega_B I_B}{N} (I_B^* - I_B)(S - S^*) \]

\[ = -2b_1 \mu (S - S^*)^2 \]

\[ + \frac{2b_2 \omega_B}{N} S^* (I_B^* - I_B)(S - S^*) - 2b_2 \frac{\omega_B I_B}{N} (I_B^* - I_B)(S - S^*) \]

\[ = -2b_1 \mu (S - S^*)^2 \]

\[ + \frac{2\omega_B}{N} (I_B^* - I_B)(S - S^*)(b_1 S^* - b_2 I_B) \]

\[ \leq 0 \text{ if } I_B \leq \frac{b_1 S^*}{b_2}, I_B^* \leq I_B \text{ and } S \geq S^*. \] (40)

This implies \( \mathcal{R}_b \leq 0 \), and it vanishes only at the endemic equilibrium \( E^*_B \).

Hence, by LaSalle’s invariance principle [30], all model solutions approach to \( E^*_B \) as \( t \to \infty \) whenever \( R^H_{\text{eff}} > 1 \).

This proves the global asymptotic stability of the endemic equilibrium point \( E^*_B \) on \( \Omega_B \) if the effective reproduction number \( R^H_{\text{eff}} > 1 \).

3.2. HIV/AIDS-Only Submodel. The HIV-only model is obtained by setting \( V = I_B = I_{BH} = T_B = T_{BH} = S_V = 0 \). Thus, we have the following dynamical system:

\[
\frac{ds}{dt} = b - (\lambda_H + \mu)S,
\]

\[
\frac{dI^*_H}{dt} = \lambda_H S - (\pi \gamma + \delta_H + \mu)I^*_H,
\]

\[
\frac{dT^*_H}{dt} = \pi \gamma I^*_H - \mu T^*_H,
\]

where

\[
\lambda_H = \frac{\omega_H I_H}{N}. \]

The solutions of the model are positive, and the trajectories evolve in a positive invariant region \( \Omega_H = \{(S, I^*_H, T^*_H) \in \mathbb{R}_+^3 : N \leq b/\mu \} \).

3.2.1. Disease-Free Equilibrium Point (DFE) of HIV/AIDS-Only Submodel. The DFE of the dynamical system in (41), denoted by \( E^0_H \), is equal to \( E^0_H = (b/\mu, 0, 0) \). It is obtained by making the right-hand side of (41) zero and the infectious and treatment classes as well.

3.2.2. Effective and Basic Reproduction Numbers of HIV/AIDS-Only Submodels. The effective reproduction number of HIV/AIDS-only infected individuals, denoted by \( R^H_{\text{eff}} \) that of the dynamical system (41), is defined as the expected number of secondary cases produced by one typical infection joining in a population made up of both susceptible and nonsusceptible hosts during its infectious period [24, 25]. It is obtained by taking the spectral radius of the matrix \( FV^{-1} = (\rho(FV^{-1})) = [\partial \mathcal{F}_i(E^0_H)/\partial x_i][\partial v_i(E^0_H)/\partial x_i]^{-1} \) of the dynamical system (41) [25, 26], where \( \mathcal{F}_i \) is the rate of appearance of a new infection in the compartment \( i \), \( \gamma \) is the transfer of infections from one compartment \( i \) to another, and \( E^0_H \) is the disease-free equilibrium point.

In model (41), \( \mathcal{F} = [\lambda_H S] \) and \( \gamma' = [\pi \gamma + \delta_H + \mu]I_H \).

The associated Jacobian matrices of \( \mathcal{F} \) and \( \gamma' \) evaluated at DFE point, respectively, are

\[
F = \omega_H \text{ and } V = \pi \gamma + \delta_H + \mu \implies V^{-1} = \frac{1}{\pi \gamma + \delta_H + \mu} \implies FV^{-1} = \frac{\omega_H}{\pi \gamma + \delta_H + \mu} \implies R^H_{\text{eff}} = \frac{\omega_H}{\delta_H + \mu}. \] (43)

On the other hand, the basic reproduction number of HIV/AIDS-only infected individuals, denoted by \( R^H_0 \) that of the dynamical system (41), is defined as the average number of secondary infections produced by a single HIV/AIDS infectious individual introduced in a completely susceptible population during his or her entire infectious period [27, 28]. It is obtained by taking the spectral radius of the matrix \( FV^{-1} = (\rho(FV^{-1})) = [\partial \mathcal{F}_i(E^0_H)/\partial x_i][\partial v_i(E^0_H)/\partial x_i]^{-1} \) of the dynamical system (41) [25, 26], where \( \mathcal{F}_i \) is the rate of appearance of a new infection in the compartment \( i \), \( \gamma \) is the transfer of infection from one compartment \( i \) to another, and \( E^0_H \) is the disease-free equilibrium point.

In model (41), \( \mathcal{F} = [\lambda_H S] \) and \( \gamma' = [\pi \gamma + \delta_H + \mu]I_H \).

The associated Jacobian matrices of \( \mathcal{F} \) and \( \gamma' \) evaluated at DFE point, respectively, are

\[
F = \omega_H \text{ and } V = \pi \gamma + \delta_H + \mu \implies V^{-1} = \frac{1}{\pi \gamma + \delta_H + \mu} \implies FV^{-1} = \frac{\omega_H}{\pi \gamma + \delta_H + \mu} \implies R^H_0 = \frac{\omega_H}{\delta_H + \mu}. \] (44)

3.2.3. Stability of the Disease-Free Equilibrium (DFE) of HIV/AIDS-Only Submodel

**Theorem 7.** The disease-free equilibrium \( E^0_H = (b/\mu, 0, 0) \) of (41) is locally asymptotically stable if the basic reproduction number \( R^H_0 < 1 \) and is unstable otherwise.

**Proof.** The Jacobean matrix of (41) at the DFE point \( E^0_H = (b/\mu, 0, 0) \) is as follows:

\[
F = \omega_H \text{ and } V = \pi \gamma + \delta_H + \mu \implies V^{-1} = \frac{1}{\pi \gamma + \delta_H + \mu} \implies FV^{-1} = \frac{\omega_H}{\pi \gamma + \delta_H + \mu} \implies R^H_0 = \frac{\omega_H}{\delta_H + \mu}. \] (44)
The disease-free equilibrium point 

\[ E_0 = \left( \frac{\omega_0}{\omega_0 + \delta_H + \mu} \right) I 
\]

is asymptotically stable if \( RH \leq 1 \). Hence, the disease-free equilibrium point \( E_0 \) of (41) is locally asymptotically stable if \( RH < 1 \) and unstable otherwise.

**Theorem 8.** The disease-free equilibrium point \( E_0 \) of (41) is globally asymptotically stable in the feasible region \( \Omega_H \) if \( RH < 1 \) and unstable otherwise.

\[
J(E_0^H) = \begin{bmatrix}
\frac{\partial f_1(E_0^H)}{\partial S} & \frac{\partial f_1(E_0^H)}{\partial I_H} & \frac{\partial f_1(E_0^H)}{\partial T_H} \\
\frac{\partial f_2(E_0^H)}{\partial S} & \frac{\partial f_2(E_0^H)}{\partial I_H} & \frac{\partial f_2(E_0^H)}{\partial T_H} \\
\frac{\partial f_3(E_0^H)}{\partial S} & \frac{\partial f_3(E_0^H)}{\partial I_H} & \frac{\partial f_3(E_0^H)}{\partial T_H} \\
\end{bmatrix} = \begin{bmatrix}
-\mu & -\omega_H & 0 \\
0 & \omega_H - (\pi \gamma + \delta_H + \mu) & 0 \\
0 & \pi \gamma & -\mu \\
\end{bmatrix}
\]

The corresponding characteristic equation is as follows:

\[
-(\mu + \lambda) \begin{vmatrix}
-\omega_H & 0 \\
0 & (\pi \gamma + \delta_H + \mu) - \omega_H + \lambda & 0 \\
0 & \pi \gamma & -(\mu + \lambda) \\
\end{vmatrix} = 0.
\]

After some necessary steps, the roots of the characteristic equation are \( \lambda_1 = \lambda_2 = -\mu \) or \( \lambda_3 = -(\mu + \pi \gamma + \delta_H) + \omega_H \).

But, to show that all eigenvalues are negative, it is enough to show that \( \lambda_3 \) is negative for \( RH < 1 \).

Now

\[
\lambda_3 = -(\mu + \pi \gamma + \delta_H) + \omega_H \\
= -(\mu + \pi \gamma + \delta_H) + RH_0(\mu + \pi \gamma + \delta_H) \\
= (\mu + \pi \gamma + \delta_H)(RH_0 - 1).
\]

(1) \( \lambda_3 \) is negative if \( RH_0 < 1 \)

(2) All eigenvalues of the Jacobean matrix have negative real parts when \( RH_0 < 1 \)

Hence, the disease-free equilibrium point \( E_0 \) of (41) is locally asymptotically stable if \( RH_0 < 1 \) and unstable otherwise.

**Proof.** Consider the following LaSalle-Lyapunov candidate function:

\[
\mathcal{L}_H = h_1 I_H,
\]

where \( h_1 = 1/\pi \gamma + \delta_H + \mu \).

\( \mathcal{L}_H \) is a continuous function, for all \((S, I_H, T_H) \in \mathbb{R}^3_{>0} \), and has first-order partial derivative and has a minimum at \( E_0^H = (b/\mu, 0, 0) \).

Its time derivative along the solution path yields the following:

\[
\dot{\mathcal{L}}_H = h_1 \left( \frac{\omega_0 I_H S}{N} - (\pi \gamma + \delta_H + \mu) \right) I_H \\
= h_1 \left( \frac{\omega_0 S}{N} - (\pi \gamma + \delta_H + \mu) \right) I_H.
\]

Since the state variables of the model when HIV/AIDS is endemic in the population do not exceed the state variables of the model in a population free of HIV/AIDS, at the disease-free equilibrium \( E_0^H = (b/\mu, 0, 0) \) with \( RH_0 < 1 \), \( \mathcal{L}_H \) can be simplified as follows:

\[
\dot{\mathcal{L}}_H \leq h_1 \left( \frac{S}{N} - (\pi \gamma + \delta_H + \mu) \right) I_H \\
= h_1 \left( \frac{S}{N} - (\pi \gamma + \delta_H + \mu) \right) I_H \\
= h_1 \left( \frac{\pi \gamma + \delta_H + \mu}{R_0 - 1} I_H \right). \\
\]

This implies \( \dot{\mathcal{L}}_H \leq 0 \) when \( RH_0 < 1 \). Furthermore, \( \dot{\mathcal{L}}_H = 0 \) if and only if \( I_H = 0 \). Thus, by LaSalle’s invariance principle [30], the largest invariant set in \( \Omega_H \) contained in \((S, I_H, T_H) \in \mathbb{R}^3_{>0} \) is reduced to the DFE. This proves the global asymptotic stability of the DFE \( E_0^H \) on \( \Omega_H \) if the basic reproduction number \( R^H_0 < 1 \) and unstable otherwise.

3.2.4. Existence and Stability of the Endemic Equilibrium Point (EE) of HIV/AIDS-Only Submodel. Let an arbitrary equilibrium point of HIV/AIDS-only submodel (41) be denoted by \( E^* = (S^*, I^*_{H'}, T^*_{H'}) \) and \( \lambda^*_{eff} = \omega_H I^*_H/N^* \) be the associated infection rate (“force of infection”) at the endemic equilibrium point. After some calculations, we get \( \lambda^*_{eff} = \mu(\pi \gamma + \delta_H + \mu)/(R^H_{eff} - 1)/\pi \gamma + \mu \). This implies \( \lambda^*_{eff} > 0 \) if \( R^H_{eff} > 1 \), and hence, an endemic equilibrium point \( E^* = (S^*, I^*_{H'}, T^*_{H'}) \) of a dynamical system (41) exists whenever \( R^H_{eff} > 1 \), where

\[
E^*_{H'} = \left( \frac{b(\pi \gamma + \mu)}{\mu(\omega_H - (\pi \gamma + \delta_H + \mu)) + \mu(\pi \gamma + \mu)} \frac{b(\omega_H - (\pi \gamma + \delta_H + \mu))}{(\pi \gamma + \delta_H + \mu)((\omega_H - (\pi \gamma + \delta_H + \mu)) + \pi \gamma + \mu)} \frac{\pi \gamma b(\omega_H - (\pi \gamma + \delta_H + \mu))}{\mu(\pi \gamma + \delta_H + \mu)((\omega_H - (\pi \gamma + \delta_H + \mu)) + \pi \gamma + \mu)} \right). 
\]
The Jacobian matrix of (41) is as follows:

\[
J_H = \begin{pmatrix}
\left(-\frac{\omega H I^*_H}{N^*} + \mu\right) & -\frac{\omega H S^*}{N^*} & 0 \\
\frac{\omega H I^*_H}{N^*} & \frac{\omega H S^*}{N^*} - (\pi \gamma + \delta_H + \mu) & 0 \\
0 & \pi \gamma & -\mu
\end{pmatrix}.
\]  
(52)

The Jacobian matrix of (41) at the endemic equilibrium point is as follows:

\[
J_H(E^*_H) = \begin{pmatrix}
-\mu(\omega H S^* + \mu) & \pi \gamma + \delta_H + \mu & 0 \\
\omega H S^* & \mu(\omega H S^* + \mu) & 0 \\
0 & \pi \gamma & -\mu
\end{pmatrix}.
\]  
(53)

Let \(\lambda_i\) be the eigenvalues of (53) where \(i = 1, 2, 3\). Then, the eigenvalues of (53) are \(\lambda_1 = -\mu < 0\) or \(m_2 \lambda^2 + m_1 \lambda + m_0 = 0\) where

\[
m_2 = 1,
\]

\[
m_1 = \frac{\mu((\pi \gamma + \delta_H + \mu) (R_H^{\text{eff}} - 1) + \pi \gamma + \mu)}{\pi \gamma + \mu},
\]

\[
m_0 = \frac{\mu(\pi \gamma + \delta_H + \mu)^2 (R_H^{\text{eff}} - 1)}{\pi \gamma + \mu}.
\]  
(54)

To check the algebraic sign of the remaining two eigenvalues for the quadratic equation \(m_2 \lambda^2 + m_1 \lambda + m_0 = 0\), we can apply Routh-Hurwitz stability criteria. Since \(m_2 = 1 > 0\), both \(m_1\) and \(m_0\) should be positive.

Clearly, \(m_1\) and \(m_0\) are positive if \(R_H^{\text{eff}} > 1\).

This implies \(\lambda_3 < 0\) and \(\lambda_2 < 0\).

Hence, since all eigenvalues are negative, the endemic equilibrium \(E^*_H = (S^*, I^*_H, T^*_H)\) of (41) is locally asymptotically stable if \(R_H^{\text{eff}} > 1\).

**Theorem 9.** The endemic equilibrium \(E^*_H = (S^*, I^*_H, T^*_H)\) of (41) is locally asymptotically stable if \(R_H^{\text{eff}} > 1\).

**Proof.** Consider the following LaSalle-Lyapunov candidate function:

\[
\mathcal{L}_H(S, I_H) = h_1(S - S^*)^2 + h_2(I_H - I^*_H)^2, h_1 > 0, h_2 > 0.
\]  
(55)

Differentiating \(\mathcal{L}_H(S, I_H)\) with respect to time gives the following:

\[
\dot{\mathcal{L}}_H(S, I_H) = 2h_1(S - S^*) \dot{S} + 2h_2(I_H - I^*_H) \dot{I}_H
\]

\[
= 2h_1(S - S^*) \left(b - \frac{\omega H I_H}{N} + \mu\right) S
\]

\[
+ 2h_2(I_H - I^*_H) \left(\omega H I_H - (\pi \gamma + \delta_H + \mu) I_H\right).
\]  
(56)

Substituting \(b = (\omega H I^*_H / N^* + \mu) S^*\) and \(\pi \gamma + \delta_H + \mu = \omega H S^*/N^*\) with \(N = N^*\) in (10) yields the following:

\[
\mathcal{L}_H(S, I_H) = 2h_1(S - S^*) \left(\frac{\omega H I_H}{N} S + \omega H I_H - \omega H S^* - \mu S\right)
\]

\[
+ 2h_2(I_H - I^*_H) \left(\omega H I_H S - \omega H S^*\right)
\]

\[
= 2h_1(S - S^*) \left(\mu(S^* - S) + \omega H (I^*_H - I_H) + (S^* - S)\right)
\]

\[
+ 2h_2 \omega H (I^*_H - I_H) (S^* - S)
\]

\[
= -2h_1(\mu(S^* - S) + \omega H (I^*_H - I_H)) (S^* - S)
\]

\[
- 2h_2 \omega H (I - I_H) (S^* - S)
\]

\[
= -2h_1(\mu(S - S^*) + \omega H (I^*_H - I_H)) (S^* - S)
\]

\[
+ 2h_1 \omega H (I^*_H - I_H) (S^* - S)
\]

\[
= 0 \text{ if } I_H \geq \frac{h_1 S^*}{h_2}, S^* \leq S, I^*_H \geq I_H.
\]  
(57)

This implies \(\dot{\mathcal{L}}_H(S, I_H) \leq 0\), and it vanishes only at the endemic equilibrium \(E^*_H\).

Hence, by LaSalle’s invariance principle [30], all model solutions approach to \(E^*_H\) as \(t \to +\infty\) whenever \(R_H^{\text{eff}} > 1\).

This proves the global asymptotic stability of the endemic equilibrium point \(E^*_H\) on \(\Omega_H\) if the basic reproduction number \(R_H^{\text{eff}} > 1\).

**4. Analysis of the Full Model**

The solutions of the HBV-HIV/AIDS coinfection model are positive, and the trajectories evolve in a positive invariant region \(\Omega = \{(S, V, I_H, I_H, T_H, T_H, \text{BIV}, T_B, T_B, S_V) \in \mathbb{R}_{>0} : N \leq b / \mu\} \) as we proved in Sections 2.1 and 2.2.
4.1. Disease-Free Equilibrium (DFE) of HBV-HIV/AIDS Coinfection Model. The disease-free equilibrium of the full model represented by \( E^0_{BH} \) in (1) is obtained by setting \( I_B = I_{BH} = T_B = T_{BH} = S_V = 0 \), and it is equal to \( E^0_{BH} = (b(q + \mu(1 - \rho)))/(\mu(q + \mu), \rho b(q + \mu, 0, 0, 0, 0, 0)) \).

4.2. Basic Reproduction Number \( R^0_{BH} \) of the Full Model. The basic reproduction number of HBV-HIV/AIDS coinfected individuals, denoted by \( R^0_{BH} \) of the dynamical system (1), is defined as the average number of secondary infections produced by a single HBV-HIV/AIDS coinfected individual introduced in a completely susceptible population during his or her entire infectious period [27, 28]. It is obtained by taking the spectral radius of the matrix \( FV^{-1} = (\rho(FV^{-1})) = \left| \frac{\partial F(E^0_{BH})}{\partial x} \right| \frac{\partial V(E^0_{BH})}{\partial x} \right|^{-1} \) of the dynamical system (1) [25, 26] where \( F \), is the rate of appearance of a new infection in the compartment \( i \), \( v_i \) is the transmission rate of infections from one compartment \( i \) to another, and \( E^0_{BH} \) is the disease-free equilibrium point.

In model (1), we have three infection classes. Therefore,

\[
F = \begin{bmatrix}
\omega_B & 0 & \omega_B \\
0 & \omega_{IH} & \omega_{IH} \\
0 & 0 & 0
\end{bmatrix}
\quad \text{and} \quad
V = \begin{bmatrix}
\sigma + \delta_B + \mu & 0 & 0 \\
0 & \delta_H + \mu & -r \\
0 & 0 & r + \delta_{BH} + \mu
\end{bmatrix}
\Rightarrow V^{-1} = \begin{bmatrix}
\frac{1}{\mu + \sigma + \delta_B} & 0 & 0 \\
0 & \frac{1}{\mu + \delta_B} & \frac{r}{(r + \mu + \delta_{BH})(\mu + \delta_H)} \\
0 & 0 & \frac{1}{r + \mu + \delta_{BH}}
\end{bmatrix}
\Rightarrow FV^{-1}
\]

The corresponding characteristic equation is as follows:

\[
\left| \begin{array}{ccc}
\frac{\omega_B}{\mu + \sigma + \delta_B} - \lambda & 0 & \omega_B \\
0 & \frac{\omega_{IH}}{\mu + \delta_B} - \lambda & \frac{\omega_{IH}}{r + \mu + \delta_{BH}} \\
0 & 0 & \frac{\omega_{IH}}{\mu + \delta_B} - \lambda
\end{array} \right| = 0 \Rightarrow \left( \frac{\omega_B}{\mu + \sigma + \delta_B} - \lambda \right) \left( \frac{\omega_{IH}}{\mu + \delta_B} - \lambda \right) = 0 \Rightarrow \lambda_1 = 0, \lambda_2 = \frac{\omega_B}{\mu + \sigma + \delta_B}, \text{and} \lambda_3 = \frac{\omega_{IH}}{\mu + \delta_B}
\]

Now, \( \rho(FV^{-1}) = \max \{ \omega_B/\mu + \sigma + \delta_B, \omega_{IH}/\mu + \delta_B \} \).
Hence, \( R^0_{BH} = \max \{ \omega_B/\mu + \sigma + \delta_B, \omega_{IH}/\mu + \delta_B \} \).

4.3. Effective Reproduction Number \( R^*_{BH} \) of the Full Model. The effective reproduction number of HBV-HIV/AIDS-coinfected individuals, denoted by \( R^*_{BH} \), is defined as the expected number of secondary cases produced by one typical infection joining in a population made up of both susceptible and nonsusceptible hosts during its infectious period [24, 25]. To obtain \( R^*_{BH} \), we used the next generation matrix method that was formulated by [26, 27].
The associated Jacobian matrices of $F$ and $V$ evaluated at DFE point, respectively, are

$$
F = \begin{bmatrix}
\frac{\omega_B(q + \mu(1 - \rho))}{q + \mu} & \frac{\omega_B(q + \mu(1 - \rho))}{q + \mu} \\
0 & \frac{\omega_H(q + \mu(1 - \rho))}{q + \mu} \\
0 & \omega_H \\
0 & \omega_H \\
\end{bmatrix}
$$

and $V = \begin{bmatrix}
\sigma + \delta_B + \mu & 0 & 0 \\
0 & \pi \gamma + \delta_H + \mu & -r \\
0 & 0 & r + \beta + \delta_{BH} + \mu \\
\end{bmatrix} \implies V^{-1}
$$

$$
= \begin{bmatrix}
1 & 0 & 0 \\
0 & \frac{1}{\mu + \pi \gamma + \delta_H} & \frac{r}{(r + \beta + \mu + \delta_{BH})(\mu + \pi \gamma + \delta_H)} \\
0 & 0 & \frac{1}{r + \beta + \mu + \delta_{BH}} \\
\end{bmatrix} \implies FV^{-1}
$$

$$
= \begin{bmatrix}
\frac{\omega_B(q + \mu(1 - \rho))}{(q + \mu)(\alpha + \mu + \sigma + \delta_B)} & 0 & \frac{\omega_B(q + \mu(1 - \rho))}{(q + \mu)(r + \beta + \mu + \delta_{BH})} \\
0 & \frac{\omega_H}{\mu + \pi \gamma + \delta_H} & \frac{\omega_H}{r + \beta + \mu + \delta_{BH}} + \frac{\rho \omega_H}{(r + \beta + \mu + \delta_{BH})(\mu + \pi \gamma + \delta_H)} \\
0 & 0 & 0 \\
\end{bmatrix}.
$$

After some steps, the eigenvalues of (62) are $\lambda_1 = 0, \lambda_2 = (q + \mu - \mu \rho)\omega_B/(q + \mu)(\alpha + \mu + \sigma + \delta_B)$, and $\lambda_3 = \omega_H/\mu + \pi \gamma + \delta_H$.

Now,

$$
\rho(FV^{-1}) = \max \{ \omega_B(q + \mu - \mu \rho)/(q + \mu)(\alpha + \mu + \sigma + \delta_B), \omega_H/\mu + \pi \gamma + \delta_H \} \implies R_{BH}^{eff} = \max \{ \omega_B(q + \mu - \mu \rho)/(q + \mu)(\alpha + \mu + \sigma + \delta_B), \omega_H/\mu + \pi \gamma + \delta_H \}.
$$

4.4. Stability of the Disease-Free Equilibrium (DFE) of the HBV-HIV/AIDS Coinfection Model

**Theorem 11.** The disease-free equilibrium point $E_{BH}^0 = (b(q + \mu(1 - \rho))/\mu(q + \mu), pb/q + \mu, 0, 0, 0, 0, 0, 0, 0)$ of (1) is locally asymptotically stable if $R_{BH}^{eff} < 1$. Proof. The Jacobian matrix of (1) at the DFE point $E_{BH}^0 = (b(q + \mu(1 - \rho))/\mu(q + \mu), pb/q + \mu, 0, 0, 0, 0, 0, 0, 0)$ is as follows:

$$
J(E_{BH}^0) = \begin{bmatrix}
-\mu & q & \sigma - \omega_{hd} & -\omega_{hd} & -\omega_H \phi & -\omega_H \theta & -\omega_H \epsilon & 0 & 0 \\
0 & -(q + \mu) & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & \omega_{hd} - (\sigma + \alpha + \delta_B + \mu) & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \omega_H - (\pi \gamma + \delta_H + \mu) & \omega_H + r & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & -\phi + \mu & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \pi \gamma & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & \beta & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & \phi & \theta & \epsilon & -\mu \\
\end{bmatrix}.
$$


where

\[ k_1 = (q + \mu), k_2 = (\sigma + \alpha + \delta_B + \mu), k_3 = (\pi\gamma + \delta_H + \mu), k_4 = (r + \beta + \delta_{BH} + \mu), k_5 = (\phi + \mu), k_6 = (\theta + \mu), k_7 = (\varepsilon + \mu), d = \frac{(q + \mu(1 - \rho))}{q + \mu}. \]  

(65)

Let \( \lambda_i \) be eigenvalues of \( J(E^0_{BH}) \) where \( i = 1, \ldots, 9 \).

Then, the eigenvalues of \( J(E^0_{BH}) \) are \( \lambda_1 = -\mu < 0 \) or \( \lambda_2 = -(q + \mu) < 0 \) or \( \lambda_3 = -(c + \mu) < 0 \) or \( \lambda_4 = -\theta < 0 \) or \( \lambda_5 = -(\mu + \phi) < 0 \) or \( \lambda_6 = -(r + \beta + \mu + \delta_{BH}) < 0 \) or \( \lambda_7 = -(\mu + \delta_B + \mu) < 0 \) and \( \lambda_8 = -(\mu + \delta_H + \mu) < 0 \).

Consider the following Lasalle-Lyapunov candidate function:

\[ V = \frac{(b(q + \mu(1 - \rho)))}{\mu(q + \mu)}, \frac{\rho b}{q + \mu}, 0, 0, 0, 0, 0, 0. \]  

(71)

with \( R^{BH}_{eff} = \max \{ \frac{\omega_B(q + \mu - \mu\rho)}{(q + \mu)(\alpha + \mu + \sigma + \delta_B)}, \omega_H(\mu + \pi\gamma + \delta_H) \} \). \( \mathcal{L} \) can be simplified as follows:

\[ \mathcal{L} = a_1 I_B + a_2 I_H + I_{BH}. \]  

(67)

where

\[ a_1 = \frac{1}{2}(r + \beta + \delta_{BH} + \mu), \]  

(68)

\[ a_2 = \frac{1}{2}(r + \beta + \delta_{BH} + \mu). \]

\( \mathcal{L} \) is a continuous function for all \( (S, V, I_B, I_H, I_{BH}, T_B, T_H, T_{BH}, S_G) \in \mathbb{R}^{7+\delta} \), and it has first-order partial derivative and has a minimum at

\[ E^0_{BH} = \left( \frac{b(q + \mu(1 - \rho))}{\mu(q + \mu)}, \frac{\rho b}{q + \mu}, 0, 0, 0, 0, 0, 0, 0 \right). \]  

(69)

Its time derivative along the solution path yields the following:

\[ \dot{\mathcal{L}} = a_1 \dot{I}_B + a_2 \dot{I}_H + \dot{I}_{BH} \]

\[ = a_1 \left( \omega_B(I_B + I_{BH}) \frac{S}{N} - \omega_H (I_H + I_{BH}) \frac{I_H}{N} - (\sigma + \alpha + \delta_B + \mu) I_B \right) + a_2 \left( \omega_H (I_H + I_{BH}) \frac{(S + V)}{N} + r I_{BH} \right) - \omega_B (1 - \pi)(I_B + I_{BH}) I_H - (\pi\gamma + \delta_H + \mu) I_H \]

\[ + \omega_B (1 - \pi)(I_B + I_{BH}) I_H + \omega_H (I_H + I_{BH}) I_B - (r + \beta + \delta_{BH} + \mu) I_{BH}. \]  

(70)

Since the state variables of the model when the HBV-HIV/AIDS is endemic in the population do not exceed the state variables of the model in a population free of HBV-HIV/AIDS, at the disease-free equilibrium

\[ E^0_{BH} = \left( \frac{b(q + \mu(1 - \rho))}{\mu(q + \mu)}, \frac{\rho b}{q + \mu}, 0, 0, 0, 0, 0, 0, 0 \right). \]  

(72)

Substituting \( a_1 \) and \( a_2 \), we get
\[
\frac{1}{2}(r + \beta + \delta_{BH} + \mu)(R_B^{BH} - 1)I_B + \frac{1}{2}(r + \beta + \delta_{BH} + \mu)(R_H^{BH} - 1)I_H + \left(\frac{R_B^{BH}}{2} + \frac{R_H^{BH}}{2} - 1\right)I_{BH}
\]

\[
\leq \left(\frac{r + \beta + \delta_{BH} + \mu}{(r + \beta + \delta_{BH} + \mu)}\left(\frac{R_B^{BH}}{2} + \frac{R_H^{BH}}{2} - 1\right)\right)I_{BH} - \frac{1}{2}(r + \beta + \delta_{BH} + \mu)(R_B^{BH} - 1)I_B
\]

\[
+ \frac{1}{2}(r + \beta + \delta_{BH} + \mu)(R_H^{BH} - 1)I_H + \left(\frac{R_B^{BH}}{2} + \frac{R_H^{BH}}{2} - 1\right)I_{BH}
\]

\[
= \frac{1}{2}(r + \beta + \delta_{BH} + \mu)(R_B^{BH} - 1)I_B + \frac{1}{2}(r + \beta + \delta_{BH} + \mu)(R_H^{BH} - 1)I_H + \left(\frac{r + \beta + \delta_{BH} + \mu}{(r + \beta + \delta_{BH} + \mu)}\left(\frac{R_B^{BH}}{2} + \frac{R_H^{BH}}{2} - 1\right)\right)I_{BH}
\]

This implies \( \mathcal{D} \leq 0 \) when \( R_B^{BH} \) is less than 1. Furthermore, \( \mathcal{D} = 0 \) if and only if \( I_B = I_H = I_{BH} = 0 \). Thus, by LaSalle’s invariance principle [30], the largest invariant set in \( \Omega \) contained in \( \{(S, V, I_B, I_H, I_{BH}, V_H, T_B, T_H, T_{BH}, S_V, V_V) \in \mathbb{R}^{18}\} \) is reduced to the DFE. This proves the global asymptotic stability of the DFE \( E_0^D \) on \( \Omega \) if the effective reproduction number \( R_B^{BH} \) is less than 1 and is unstable otherwise. \( \square \)

4.5. Existence and Stability of Endemic Equilibrium (EE) of HBV-HIV/AIDS Coinfection Model. Let the unique endemic equilibrium point of a dynamical system in (1) which exists when the disease persists in the community be denoted by \( E^*_{BH} = (S^*, V^*, I_B^*, I_H^*, I_{BH}^*, V_H^*, T_B^*, T_H^*, T_{BH}^*, S_V^*, V_V^*) \). From the analysis of HBV-only submodel (22) and HIV/AIDS-only submodel (41), there exists an endemic equilibrium point for HBV-only submodel and HIV/AIDS-only submodel if \( R_B^H > 1 \) and \( R_H^{eff} > 1 \), respectively.

This implies the endemic equilibrium \( E^*_{BH} = (S^*, V^*, I_B^*, I_H^*, I_{BH}^*, V_H^*, T_B^*, T_H^*, T_{BH}^*, S_V^*, V_V^*) \) for HBV-HIV/AIDS coinfection exists because the effective reproduction number for HBV- HIV/AIDS coinfection is the maximum of \( R_B^{eff} \), \( R_H^{eff} \), which is greater than one where

\[
S^* = \frac{b(q + (1 - \rho)(\lambda^{*}_{BH} + \mu))(\sigma + a + \lambda^{*}_{BH} + \delta_{BH} + \mu)}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)},
\]

\[
V^* = \frac{\rho \lambda^{*}_{BH}}{\lambda^{*}_{BH} + \mu},
\]

\[
I^*_B = \frac{\lambda^{*}_{BH}b(1 - \rho)(\lambda^{*}_{BH} + \mu)(q + (1 - \rho)(\lambda^{*}_{BH} + \mu))}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)},
\]

\[
I^*_H = \frac{\lambda^{*}_{BH}(1 - \rho)(\lambda^{*}_{BH} + \mu)(a + \lambda^{*}_{BH} + \delta_{BH} + \mu)}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)},
\]

\[
I^*_{BH} = \frac{\lambda^{*}_{BH}(1 - \rho)(\lambda^{*}_{BH} + \mu)(a + \lambda^{*}_{BH} + \delta_{BH} + \mu)}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)},
\]

\[
T^*_B = \frac{(\beta + \lambda^{*}_{BH})b(1 - \rho)(\lambda^{*}_{BH} + \mu)(q + (1 - \rho)(\lambda^{*}_{BH} + \mu))}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)} + \frac{\beta + \lambda^{*}_{BH}}{\lambda^{*}_{BH} + \mu} + \frac{\lambda^{*}_{BH}(\sigma + \rho)(\lambda^{*}_{BH} + \mu)}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)},
\]

\[
T^*_H = \frac{\beta + \lambda^{*}_{BH}}{\lambda^{*}_{BH} + \mu} + \frac{\beta + \lambda^{*}_{BH}}{\lambda^{*}_{BH} + \mu} + \frac{\lambda^{*}_{BH}(\sigma + \rho)(\lambda^{*}_{BH} + \mu)}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)},
\]

\[
S^*_V = \frac{\mu(\delta + \lambda^{*}_{BH})(1 - \rho)(\lambda^{*}_{BH} + \mu)(a + \lambda^{*}_{BH} + \delta_{BH} + \mu)}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)} + \frac{\lambda^{*}_{BH}b(1 - \rho)(\lambda^{*}_{BH} + \mu)(q + (1 - \rho)(\lambda^{*}_{BH} + \mu))}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)} + \frac{\beta + \lambda^{*}_{BH}}{\lambda^{*}_{BH} + \mu} + \frac{\beta + \lambda^{*}_{BH}}{\lambda^{*}_{BH} + \mu} + \frac{\lambda^{*}_{BH}(\sigma + \rho)(\lambda^{*}_{BH} + \mu)}{\lambda^{*}_{BH} + \rho(\lambda^{*}_{BH} + \mu)},
\]
The stability analysis of the endemic equilibrium of the full model (1) in terms of the model parameters analytically is difficult. Hence, we will give an explanation of the stability analysis of the endemic equilibrium \( E^*_{BH} \) of the full model (1) under the section numerical simulation.

5. Sensitivity Analysis

In this subsection, sensitivity analysis is performed to identify the most influential parameters for the spread as well as for control of infection in the community. To perform this, we use the techniques described in [36]. The results of sensitivity analysis and the set of parameters used are given in Figure 2 and Table 2, respectively.

Figure 2 is about the sensitivity index of parameters. It shows that the most sensitive parameters are \( \omega_B \) and \( \omega_H \), which are the effective contact rate for HBV and HIV/AIDS transmission, respectively. The least sensitive parameters are \( \delta_B \) and \( q \) which are HBV-induced mortality and vaccine efficacy waning rate, respectively.

6. Numerical Simulation

We performed the numerical simulation using the parameter values in Table 2 and the initial values given below.

Initially, \( S(0) = 100,000, \ V(0) = 1000, \ I_B(0) = 10,000, \ I_H(0) = 3000, \ I_{BH}(0) = 1000, \ T_B(0) = 500, \ T_H(0) = 500, \ T_{BH}(0) = 100, \) and \( S_V(0) = 200. \) Initially, \( S(0) = 100,000, \ V(0) = 1000, \ I_B(0) = 10,000, \ I_H(0) = 3000, \ I_{BH}(0) = 1000, \ T_B(0) = 500, \ T_H(0) = 500, \ T_{BH}(0) = 100, \) and \( S_V(0) = 200. \)

7. Results and Discussions

Numerical simulations are carried out using MATLAB numerical solver (ode45) to visualize the dynamics of HBV-only submodel (22), HIV/AIDS-only submodel (41), and HBV-HIV/AIDS coinfection model (1). Figures 3–8 are about the stability of disease-free and endemic equilibria of HB-only submodel, HIV/AIDS-only submodel, and HBV-HIV/AIDS coinfection model at different reproduction numbers and the associated parameters given in Table 2. Figures 3 and 7 were plotted using the effective reproduction number \( R_{BH}^{eff} = 0.046, \) which is less than unity, \( \omega_B = 0.09, \) and \( \mu = 0.5 \) and keeping all other associated parameters as listed in Table 2. As the figures clearly show, the behavior of the infectious classes of HBV-only submodel (22) and HBV-HIV/AIDS coinfection model (1), each infectious class converges to the disease-free equilibrium point of

| Parameters | Value | Source |
|------------|-------|--------|
| \( \mu \)  | 0.01  | [31]   |
| \( b \)    | 0.04 \( * N_0 \) | Assumption |
| \( \delta_H \) | 0.333 | [31] |
| \( \delta_B \) | 0.01 | [32] |
| \( \alpha \) | 0.3 | Assumption |
| \( \gamma \) | 0.6 | [33] |
| \( \omega_B, \omega_H \) | 0.001, 0.015, and 0.001, respectively | Assumption |
| \( \sigma \) | 0.075 | Assumption |
| \( \rho \) | 0.65 | [34] |
| \( \pi \) | 0.396 | [33] |
| \( \omega_B \) | 0.4 | [34] |
| \( \omega_H \) | 0.03 | Assumption |
| \( q \) | 0.1 | [34, 35] |
| \( \phi, \theta, \epsilon \) | 0.014, 0.013, and 0.012, respectively | Assumption |
the models. The convergence of all infectious classes to their disease-free equilibrium point of the models shows that the disease-free equilibria of the models are globally asymptotically stable, which indicates the absence of HBV and HBV-HIV/AIDS coinfection in the society. The simulation result of the stability of the DFE of HIV/AIDS-only submodel
Figure 5: Stability of DFE of HIV/AIDS-only submodel at $R_0^{H} = 0.003$, $\omega_H = 0.003$, and $\mu = 0.5$.

Figure 6: Stability of EE of HIV/AIDS-only submodel at $R_0^{H} = 3.5$, $\omega_H = 0.9$, and $\delta_H = 0.01$. 
using the basic reproduction number $R_0^H = 0.003$, $\omega_H = 0.003$, and $\mu = 0.5$ and keeping all other associated parameters as listed in Table 2 is represented by Figure 5. The figure shows the convergence of the infection class to the disease-free equilibrium point of the model, which shows that the disease-free equilibrium point of the model is globally asymptotically stable. The global stability of the DFE of the model further indicates the infection is not spreading in the society or the disease dies out in the community. On the other hand, the simulations given in Figures 4, 6, and 8 are about the stability of endemic equilibrium of HB-only submodel, HIV/AIDS-only submodel, and HBV-HIV/AIDS coinfection model, respectively. The stability of endemic equilibria of the models was determined using $R_{\text{eff}}^H = 2.37$, $\omega_B = 0.95$, and $q = 0.4$ and keeping all other associated parameters as listed in Table 2 for HB-only submodel, $R_{\text{eff}}^H = 3.5$, $\omega_H = 0.9$, and $\delta_H = 0.01$ and keeping all other associated parameters as listed in Table 2 for HIV/AIDS-only submodel, and $R_{\text{eff}}^{HBH} = 6.8$, $\omega_B = 0.9$, $\omega_H = 0.5$, and $\alpha = 0.03$ and keeping all other associated parameters as listed in Table 2 for HBV-HIV/AIDS coinfection model. In all cases, the basic and effective reproduction numbers are greater than one, and the simulation results show the convergence of the solutions of the models to their endemic equilibrium points. The convergence of the solutions of the models to their endemic equilibrium point indicates that the endemic equilibrium points of the models are locally asymptotically stable (i.e., the diseases are spreading in the society). The effect of vaccination on the susceptible individuals for HBV is shown in Figure 9. When the proportion of immunized individuals increases from 0.91 to 0.99, the number of susceptible individuals decreases due to enough immunization coverage. In the first year, the trend of decreasing of the susceptible individuals for three different proportions seems the same, but after a year, the number of susceptible individuals decreases better as the proportion of vaccinated individuals increases. Since it is difficult to immunize all susceptible individuals, the number of susceptible individuals cannot be zero over the period of administration of vaccine. The simulations given in Figures 10–12 show the effects of variation of treatment rates on the infectious population. Figure 10 shows that the number of HB infectious individuals decreases, and almost all become near to zero after four years if the treatment rate $\alpha = 0.525$. It also shows using treatment at the highest possible rate decreases the number of HBV infectious individuals faster compared to using treatment at the lowest rate. The decreasing of the number of HB infectious individuals indicates that the effective treatment reduces the viral load of HBV infectious individuals to the required undetectable level, and hence, there will be no risk of transmitting the infection to others in the society. Similarly, Figure 11 shows that the number of HIV/AIDS infectious individuals decreases and becomes near to zero after seven years by increasing the treatment rate gamma from 0.6 to 0.9. This implies the effective treatment reduced the viral load of HIV infectious individuals to the required undetectable level. The effect of varying treatment rates on
Figure 8: Stability of EE of HBV-HIV/AIDS coinfection model at $R_{\text{eff}}^{BH} = 6.8, \omega_B = 0.9, \omega_H = 0.5$, and $\alpha = 0.03$.

Figure 9: The effect of vaccination on HB susceptible individuals.
Figure 10: The effect of treatment on HBV infectious individuals.

Figure 11: Effect of treatment on HIV/AIDS infectious individuals.
HBV and HIV/AIDS coinfectious individuals simultaneously is examined in Figure 12. As the figure shows, the number of HBV-HIV/AIDS coinfectious individuals initially 500 was decreased to about less than 20 individuals at the end of five years when the treatment rate beta = 0.18, which shows using treatment at the highest possible rate decreases the number of HBV-HIV/AIDS coinfectious individuals better compared to using treatment at the lowest rates. The decreasing of the number of HBV-HIV/AIDS coinfectious individuals indicates that the effective treatment reduces
the viral load of HBV-HIV/AIDS coinfectious individuals to the required undetectable level and the risk of transmitting the infection to others in the society decreases. Figures 13–16 are the results of the numerical simulations of the effective reproduction number of HBV-HIV/AIDS coinfection model with variable parameter values. The simulation results in Figures 13 and 16 show that if the effective contact rates \( \omega_B \) and \( \omega_H \) are less than 0.42 and 0.58, respectively, the reproduction number of HBV-HIV/AIDS coinfection is less than one, which indicates HBV-HIV/AIDS coinfection dies out, whereas if the effective contact rates \( \omega_B \) and \( \omega_H \) are greater than 0.42 and 0.58, respectively, the reproduction number of HBV-HIV/AIDS coinfection is greater than one, which shows the infection is spreading in the society.

Figure 14: Simulation of the reproduction number of HBV-HIV/AIDS coinfection at a variable proportion of vaccinated individuals \( \rho \).

Figure 15: Simulation of the reproduction number of HBV-HIV/AIDS coinfection at variable treatment rate \( \alpha \).
community. On the other hand, Figures 14 and 15, respectively, show the absence of disease in the society if the proportion of vaccinated individuals $\rho$ and the treatment rate $\alpha$ are greater than 0.14 and 0.28, respectively, and the existence of HBV-HIV/AIDS coinfection in the society if the proportion of vaccinated individuals $\rho$ and the treatment rate $\alpha$ are less than 0.14 and 0.28, respectively.

### 8. Conclusion

In this study, we formulated and analyzed a deterministic model to see the effect of vaccination and treatment on the transmission dynamics of HBV-HIV/AIDS coinfection. The basic reproduction number of HBV-HIV/AIDS coinfection model derived before implementing any intervention was $\mathcal{R}_0^{BH} = \max \{ \omega_B/\mu + \sigma + \delta_B, \omega_H/\mu + \delta_H \}$ and the effective reproduction number derived after implementing both vaccination and treatment was $\mathcal{R}_\text{eff}^{BH} = \max \{ \omega_B(q + \mu(1 - \rho))/(q + \mu)(\alpha + \mu + \sigma + \delta_B), \omega_H/\mu + \pi_H + \delta_H \}$. Based on the data given in Table 2, we evaluated the numerical values of the basic and effective reproduction numbers to evaluate the effectiveness of our intervention. As the numerical values indicate, the basic reproduction number $\mathcal{R}_0^{BH} = \max \{ 4.2, 0.09 \} = 4.2$ and the effective reproduction number $\mathcal{R}_\text{eff}^{BH} = \max \{ 0.94, 0.28 \} = 0.94$. This tells us one infectious individual infects at least 4 healthy individuals in his/her infectious time if we do not implement any intervention mechanism. However, after incorporating vaccination and treatment efforts together in our model to reduce the burden caused by HBV-HIV/AIDS coinfection, the effective reproduction number becomes $\mathcal{R}_\text{eff}^{BH} = \max \{ 0.94, 0.28 \} = 0.94$, which is less than the threshold value one. This implies the disease dies out in the community, and there will be no risk of transmitting the infection to others due to the effective vaccination and treatment efforts. We proved that the disease-free equilibrium points of the sub and full models are locally and globally asymptotically stable if the associated reproduction numbers $\mathcal{R}_\text{eff}^B$, $\mathcal{R}_\text{eff}^H$, and $\mathcal{R}_\text{eff}^{BH}$ are less than one and the endemic equilibrium points of the sub and full models are locally and globally asymptotically stable whenever the associated reproduction numbers $\mathcal{R}_\text{eff}^B$, $\mathcal{R}_\text{eff}^H$, and $\mathcal{R}_\text{eff}^{BH}$ are greater than one. From the sensitivity analysis carried out to illustrate the influence of different parameters on the effective reproduction number of HBV-HIV coinfection model, $\omega_B$ and $\omega_H$, which are the effective contact rate for HBV and HIV transmission, respectively, are the most sensitive parameters. As the simulation results show, if the effective contact rates $\omega_B$ and $\omega_H$ are less than 0.42 and 0.58 for HBV and HIV transmission, respectively, the proportion of immune individuals following vaccination to HBV infection is greater than 0.14, the treatment rate $\alpha$ for HB chronic carriers is greater than 0.28, and the treatment rate $\beta$ is greater than or equal 0.18 for HBV-HIV/AIDS coinfected individuals, then the number of HBV infectious individuals, the number of HIV infectious individuals, and the number of HBV and HIV/AIDS coinfected individuals decrease significantly and even fall to zero over time. Hence, it is concluded that decreasing the effective contact rates for HBV and HIV transmission and the use of vaccination and treatment at the highest possible rate are essential to control the spread of HBV-HIV/AIDS coinfection. From the numerical results, we recommend that public policymakers and other concerned bodies must focus on increasing vaccination coverage against HBV infection, treatment of hepatitis B and HIV/AIDS infections, and HBV-HIV/AIDS coinfection to control the spread of HBV-HIV/AIDS coinfection.
Finally, we should mention that, for the sake of simplicity, the role of screening in the transmission dynamics of HBV-HIV/AIDS coinfection in a population was not considered in this study. It may affect the transmission dynamics of HBV-HIV/AIDS coinfection in a population. We leave this for future consideration.

Data Availability

All the necessary data used to support the findings of this study are included in the article.

Conflicts of Interest

The authors declare that there is no conflict of interest regarding the publication of this paper.

References

[1] M. H. Nguyen, G. Wong, E. Gane, J.-H. Kao, and G. Dusheiko, "Hepatitis B virus: advances in prevention, diagnosis, and therapy," Clinical Microbiology Reviews, vol. 33, no. 2, 2020.
[2] WHO, Global Hepatitis Report, World Health Organization, Geneva, Switzerland, 2017.
[3] I. A. Moneim, G. A. Mosa, and K. Saud, "Stochastic and Monte Carlo simulation for the spread of hepatitis B," Australian Journal of Basic and Applied Sciences, vol. 3, no. 3, pp. 1607–1615, 2009.
[4] A. Busca and A. Kumar, "Innate immune responses in hepatitis B virus (HBV) infection," Virology Journal, vol. 11, no. 1, pp. 1–8, 2014.
[5] A. R. Kimbir, T. Aboiyar, O. Abu, E. S. Onah, and F. Polytechnic, "Simulation of a mathematical model of hepatitis B virus transmission dynamics in the presence of vaccination and treatment," Mathematical Theory and Modeling, vol. 4, no. 12, pp. 44–60, 2014.
[6] WHO, Global Health Sector Strategy on HIV 2016-2021 towards Ending AIDS, World Health Organization, 2016.
[7] D. W. Gunda, I. Nkandala, S. B. Kilonzo, B. B. Kilangi, and B. C. Mpondoo, "Prevalence and risk factors of mortality among adult HIV patients initiating ART in rural setting of HIV care and treatment services in North Western Tanzania: a retrospective cohort study," Journal of Sexually Transmitted Diseases, vol. 2017, Article ID 7075601, 8 pages, 2017.
[8] J. Lutera, D. Mbete, and S. Wangila, "Co-infection model of HIV/AIDS-pneumonia on the effect of treatment at initial and final stages," Journal of Mathematics, vol. 14, 81 pages, 2018.
[9] F. Khademi, A. Yousefi, A. Sahebkar, and F. Ghanbari, "Bacterial co-infections in HIV/AIDS-positive subjects: a systematic review and meta-analysis," vol. 60, no. 3, pp. 339–350, 2018.
[10] K. A. Johansson, B. Robberstad, and O. F. Norheim, "Further benefits by early start of HIV treatment in low income countries: survival estimates of early versus deferred antiretroviral therapy," AIDS Research and Therapy, vol. 7, no. 1, pp. 1–9, 2010.
[11] E. O. Omondi, R. Mbogo, and L. Luboobi, "Mathematical analysis of sex-structured population model of HIV infection in Kenya," Letters in Biomathematics, vol. 5, no. 1, pp. 174–194, 2018.
[12] K. P. Singh, M. Crane, J. Audsley, and S. R. Lewin, "HIV-hepatitis B virus co-infection: epidemiology, pathogenesis and treatment," AIDS, vol. 31, no. 15, pp. 2035–2052, 2017.
[13] S. Bowong, J. Kamganga, J. Tewa, and B. Tsanou, "Modelling and analysis of hepatitis B and HIV co-infections," in Proceedings of the 10th African Conference on Research in Computer Science and Applied Mathematics, pp. 109–116, 2010.
[14] H. Nampala, M. Jablonska-sabuka, and M. Singull, "Mathematical analysis of the role of HIV/HBV latency in hepatocytes," Journal of Applied Mathematics, vol. 2021, Article ID 5525857, 15 pages, 2021.
[15] R. Rajbhandari, T. Jun, H. Khalili, R. T. Chung, and A. N. Ananthakrishnan, "HBV/HIV coinfection is associated with poorer outcomes in hospitalized patients with HBV or HIV," Journal of Viral Hepatitis, vol. 23, no. 10, pp. 820–829, 2016.
[16] J. Pinchoff, O. C. Tran, L. Chen, K. Bornschlegel, and A. Drobnik, "Impact of hepatitis B on mortality and specific causes of death in adults with and without HIV co-infection in NYC, 2000–2011," Epidemiology and Infection, vol. 144, no. 16, pp. 3354–3364, 2016.
[17] Z.-H. Shen, Y.-M. Chu, M. A. Khan, S. Muhammad, O. A. Al-Hartomy, and M. Higazy, "Mathematical modeling and optimal control of the COVID-19 dynamics," Results in Physics, vol. 31, article 105028, 2021.
[18] S. Kumar, R. Kumar, I. Singh, K. S. Nisar, and D. Kumar, "An efficient numerical scheme for fractional model of HIV-1 infection of CD4 + T-cells with the effect of antiviral drug therapy," Alexandria Engineering Journal, vol. 59, no. 4, pp. 2053–2064, 2020.
[19] X. Li, Y. Wang, M. Altaf, M. Y. Alshahrani, and T. Muhammad, "A dynamical study of SARS-COV-2: A study of third wave," Results in Physics, vol. 29, article 104705, 2021.
[20] E. Bonyah, M. A. Khan, K. O. Okosun, and J. F. Gómez-Aguilar, "On the co-infection of dengue fever and Zika virus," Optimal Control Applications & Methods, vol. 40, no. 3, pp. 394–421, 2019.
[21] K. O. Okosun, M. A. Khan, E. Bonyah, and S. T. Ogunlade, "On the dynamics of HIV-AIDS and cryptosporidiosis," The European Physical Journal Plus, vol. 132, no. 8, 2017.
[22] K. O. Okosun, M. A. Khan, E. Bonyah, and O. O. Okosun, "Cholera-schistosomiasis coinfection dynamics," Optimal Control Applications & Methods, vol. 40, no. 4, pp. 703–727, 2019.
[23] H. Nampala, L. S. Luboobi, J. Y. T. Mugisha, C. Obua, and M. Jablonska-Sabuka, "Modelling hepatotoxicity and antiretroviral therapeutic effect in HIV/HBV coinfection," Mathematical Biosciences, vol. 302, pp. 67–79, 2018.
[24] S. Zhao, S. S. Musa, J. T. Hebert et al., "Modelling the effective reproduction number of vector-borne diseases: the yellow fever outbreak in Luanda, Angola 2015-2016 as an example," PeerJ, vol. 8, no. 2, pp. e8601–e8621, 2020.
[25] P. Van Den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission," Mathematical Biosciences, vol. 180, no. 1–2, pp. 29–48, 2002.
[26] O. Diekmann, J. A. P. Heesterbeek, and J. A. J. Metz, "On the definition and the computation of the basic reproduction ratio RO in models for infectious diseases in heterogeneous populations," Journal of Mathematical Biology, vol. 28, no. 4, pp. 365–382, 1990.
[27] E. A. Bakare and C. R. Nwozo, “Bifurcation and sensitivity analysis of malaria–schistosomiasis co-infection model,” *International Journal of Applied and Computational Mathematics*, vol. 3, no. 1, pp. 971–1000, 2017.

[28] E. Mayanja, L. S. Luboobi, J. Kasozi, and R. N. Nsubuga, “Mathematical modelling of HIV-HCV coinfection dynamics in absence of therapy,” *Computational and Mathematical Methods in Medicine*, vol. 2020, Article ID 2106570, 27 pages, 2020.

[29] J. P. LaSalle, “Stability theory for ordinary differential equations,” *Journal of Difference Equations*, vol. 4, 65 pages, 1968.

[30] J. P. La Salle, “The stability of dynamical systems,” in *CBMSNSF Regional Conference Series in Applied Mathematics*, SIAM, Philadelphia, USA., 1976.

[31] S. W. Teklu and T. T. Mekonnen, “HIV / AIDS-pneumonia coinfection model with treatment at each infection stage : mathematical analysis and numerical simulation,” *Journal of Applied Mathematics*, vol. 2021, Article ID 5444605, 21 pages, 2021.

[32] I. Zada, M. N. Jan, N. Ali, D. Alrowail, and K. S. Nisar, “Mathematical analysis of hepatitis B epidemic model with optimal control,” *Adv. Difference Equ.*, vol. 2021, no. 1, 2021.

[33] D. Omale, “Mathematical modelling on the control of HIV/AIDS with campaign on vaccination and therapy,” *ITM Web of Conferences*, vol. 31, 2020.

[34] S. Bowong and J. Kurths, “Modelling tuberculosis and hepatitis B co-infections,” *Mathematical Modelling of Natural Phenomena*, vol. 5, no. 6, pp. 196–242, 2010.

[35] W. J. Edmunds, G. F. Medley, and D. J. Nokes, “Vaccination against hepatitis B virus in highly endemic areas: waning vaccine-induced immunity and the need for booster doses,” *Transactions of the Royal Society of Tropical Medicine and Hygiene*, vol. 90, no. 4, pp. 436–440, 1996.

[36] J. Malinzi, R. Ouifki, A. Eladdadi, D. F. M. Torres, and K. A. J. White, “Enhancement of chemotherapy using oncolytic: mathematical and optimal control analysis,” *Mathematical Biosciences and Engineering*, vol. 15, no. 6, pp. 1435–1463, 2018.