Research Article

Coinfection Dynamics of HBV-HIV/AIDS with Mother-to-Child Transmission and Medical Interventions

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In this study, we analyzed the effect of mother-to-child transmission (MTCT) of hepatitis B virus (HBV) and human immunodeficiency virus (HIV) on the transmission dynamics of their coinfection to make a recommendation based on reasons to public health sector, policy makers, and programme implementers. We proved that the solutions of the sub and full models are positive and bounded. The effective reproduction numbers of the models are derived using the next generation matrix method. The disease-free and endemic equilibria of the submodels and the coinfection model are computed, and the stability of those equilibria is analyzed using Routh-Hurwitz criteria after computing the associated effective reproduction numbers. We performed a sensitivity analysis to show the influence of different parameters on the effective reproduction number of HBV-HIV/AIDS coinfection model, and we identified the most sensitive parameters are τ_2 and α_1, which are the rate of MTCT of HIV and treatment rate for HBV infected class, respectively. The numerical simulation of the model is done using MATLAB and the findings from the simulations are discussed. From the results of numerical simulations, we observed that an increase in the rates of MTCT of HBV and HIV exacerbated HBV-HIV/AIDS coinfection, while a decrease in the rates of MTCT of these infections would decline the number of cases, minimize the spread, and help to eliminate HBV-HIV/AIDS coinfection from the society gradually.

1. Introduction

Hepatitis B is a liver infection caused by HBV. It affects everyone including expectant women and their newly born children globally. The main routes of transmission are vertical transmission (MTCT), contact with infected blood, semen, and other bodily fluids [1, 2]. MTCT, by which HBV is transmitted from infected mothers to their infants, during pregnancy, at birth, or postnatally (during childcare or through breast milk), contributes significantly to the persistence of the high number of HBV carriers globally [3–6]. The chance of MTCT amongst infants born to HBV infected mothers is ranges from 10 to 40% in HBeAg negative mothers and greater than 90% in HBeAg positive mothers

with HBV deoxyribonucleic acid (DNA) levels greater than 200,000 IU/ml [7]. Elimination of MTCT of HBV has been identified as a global public health priority. To prevent MTCT of HBV, World Health Organization advises universal immunization with at least three doses of HBV vaccine as first-line prevention against around birth infection for all newborns. Newborns should get their first dose of the vaccine along with hyperimmune hepatitis B immunoglobulin (HBIG) at birth [6]. However, in many resource-constrained countries, the initial dose is provided as a pentavalent vaccine in the EPI (Expanded Program on Immunization) program at 6 weeks of age, and therefore babies from birth to six weeks of age are not protected against vertical transmission [8].

HIV also affects everyone including expectant women and their newly born children globally. It is a causative agent for acquired immunodeficiency syndrome (AIDS) which damages both humoral and cellular immunity resulting in increased susceptibility of the host to a wide range of [9, 10]. The main routes of transmission of HIV are the same as that of HBV [9, 11, 12]. MTCT, by which HIV is transmitted from infected mothers to their infants, contributes significantly to the persistence of the high numbers of HIV carriers globally [13, 14]. Without prevention, the chance of MTCT of HIV is 15–25% in developed countries and 25–35% in developing countries [15]. Primary HIV protection, the minimization of unintended pregnancies, good access to HIV diagnosis and counselling, the start of treatment, viral suppression for mothers living with HIV, safe delivery practices, good infant feeding practices, and access to postnatal antiretroviral (ARV) prophylaxis for infants are all important factors in the prevention of MTCT of HIV [16, 17]. Coinfection increases the morbidity and mortality beyond those caused by either infection alone. People coinfected with both infections 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 either infection alone [18, 19]. Thus, it is important to consider and study the effect of MTCT which contributes significantly to the persistence of the high number of HBV and HIV carriers globally.

In the field of epidemiology, models are important tools in the study of extremely complex communicable diseases. Recently, numerous scholars are concentrated on the study of the transmission dynamics of a particular and coinfection of various diseases and how the diseases might be effectively managed and potentially eliminated. Wodajo and Mekonnen [20] proposed a mathematical model to study the dynamics of hepatitis B virus infection under the administration of vaccination and treatment, where HBV infection is transmitted in two ways through vertical and horizontal transmission. Their results show that the combined efforts of vaccination, effective treatment, and interruption of transmission make elimination of the infection plausible and may eventually lead to the eradication of the virus. Omondi et al. [21] developed a mathematical model describing the dynamics of HIV transmission by incorporating sexual orientation of individuals. They investigated the effect of the introduction of preexposure prophylaxis (PrEP) on the dynamics of the HIV. The results show that the introduction of PrEP has a positive effect on the limitation of the spread of HIV. Melese and Alemneh [22] developed a transmission dynamics model for VL–HIV coinfection by splitting the population into ten compartments. From the result they achieved, the authors concluded that increasing the rate of visceral leishmaniasis (VL) recovery (ϕ_1), the recovery rate for VL–HIV Coinfection (ϕ_2), removing reservoirs (c_1), and minimizing the contact rate (β_h) are important in controlling the transmission of individual and coinfection disease of VL and HIV. Teklu and Rao [23] proposed and analyzed a realistic compartmental mathematical model on the spread and control of HIV/AIDS-pneumonia coepidemic incorporating pneumonia vaccination and treatment for both infections at each infection stage in a population. The authors showed

that pneumonia vaccination and treatment against disease have positive effect in decreasing pneumonia and coepidemic disease expansion and reducing the progression rate of HIV infection to the AIDS stage.

However, mathematical models formulated to study the codynamics of HBV and HIV/AIDS are few in the literature, although the coexistence between the two infections exists. In our review of the literature, we found only three mathematical models of HBV-HIV/AIDS coinfection and we used them as the basis for our developed model as follows: Bowong et al. [24] developed a deterministic model for HBV and HIV coinfection. In their model, the authors did not consider treatment for all infections rather intended only on prevention against HBV infection. Nampala et al. [25] formulated an epidemiological model of hepatotoxicity and antiretroviral healing effects in HBV-HIV coinfection. The authors used numerical techniques to study the healing as well as toxic effect of the recently used HBV-HIV therapy, and as a result formulated a desirable combination for treating the effect of HBV and HIV infections. These authors concentrated on hepatotoxicity and treatment effects in their model rather than including protection strategies against all infections. The combined effect of vaccination and treatment on the transmission dynamics of HBV-HIV/AIDS coinfection has been studied by Endashaw and Mekonnen [26]. Their model subdivides the total population into nine mutually exclusive compartments depending on the disease status. The model has no recovery compartment for those who are recovered from HBV infection naturally. Moreover, the authors did not consider the effect of MTCT of both HBV and HIV infections on the transmission dynamics of the coinfection of these two viruses rather focusing on the horizontal transmission alone. They obtained results showing 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 are very essential to control the spread of HBV-HIV/AIDS coinfection as an important public health problem. We motivated by a study conducted by Endashaw and Mekonnen [26] and extended it by considering a recovery compartment for those individuals who are recovered from HBV infection naturally and MTCT of both HBV and HIV which are not considered in their study. Moreover, to the authors' knowledge, none of the authors of these existing coinfection models have considered the effect of MTCT of hepatitis B virus and HIV on the coinfection dynamics of HBV-HIV/AIDS. As a result, we examine the effect of MTCT of HBV and HIV on the transmission dynamics of the coinfection of these two viruses with medical interventions. This extended model will be used to evaluate the effect of MTCT of both infections on their codynamics model with preventive (vaccination) and therapeutic (treatment) intervention strategies.

2. Baseline Model Description and Formulation

We grouped the entire population $N(t)$ into ten compartments as those who are susceptible to both diseases ($P_1(t)$), immune to HBV after vaccination ($P_2(t)$), only infected with HBV ($P_3(t)$), only infected with HIV ($P_4(t)$), infected with both HBV and HIV ($P_5(t)$), those who are receiving

HBV treatment $(P_6(t))$, those who are receiving HIV treatment $(P_7(t))$, HBV-HIV/AIDS treated section $(P_8(t))$, individuals with suppressed viral load $(P_9(t))$, and HBV recovered class $(P_{10}(t))$. The entire population at time t, denoted by N(t), is given by

$$N(t) = P_1(t) + P_2(t) + P_3(t) + P_4(t) + P_5(t) + P_6(t) \\ + P_7(t) + P_8(t) + P_9(t) + P_{10}(t). \quad (1)$$

(i) The newly immunized individuals enter into the immunized compartment P_2 at a constant recruitment rate Λ

(ii) The $P_4(t)$ compartment rises because of HIV infected newborns at a rate $(1 - d_2)\tau_2 P_4$ and the transfer of individuals from $P_1(t)$ and $P_2(t)$ compartments by the infection rate λ_2. It is supposed that an accurate contact of P_4 compartment with both P_1 and P_2 classes lead to the birth of infected neonates with a rate of τ_2. Of these newly born but infected neonates a fraction d_2 dies during the birth due to the infection and the remaining complementary fraction $(1 - d_2)$ enter into P_4 class. Similarly, the $P_3(t)$ class increases due to HBV infected newborns at a rate $(1 - d_1)\tau_1 P_3$ and the entering of individuals from $P_2(t)$ compartment by the force of infection λ_1. It is assumed that the real contact among P_3 and P_2 classes lead to the birth of infected neonates with a rate of τ_1. Of these newly born but infected neonates a fraction d_1 dies during birth due to the infection and the remaining complementary fraction $(1 - d_1)$ enters into P_3 class

(iii) The combination among people is the same and become infected with HBV and HIV by the force of infection λ_1 and λ_2, respectively

(iv) The efficacy of the vaccine wanes out at a rate ω and the natural recovery rate of individuals from HBV infection in HBV-only infected class is r_2

(v) The susceptible class rises due to the recruitment of unimmunized individuals, the transfer of naturally recovered individuals from recovered class, and the transfer of individuals whose vaccine efficacy wanes

(vi) Individuals in P_1 and P_2 compartments get HIV infection by the force of infection λ_1, whereas individuals in P_2 acquire HBV infection by the force of infection λ_2. Individuals in HBV-only infected class get HIV infection by force of infection λ_2 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 λ_1 within a brief

period of time before an infection with the first strain (HIV) has been established and an immune response has developed.

(vii) Individuals in P_3 and P_5 progress to P_6 and P_8 classes, respectively, after getting treatment. The proportion ζ of individuals in P_4 progress to P_7 due to the treatment rate α_2 and the remaining proportion $(1 - \zeta)$ of individuals progress to P_5 by force of infection λ_1 before an infection with the first strain (HIV) has been established and an immune response has developed

(viii) Individuals who are recovered from HBV infection naturally in HBV-HIV/AIDS coinfected class enter in to HIV-only infected class at a rate r_1

(ix) Since the effective treatment reduces the viral load of the infected individuals in P_6, P_7, and P_8 classes to the required undetectable level, individuals in these compartments progress to a suppressed viral load class at the progress rate θ_1, θ_2, and θ_3, respectively

(x) If the vaccine efficacy not wanes, people who are immunized against HBV infection are susceptible to HIV-only

(xi) 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

(xii) Recovered class increases due to a transfer of naturally recovered individuals from HBV infected class and decreases due to the progress of HBV recovered individuals to the susceptible class

(xiii) Those persons who get rid of HBV infection due to natural immunity in P_3 class enter into the recovered class at rate of r_2, but they do not susceptible to reacquiring the infection because they developed antibodies that protect them from HBV infection for the rest of their lives.

Using Table 1 and the model assumptions, the flow diagram for the transmission dynamics of the full model is given by:

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

$$\frac{dP_1}{dt} = (1 - \eta)\Lambda + \omega P_2 + r_3 P_{10} - (\lambda_1 + \lambda_2 + d_0)P_1,$$

$$\frac{dP_2}{dt} = \eta\Lambda - (\lambda_2 + \omega + d_0)P_2,$$

$$\frac{dP_3}{dt} = (1 - d_1)\tau_1 P_3 + \lambda_1 P_1 - (r_2 + \alpha_1 + \lambda_2 + d_3 + d_0)P_3,$$

$$\frac{dP_4}{dt} = (1 - d_2)\tau_2 P_4 + \lambda_2(P_1 + P_2) + r_1 P_5$$
$$- (\zeta\alpha_2 + (1 - \zeta)\lambda_1 + d_4 + d_0)P_4,$$

$$\frac{dP_5}{dt} = (1 - \zeta)\lambda_1 P_4 + \lambda_2 P_3 - (r_1 + \alpha_3 + d_5 + d_0)P_5,$$

$$\frac{dP_6}{dt} = \alpha_1 P_3 - (\theta_1 + d_0)P_6,$$

$$\frac{dP_7}{dt} = \zeta\alpha_2 P_4 - (\theta_2 + d_0)P_7, \qquad (2)$$

$$\frac{dP_8}{dt} = \alpha_3 P_5 - (\theta_3 + d_0)P_8,$$

$$\frac{dP_9}{dt} = \theta_1 P_6 + \theta_2 P_7 + \theta_3 P_8 - d_0 P_9,$$

$$\frac{dP_{10}}{dt} = r_2 P_3 - (r_3 + d_0)P_{10},$$

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

$$\lambda_1 = h_1 \frac{(P_3 + P_5)}{N}. \qquad (3)$$

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

$$\lambda_2 = h_2 \frac{((+P_4 + P_5)}{N}, \qquad (4)$$

with initial conditions,

$$P_1(0) > 0, P_2(0) > 0, P_3(0) > 0, P_4(0) > 0, P_5(0) > 0, P_6(0)$$
$$> 0, P_7(0) > 0, P_8(0) > 0, P_9(0) > 0, \text{ and } P_{10}(0) > 0. \qquad (5)$$

2.1. Positivity of the Solutions and Boundedness of the Solution Region of the Full Model (1)

Theorem 1. *At the initial conditions (5), the solutions of the dynamical system (2) are nonnegative for time $t > 0$ in the region*

$$\Omega = \left\{ (P_1, P_2, P_3, P_4, P_5, P_6, P_7, P_8, P_9, P_{10}) \in \mathbb{R}^{10}_+. \right\} \qquad (6)$$

Proof. Let us take the first differential equation from the dynamical system (2)

$$\frac{dP_1}{dt} = (1 - \eta)\Lambda + \omega P_2 + r_3 P_{10} - (\lambda_1 + \lambda_2 + d_0)P_1. \qquad (7)$$

TABLE 1: Model Parameters and their descriptions.

Parameters	Descriptions
d_0	Natural death rate
Λ	Rate of recruitment of individuals into immunized compartment
d_1	Probability of death of newborns infected with HBV at birth
d_2	Probability of death of newborns infected with HIV at birth
τ_1	Rate of MTCT of HBV
τ_2	Rate of MTCT of HIV
d_3	Mortality rate related to hepatitis B virus
d_4	Death rate due to HIV/AIDS
d_5	Mortality rate due to coinfection
α_1	Rate of treatment for hepatitis B virus infected individuals
α_2	Rate of treatment for ζ proportion of HIV/AIDS patients
α_3	Rate of treatment for those who are coinfected
λ_1	Infection related with hepatitis B virus
λ_2	Infection related with HIV/AIDS
r_1	A rate of return of HBV infected population from P_3 class naturally to former status
r_2	A rate of return of HBV infected population from coinfected class to former status
η	Fraction of vaccinated population
ζ	Fraction of HIV/AIDS patients entering to treatment compartment.
θ_1	The transfer rate of HBV patients to suppressed viral load compartment after treatment
θ_2	The transfer rate of HIV/AIDS patients to suppressed viral load class after treatment
θ_3	The transfer rate of coinfected population to suppressed viral load class after treatment
h_1	Contact rate for HBV
h_2	Contact rate for HIV
ω	The waning rate of vaccine efficacy
r_3	The transfer rate of HB recovered individuals to susceptible class

A separable first order ordinary differential equation of (7) for the variable P_1 is

$$\frac{dP_1}{dt} + (\lambda_1 + \lambda_2 + d_0)P_1 = (1 - \eta)\Lambda + \omega P_2 + r_3 P_{10}. \qquad (8)$$

Now taking the integrating factor $e^{\int (\lambda_1 + \lambda_2 + d_0)dt}$, we get

$$P_1(t) = e^{-(\lambda_1 + \lambda_2 + d_0)t} \left[((1 - \eta)\Lambda + \omega P_2 + r_3 P_{10}) \int e^{(\lambda_1 + \lambda_2 + d_0)t} dt \right]. \qquad (9)$$
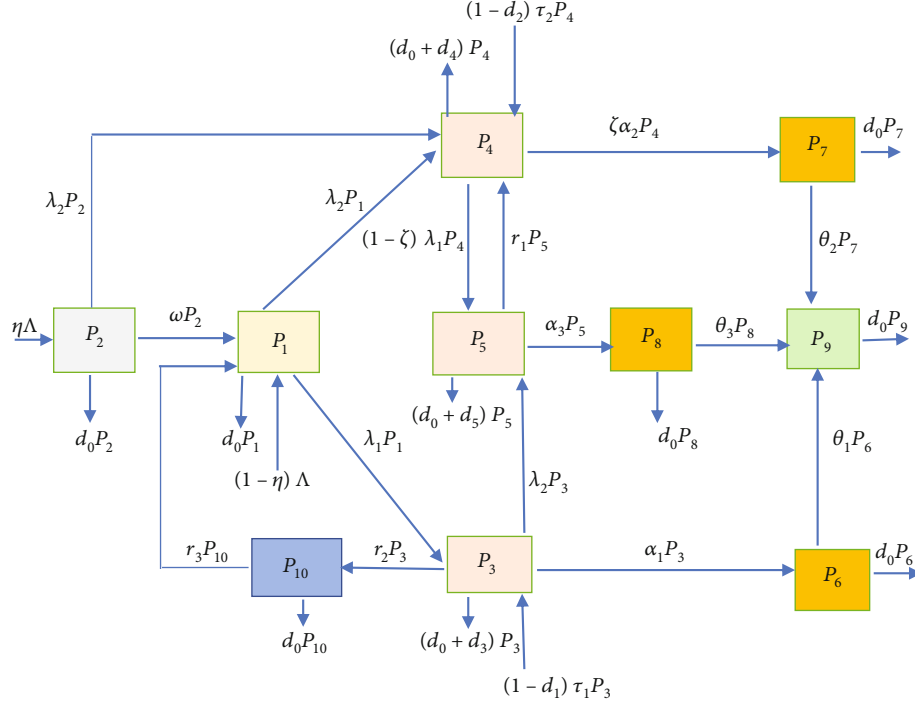
FIGURE 1: Flow diagram of the full model.

Integrating and simplifying (9) gives us

$$P_1(t) = C_1 e^{-(\lambda_1 + \lambda_2 + d_0)t} + \left(\frac{(1-\eta)\Lambda + \omega P_2 + r_3 P_{10}}{\lambda_1 + \lambda_2 + d_0}\right). \quad (10)$$

where C_1 is constant. □

After rearranging and computing (10) at $(t) = 0$ we get

$$C_1 = P_1(0) - \left(\frac{(1-\eta)\Lambda + \omega P_2 + r_3 P_{10}}{\lambda_1 + \lambda_2 + d_0}\right) \quad (11)$$

Substituting (11) in (10) we get

$$P_1(t) = P_1(0)e^{-(\lambda_1 + \lambda_2 + d_0)t} + \frac{(1-\eta)\Lambda + \omega P_2 + r_3 P_{10}}{\lambda_1 + \lambda_2 + d_0} \\ \cdot \left(1 - e^{-(\lambda_1 + \lambda_2 + d_0)t}\right). \quad (12)$$

which is the solution of (7).

Since, $P_1(0) > 0$, $e^{-(\lambda_1 + \lambda_2 + d_0)t} > 0$ and $(1-\eta)\Lambda + \omega P_2 + r_3 P_{10}/\lambda_1 + \lambda_2 + d_0 > 0$ for $t \geq 0$, equation is positive. Therefore, $P_1(t) = P_1(0)e^{-(\lambda_1 + \lambda_2 + d_0)t} + (1-\eta)\Lambda + \omega P_2 + r_3 P_{10}/\lambda_1 + \lambda_2 + d_0(1 - e^{-(\lambda_1 + \lambda_2 + d_0)t}) > 0$

Following the same procedure, $P_2(t) > 0$, $P_3(t) > 0$, $P_4(t) > 0$, $P_5(t) > 0$, $P_6(t) > 0$, $P_7(t) > 0$, $P_8(t) > 0$, $P_9(t) > 0$, and $P_{10}(t) > 0$. Hence, from the above proof, we can conclude that whenever the initial values of the systems are all positive, then all the solutions of the dynamical system (2) are positive.

Theorem 2. *The dynamical system (2) is positively invariant in the region*

$$\Omega = \left\{(P_1, P_2, P_3, P_4, P_5, P_6, P_7, P_8, P_9, P_{10}) \in \mathbb{R}^{10}_+ : N \leq \frac{\Lambda}{d_0}\right\} \quad (13)$$

Proof. The dynamics of total population $N(t)$ with respect to time t is computed as

$$\frac{dN}{dt} = \Lambda - d_0 N + (\tau_1 - (d_1\tau_1 + d_3))P_3 \\ + (\tau_2 - (d_2\tau_2 + d_4))P_4 - d_5 P_5 \quad (14)$$

In the absence of mortality due to HBV infection, HIV/AIDS, and HBV-HIV/AIDS coinfection and comparing both sides of the equation (14) using standard comparison theorem,

$$\frac{dN}{dt} \leq \Lambda - dN. \quad (15)$$

The solution for the inequality (15) after some steps becomes

$$N(t) \leq N(0)e^{-d_0 t} + \frac{\Lambda}{d_0}\left(1 - e^{-d_0 t}\right). \quad (16)$$

As $t \longrightarrow \infty$, the population size $N(t) \longrightarrow \Lambda/d_0$ whenever $N(0) \leq \Lambda/d_0$. This implies

$$0 \le N(t) \le \frac{\Lambda}{d_0}. \tag{17}$$

The inequality (17) shows that all the feasible solutions of the components of a dynamical system (2) with initial conditions enter the region $\Omega = \{(P_1, P_2, P_3, P_4, P_5, P_6, P_7, P_8, P_9, P_{10}) \in \mathbb{R}^{10}_+ : N \le \Lambda/d_0\}$ for all $t > 0$. Thus, the region Ω is positively invariant. $\square$

3. Analysis of the HBV and HIV/AIDS Submodels

To lay down the foundation for the analysis of model (2), it is important to observe the dynamics of the submodels in advance.

3.1. HBV-Only Submodel. From the model (2), the HBV-only submodel is obtained by setting $P_4 = P_5 = P_7 = P_8 = P_9 = P_{10} = 0$. Thus, we have the following dynamical system

$$\frac{dP_1}{dt} = (1 - \eta)\Lambda + \omega P_2 + r_3 P_{10} - (\lambda_1 + d_0)P_1,$$

$$\frac{dP_2}{dt} = \eta\Lambda - (\omega + d_0)P_2,$$

$$\frac{dP_3}{dt} = (1 - d_1)\tau_1 P_3 + \lambda_1 P_1 - (r_2 + \alpha_1 + d_3 + d_0)P_3, \tag{18}$$

$$\frac{dP_6}{dt} = \alpha_1 P_3 - d_0 P_6,$$

$$\frac{dP_{10}}{dt} = r_2 P_3 - (r_3 + d_0)P_{10},$$

where the force of infection is

$$\lambda_1 = h_1 \frac{P_3}{N_1}. \tag{19}$$

with initial conditions,

$$P_1(0) > 0, P_2(0) > 0, P_3(0) > 0, P_6(0) > 0, \text{ and } P_{10}(0) > 0. \tag{20}$$

The total population of the dynamical system (18) is given by

$$N_1(t) = P_1(t) + P_2(t) + P_3(t) + P_6(t) + P_{10}(t). \tag{21}$$

3.1.1. Positivity of the Solutions and Boundedness of the Solution Region of HBV-Only Submodel. As we did in subsection 2.1, it is important to prove that all the solutions of the dynamical system (18) with positive initial conditions remain positive and the solution region is bounded. This fact is verified in theorem 3 and 4 as follows:

Theorem 3. *The solutions of the dynamical system (18) with positive initial conditions are positive for time $t \ge 0$ in the region*

$$\Omega_1 = \left\{(P_1, P_2, P_3, P_6, P_{10}) \in \mathbb{R}^5_+\right\}. \tag{22}$$

Proof. Taking $dP_1/dt = (1 - \eta)\Lambda + \omega P_2 + r_2 P_3 - (\lambda_1 + d_0)P_1$ from the dynamical system (18) we get

$$\frac{dP_1}{dt} + (\lambda_1 + d_0)P_1 = (1 - \eta)\Lambda + \omega P_2 + r_2 P_3. \tag{23}$$

Equation (23) is a separable first order ordinary differential equation for the variable P_1. After integrating and some simplifications, we get the solution

$$P_1(t) = P_1(0)e^{-(\lambda_1 + d_0)t} + \frac{(1 - \eta)\Lambda + \omega P_2 + r_2 P_3}{\lambda_1 + d_0}\left(1 - e^{-(\lambda_1 + d_0)t}\right). \tag{24}$$

Since, $P_1(0) > 0$, $e^{-(\lambda_1 + d_0)t} > 0$ and $(1 - \eta)\Lambda + \omega P_2 + r_2 P_3$ $/\lambda_1 + d_0 > 0$ for $t \ge 0$, the solution we found is positive. Following the same procedure, $P_2(t) > 0$, $P_3(t) > 0$, $P_6(t) > 0$, and $P_{10}(t) > 0$. Therefore, the solutions of the dynamical system are positive whenever the initial values positive. $\square$

Theorem 4. *The dynamical system (18) is positively invariant in the region*

$$\Omega_1 = \left\{(P_1, P_2, P_3, P_6, P_{10}) \in \mathbb{R}^5_{>+} : N_1 \le \frac{\Lambda}{d_0}\right\}. \tag{25}$$

Proof. The dynamics of total population $N_1(t)$ with respect to time t is computed as

$$\frac{dN_1}{dt} = \Lambda - d_0 N_1 - d_3 P_3. \tag{26}$$

In the absence of mortality due to HBV infection and comparing both sides of equation (26) using standard comparison theorem, we obtain

$$\frac{dN_1}{dt} \le \Lambda - d_0 N_1. \tag{27}$$

After some steps the solution for equation (27) is

$$N_1(t) \le N_1(0)e^{-d_0 t} + \frac{\Lambda}{d_0}\left(1 - e^{-d_0 t}\right). \tag{28}$$

As $t \longrightarrow \infty$, the population size $N_1(t) \le \Lambda/d_0$

This implies $0 \le N_1(t) \le \Lambda/d_0$, which shows all the feasible solutions of the model (18) with initial conditions enter the region $\Omega_1 = \{(P_1, P_2, P_3, P_6, P_{10}) \in \mathbb{R}^5_+ : N_1 \le \Lambda/d_0\}$.

Thus, the region Ω_1 is bounded. $\square$

3.1.2. Disease-Free Equilibrium Point (DFE) of HBV-Only Submodel. At DFE point it is assumed that there is no disease in the population. The DFE of the dynamical system (18) represented by E^0_1 is obtained by setting the right-hand side of the dynamical system equal to zero, providing that $\{P_3 = P_6 = 0\}$. After some simple calculation, E^0_1 is equal to

$$E^0{}_1 = \left(\frac{\Lambda(\omega + d_0(1 - \eta))}{d_0(\omega + d_0)}, \frac{\eta \Lambda}{\omega + d_0}, 0, 0, 0 \right). \tag{29}$$

3.1.3. Effective Reproduction Number of HBV-Only Submodel.
The effective reproduction number of hepatitis B infected individuals, denoted by $R^1{}_{eff}$ that of the dynamical system (18), 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 [27, 28]. It is obtained by taking the spectral radius of the matrix $FV^{-1} = (\rho(FV^{-1})) = [\partial \mathscr{F}_i(E^0{}_1)/\partial x_j][\partial v_i(E^0{}_1)/\partial x_j]^{-1}$ of the dynamical system (18) [28, 29], where $\mathscr{F}_i$ is the rate of appearance of a new infection in the compartment i, v_i is the transfer of infection from one compartment i to another, and $E^0{}_1$ is the disease-free equilibrium point.

The corresponding Jacobian matrices of $\mathscr{F}$ and $\mathscr{V}$ computed at DFE point $E^0{}_1$, respectively, are given as follows:

$$F = \left[\frac{(1 - d_1)\tau_1(\omega + d_0) + h_1(\omega + d_0(1 - \eta))}{\omega + d_0} \right], \tag{30}$$

and

$$V = [r_2 + \alpha_1 + d_3 + d_0]. \tag{31}$$

From this, it follows that

$$R^1{}_{eff} = \rho(FV^{-1}) = \frac{(1 - d_1)\tau_1(\omega + d_0) + h_1(\omega + d_0(1 - \eta))}{(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0)}. \tag{32}$$

3.1.4. Local Stability Analysis of the Disease-Free Equilibrium Point of HBV-Only Submodel

Theorem 5. *The disease-free equilibrium point $E^0{}_1 = (\Lambda(\omega + d_0(1 - \eta))/d_0(\omega + d_0), \eta\Lambda/\omega + d_0, 0, 0, 0)$ of the dynamical system (18) is locally asymptotically stable if the effective reproduction number $R^1{}_{eff} < 1$.*

Proof. The Jacobian matrix of the dynamical system (18) at the DFE point $E^0{}_1$ is

$$J_1(E^0{}_1) = \begin{bmatrix} -d_0 & \omega & 0 & 0 & r_3 \\ 0 & -(\omega + d_0) & 0 & 0 & 0 \\ 0 & 0 & -(r_2 + \alpha_1 + d_3 + d_0) & 0 & 0 \\ 0 & 0 & \alpha_1 & -d_0 & 0 \\ 0 & 0 & r_2 & 0 & -(r_3 + d_0) \end{bmatrix}. \tag{33}$$

After some steps, the roots of (33) are

$$\lambda_1 = \lambda_2 = -d_0, \lambda_3 = -(\omega + d_0), \lambda_4 = -(r_3 + d_0),$$
$$\lambda_5 = -(r_2 + \alpha_1 + d_3 + d_0). \tag{34}$$

This shows all the eigenvalues of the Jacobean matrix have negative real parts when $R^1{}_{eff} < 1$

Hence, the disease-free equilibrium $E^0{}_1 = (\Lambda(\omega + d_0(1 - \eta))/d_0(\omega + d_0), \eta\Lambda/\omega + d_0, 0, 0, 0)$ of the dynamical system (18) is locally asymptotically stable if the effective reproduction number $R^1{}_{eff} < 1$ and unstable otherwise. □

3.1.5. Global Stability Analysis of the Disease-Free Equilibrium Point of HBV-Only Submodel

Theorem 6. *The disease-free equilibrium point $E^0{}_1 = ((\Lambda(\omega + d_0(1 - \eta))/d_0(\omega + d_0), \eta\Lambda/\omega + d_0, 0, 0, 0))$ of the dynamical system (18) is globally asymptotically stable in the feasible region Ω_1 if the effective reproduction number $R^1{}_{eff} < 1$.*

Proof. Consider the following LaSalle-Lyapunov candidate function:

$$f_1 = u_1 P_3, \tag{35}$$

where $u_1 = 1/r_2 + \alpha_1 + d_3 + d_0$.
The time derivative of (35) along the solution path yields

$$\dot{f}_1 = u_1 \left((1 - d_1)\tau_1 P_3 + \frac{h_1 P_1 P_3}{N_1} - (r_2 + \alpha_1 + d_3 + d_0)P_3 \right)$$
$$= u_1 \left((1 - d_1)\tau_1 + \frac{h_1 P_1}{N_1} - (r_2 + \alpha_1 + d_3 + d_0) \right) P_3 \tag{36}$$

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 $E^0{}_1 = (r_2 + \alpha_1 + d_3 + d_0/r_2 + \alpha_1 + d_3 + d_0, \eta\Lambda/\omega + d_0), 0, 0, 0$. with $R^1{}_{eff} = ((1 - d_1)\tau_1(\omega + d_0) + h_1(\omega + d_0(1 - \eta)))/(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0), \dot{f}_1$ can be simplified as follows:

$$\dot{f}_1 \le u_1 \left((1 - d_1)\tau_1 + \frac{h_1 P^0{}_1}{N_1{}^0} - (r_2 + \alpha_1 + d_3 + d_0) \right) P_3. \tag{37}$$

Substituting $h_1 = (R^1{}_{eff}(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0) - (\omega + d_0)(1 - d_1)\tau_1/\omega + d_0(1 - \eta))$ in (37) and simplification gives us $\dot{f}_1 \le u_1(r_2 + \alpha_1 + d_3 + d_0)(R^1{}_{eff} - 1)P_3$. After substituting u_1 we get $\dot{f}_1 \le (R^1{}_{eff} - 1)P_3$.

This shows that $\dot{f}_1 < 0$ when $R^1{}_{eff} < 1$. Furthermore, $\dot{f}_1 = 0$ if and only if $P_3 = 0$ or $R^1{}_{eff} = 1$ and $((\Lambda(\omega + d_0(1 - \eta))/d_0(\omega + d_0)), (\eta\Lambda/\omega + d_0), 0, 0, 0)$ is the only singleton set in $\{(P_1, P_2, P_3, P_6, P_{10}) \in \Omega_1 : \dot{f}_1 = 0\}$. Thus, by LaSalle's invariance principle [30], the DFE $E^0{}_1 = ((\Lambda(\omega + d_0(1 - \eta))/d_0(\omega$

$+ d_0)), (\eta \Lambda / \omega + d_0), 0, 0, 0)$ is globally asymptotically stable if $R^1_{\text{eff}} < 1$. □

3.1.6. Existence of Endemic Equilibrium (EE) of HBV-Only Submodel. Suppose that $E^*_1 = (P_1^*, P_2^*, P^*_3, P^*_6, P^*_{10})$ be an arbitrary EE equilibrium point of HB-only submodel (18), which occurs when the disease persists in the society. After long steps we obtained the endemic equilibrium point $E^*_1 = (P_1^*, P_2^*, P^*_3, P^*_6, P^*_{10})$.

where

$$P_1^* = \frac{\Lambda(r_3 + d_0)(\omega + d_0(1 - \eta))((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)}{\lambda_1^*(\omega + d_0)((r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3) + d_0(\omega + d_0)(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)},$$

$$P_2^* = \frac{\eta \Lambda}{\omega + d_0},$$

$$P_3^* = \frac{\lambda_1^* \Lambda(r_3 + d_0)(\omega + d_0(1 - \eta))}{\lambda_1^*(\omega + d_0)((r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3) + d_0(\omega + d_0)(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)},$$

$$P^*_6 = \frac{\alpha_1 \lambda_1^* \Lambda(r_3 + d_0)(\omega + d_0(1 - \eta))}{\lambda_1^* d_0(\omega + d_0)((r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3) + d_0^2(\omega + d_0)(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)},$$

(38)

and

$$P^*_{10} = \frac{r_2 \lambda_1^* \Lambda(r_3 + d_0)(\omega + d_0(1 - \eta))}{\lambda_1^*(r_3 + d_0)(\omega + d_0)((r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3) + d_0(\omega + d_0)(r_3 + d_0)^2((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)}$$

(39)

The associated force of infection of the dynamical system (18) is given by

$$\lambda_1^* = \frac{h_1 P_3^*}{N_1^*}.$$

(40)

where

$$N_1^* = \frac{\lambda_1^*(\Lambda/d_0)(((\omega + d_0(1 - \eta)))((r_3 + d_0)(\alpha_1 + d_0) + d_0 r_2) + \eta d_0(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3 d_0) + \Lambda(r_3 + d_0)(\omega + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)}{\lambda_1^*(\omega + d_0)((r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3) + d_0(\omega + d_0)(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)}.$$

(41)

After substituting P^*_3 and N_1^* in (40) and simplifying it we got

$$\lambda_1^* = \frac{d_0(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0)(R^1_{eff} - 1)}{(\alpha_1 + d_0)(\omega + d_0(1 - \eta)) + d_0 \eta(\alpha_1 + d_3 + d_0) - d_0 \eta(1 - d_1)\tau_1}.$$

(42)

Equation (42) shows that $\lambda_1^* > 0$ if $R^1_{\text{eff}} > 1$. Hence, the endemic equilibrium point E^*_1 of HBV-only submodel (18) exists whenever $R^1_{\text{eff}} > 1$.

3.1.7. Local Stability of the Endemic Equilibrium Point of HBV-Only Submodel

Theorem 7. *The endemic equilibrium point $E^*_1 = (P_1^*, P_2^*, P^*_3, P^*_6, P^*_{10})$ of the model (18) is locally asymptotically stable if $R^1_{eff} > 1$.*

Proof. The Jacobian matrix of (18) at the endemic equilibrium E^*_1 is

$$J_1(E^*_1) = \begin{pmatrix} -(E_1 + d_0) & \omega & -E_2 & 0 & r_3 \\ 0 & -(\omega + d_0) & 0 & 0 & 0 \\ E_1 & 0 & -(E_3 + E_2) & 0 & 0 \\ 0 & 0 & \alpha_1 & -d_0 & 0 \\ 0 & 0 & r_2 & 0 & -(r_3 + d_0) \end{pmatrix}, \quad (43)$$

where

$$E_1 = \frac{h_1 P^*_3}{N_1{}^*} = \frac{\Lambda(\omega + d_0)(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)(R^1{}_{eff} - 1)}{(\Lambda/d_0)((\omega + d_0(1 - \eta))((r_3 + d_0)(\alpha_1 + d_0) + d_0 r_2) + \eta d_0(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3 d_0)},$$

$$E_2 = \frac{h_1 P_1{}^*}{N_1{}^*}$$

$$= \frac{h_1(\omega + d_0(1 - \eta))((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)((\omega + d_0(1 - \eta))((r_3 + d_0)(\alpha_1 + d_0) + d_0 r_2) + \eta d_0(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3 d_0)}{(((\omega + d_0(1 - \eta)))((r_3 + d_0)(\alpha_1 + d_0) + d_0 r_2) + \eta d_0(r_3 + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1) - r_2 r_3 d_0 + \Lambda(r_3 + d_0)(\omega + d_0)((r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1)(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0)(R^1{}_{eff} - 1))},$$

$$E_3 = (1 - d_1)\tau_1 - (r_2 + \alpha_1 + d_3 + d_0). \tag{44}$$

Let λ_i be the eigenvalues of $J_1(E^*{}_1)$ where $i = 1, 2, 3, 4, 5$. The corresponding characteristic equation of (43) is

$$(\lambda + d_0)(\lambda + (\omega + d_0))(z_3\lambda^3 + z_2\lambda^2 + z_1\lambda + z_0) = 0, \tag{45}$$

This implies the first two eigenvalues of (45) are $\lambda_1 = -d_0 < 0$ and $\lambda_2 = -(\omega + d_0) < 0$. But, the algebraic sign of the remaining three eigenvalues is determined using Routh–Hurwitz stability criterion from the characteristic polynomial

$$z_3\lambda^3 + z_2\lambda^2 + z_1\lambda + z_0 = 0, \tag{46}$$

where
$z_3 = 1 > 0$, $z_2 = E_1 + E_2 + E_3 + r_3 + 2d_0$
$z_1 = (E_1 + d_0)(E_2 + E_3 + r_3 + d_0) + (E_2 + E_3)(r_3 + d_0)$,
and $z_0 = (E_1 + d_0)(E_2 + E_3)(r_3 + d_0)$.

The sign of the coefficients z_0, z_1, and z_2 of the characteristic polynomial $z_3\lambda^3 + z_2\lambda^2 + z_1\lambda + z_0 = 0$ is positive if $R^1{}_{eff} > 1$. In addition to that $z_1 z_2 - z_0 z_3 > 0$. Thus, by Routh–Hurwitz stability criterion the endemic equilibrium $E^*{}_1$ is stable if $R^1{}_{eff} > 1$ and unstable if $R^1{}_{eff} < 1$. □

3.1.8. Global Stability of the Endemic Equilibrium Point of HBV-Only Submodel

Theorem 8. *The endemic equilibrium point* $E^*{}_1 = (P_1{}^*, P_2{}^*, P^*{}_3, P^*{}_6, P^*{}_{10})$ *of the dynamical system (18) is globally asymptotically stable if* $R^1{}_{eff} > 1$.

Proof. Consider the following LaSalle-Lyapunov candidate function:

$$\mathscr{L}_1(P_1, P_3) = b_1(P_1 - P_1{}^*)^2 + b_2(P_3 - P_3{}^*)^2, \, b_1 > 0, b_2 > 0. \tag{47}$$

Differentiating $\mathscr{L}_1(P_1, P_3)$ with respect to time gives

$$\dot{\mathscr{L}}_1(P_1, P_3) = 2b_1(P_1 - P_1{}^*)$$
$$\cdot \left((1 - \eta)\Lambda + \omega P_2 + r_3 P_{10} - \left(\frac{h_1 P_3}{N_1} + d_0\right)P_1\right)$$
$$+ 2b_2(P_3 - P_3{}^*)$$
$$\cdot \left((1 - d_1)\tau_1 P_3 + \frac{h_1 P_3}{N_1}P_1 - (r_2 + \alpha_1 + d_3 + d_0)P_3\right). \tag{48}$$

Substituting $(1 - \eta)\Lambda + \omega P_2 + r_3 P_{10} = ((h_3 P^*{}_3/N_1{}^*) + d_0)$ $P_1{}^*$ and $(r_2 + \alpha_1 + d_3 + d_0) - (1 - d_1)\tau_1 = h_1 P^*{}_3/N_1{}^*$ with $N_1 \approx N_1{}^*$ in (48) yields:

$$\dot{\mathscr{L}}_1(P_1, P_3) = 2b_1(P_1 - P_1{}^*)\left(\frac{h_1 P^*{}_3}{N_1{}^*}P_1{}^* + d_0 P_1{}^* - \frac{h_1 P_3}{N_1}P_1 - d_0 P_1\right)$$
$$+ 2b_2(P_3 - P_3{}^*)\left(\frac{h_1 P_3}{N_1}P_1 - \frac{h_1 P_3}{N_1{}^*}P_1{}^*\right)$$
$$= -2b_1 d_0(P_1 - P_1{}^*)^2 - \frac{2b_1 h_1}{N_1}P_3(P_1 - P_1{}^*)^2$$
$$+ \frac{2h_1}{N_1}(P_3{}^* - P_3)(P_1 - P_1{}^*)(b_1 P_1{}^* - b_2 P_3)$$
$$\leq 0 \text{ if } P_3 \geq \frac{b_1 P_1{}^*}{b_2}, P_1 \geq P_1{}^*, P_3 \leq P^*{}_3. \tag{49}$$

This implies $\dot{\mathscr{L}}_1(P_1, P_3) \leq 0$, and it vanishes if and only if $P_1{}^* = P_1$, $P_3{}^* = P_3$, and $P_1{}^* = b_2 P_3/b_1$

This shows that $E^*{}_1 = (P_1{}^*, P_2{}^*, P^*{}_3, P^*{}_6, P^*{}_{10})$ is the largest compact invariant singleton set in $\{(P_1{}^*, P_2{}^*, P^*{}_3, P^*{}_6, P^*{}_{10}) \in \Omega_1 : \dot{\mathscr{L}}_1 = 0\}$

Hence, by LaSalle's invariance principle [30], the endemic equilibrium $E^*{}_1$ is globally asymptotically stable in the invariant region whenever $R^1{}_{eff} > 1$. □

3.2. HIV/AIDS-Only Submodel.
This submodel is obtained by setting $P_2 = P_3 = P_5 = P_6 = P_8 = P_9 = P_{10} = 0$. Thus, we have the following dynamical system

$$\frac{dP_1}{dt} = \Lambda - (\lambda_2 + d_0)P_1,$$

$$\frac{dP_4}{dt} = (1 - d_2)\tau_2 P_4 + \lambda_2 P_1 - (\zeta\alpha_2 + d_4 + d_0)P_4,$$

$$\frac{dP_7}{dt} = \zeta\alpha_2 P_4 - d_0 P_7, \tag{50}$$

where the force of infection is given by

$$\lambda_2 = \frac{h_2 P_4}{N_2}. \tag{51}$$

with initial conditions, $P_1(0) > 0$, $P_4(0) > 0$, and $P_7(0) > 0$

The total population of the dynamical system (50) is given by:

$$N_2(t) = P_1(t) + P_4(t) + P_7(t). \tag{52}$$

3.2.1. Positivity of the Solutions and Boundedness of the Solution Region of HIV/AIDS-Only Submodel. Here, we proved that all the solutions of the dynamical system (50) with positive initial conditions remain positive and the solution region is bounded as follows:

Theorem 9. *The solutions of the dynamical system (50) with positive initial conditions are positive for time $t \geq 0$ in the region*

$$\Omega_2 = \left\{ (P_1, P_4, P_7) \in \mathbb{R}^3_+ \right\} \tag{53}$$

Proof. Taking $dP_1/dt = \Lambda - (\lambda_2 + d_0)P_1$ from the dynamical system (50), we get

$$\frac{dP_1}{dt} + (\lambda_2 + d_0)P_1 = \Lambda. \tag{54}$$

(54) is a separable first order ordinary differential equation for the variable P_1. After some steps we got the solution

$$P_1(t) = P_1(0)e^{-(\lambda_2 + d_0)t} + \frac{\Lambda}{\lambda_2 + d_0}\left(1 - e^{-(\lambda_2 + d_0)t}\right). \tag{55}$$

Since, $P_1(0) > 0$, $e^{-(\lambda_2 + d_0)t} > 0$ and $\Lambda/(\lambda_2 + d_0) > 0$ for $t \geq 0$, the solution we found is positive. Following the same procedure, $P_4(t) > 0$ and $P_{10}(t) > 0$. Therefore, the solutions of the model (50) are positive whenever the initial values positive. $\square$

Theorem 10. *The dynamical system (50) is positively invariant in the region*

$$\Omega_2 = \left\{ (P_1, P_4, P_7) \in \mathbb{R}^3_{>+} : N_2 \leq \frac{\Lambda}{d_0} \right\}. \tag{56}$$

Proof. The dynamics of total population $N_2(t)$ with respect to time t is computed as

$$\frac{dN_2}{dt} = \Lambda - d_0 N_2 - d_4 P_4. \tag{57}$$

In the absence of mortality due to HIV infection and comparing both sides of equation (57) using standard comparison theorem, we obtain

$$\frac{dN_2}{dt} \leq \Lambda - d_0 N_2. \tag{58}$$

After some steps the solution for the inequality (58) is

$$N_2(t) \leq N(0)e^{-d_0 t} + \frac{\Lambda}{d_0}\left(1 - e^{-d_0 t}\right). \tag{59}$$

As $t \longrightarrow \infty$, the population size $N_2(t) \leq \Lambda/d_0$

This implies $0 \leq N_2(t) \leq \Lambda/d_0$ which shows all the feasible solutions of the model (50) with initial conditions enter the region

$$\Omega_2 = \left\{ (P_1, P_4, P_7) \in \mathbb{R}^3_+ : N_2 \leq \frac{\Lambda}{d_0} \right\}. \tag{60}$$

Thus, the region Ω_2 is positively invariant. $\square$

3.2.2. Existence of the Disease - Free Equilibrium Point of HIV/AIDS-Only Submodel. The disease-free equilibrium point of HIV/AIDS-only submodel (50), represented by $E^0_2 = (P_1^0, P_4^0, P_7^0)$, is obtained by setting the right-hand side of all the components of the model equal to zero, providing that $P_4 = 0$. It is equal to

$$E^0_2 = (P_1^0, P_4^0, P_7^0) = \left(\frac{\Lambda}{d_0}, 0, 0\right). \tag{61}$$

3.2.3. Effective Reproduction Numbers of HIV/AIDS-Only Submodel. In the same way that we have shown in sub section 3.1.3, the effective reproduction number of HIV/AIDS-only infected individuals is

$$R^2_{\text{eff}} = \frac{(1 - d_2)\tau_2 + h_2}{\zeta\alpha_2 + d_4 + d_0}. \tag{62}$$

3.2.4. Local Stability Analysis of the Disease-Free Equilibrium Point of HIV/AIDS-Only Submodel

Theorem 11. *The disease-free equilibrium point $E^0_2 = (\Lambda/d_0, 0, 0)$ of the dynamical system (50) is locally asymptotically stable if the effective reproduction number $R^2_{\text{eff}} < 1$.*

Proof. The Jacobean matrix of the dynamical system (50) at the DFE point $E^0_2 = (\Lambda/d_0, 0, 0)$ is

$$J_2(E^0_2) = \begin{bmatrix} -d_0 & -h_2 & 0 \\ 0 & (1 - d_2)\tau_2 + h_2 - (\zeta\alpha_2 + d_4 + d_0) & 0 \\ 0 & \zeta\alpha_2 & -d_0 \end{bmatrix}. \tag{63}$$

The corresponding characteristic equation of (63) is

$$\begin{vmatrix} -(d_0 + \lambda) & -h_2 & 0 \\ 0 & -((\zeta\alpha_2 + d_4 + d_0) - (h_2 + (1 - d_2)\tau_2) + \lambda) & 0 \\ 0 & \zeta\alpha_2 & -(d_0 + \lambda) \end{vmatrix} = 0. \tag{64}$$

After some necessary steps, the roots of the characteristic equation (64) are $\lambda_1 = \lambda_2 = -d_0$ and $\lambda_3 = -(\zeta\alpha_2 + d_4 + d_0) + h_2 + (1 - d_2)\tau_2$.

The simplified value of $\lambda_3 = (\zeta\alpha_2 + d_4 + d_0)(R^2{}_{eff} - 1)$.

Clearly, λ_3 is negative if $R^2{}_{eff} < 1$. This shows that all the eigenvalues of (64) have negative real parts when $R^2{}_{eff} < 1$. Hence, the disease-free equilibrium $E^0{}_2 = (\Lambda/d_0, 0, 0)$ of the dynamical system (50) is locally asymptotically stable if the effective reproduction number $R^2{}_{eff} < 1$ and unstable otherwise. □

3.2.5. Global Stability Analysis of the Disease-Free Equilibrium Point of HIV/AIDS-Only Submodel

Theorem 12. *The disease-free equilibrium point $E^0{}_2 = (\Lambda/d_0, 0, 0)$ of the dynamical system (50) is globally asymptotically stable in the feasible region Ω_2 if the effective reproduction number $R^2{}_{eff} < 1$.*

Proof. To prove the global asymptotic stability of the disease-free equilibrium point, we used LaSalle-Lyapunov candidate function as follows:

Let W_2 be a LaSalle-Lyapunov candidate function such that

$$W_2 = i_1 P_4. \tag{65}$$

where

$$i_1 = \frac{1}{\zeta\alpha_2 + d_4 + d_0}. \tag{66}$$

The time derivative of (65) along the solution path yields

$$\dot{W}_2 = i_1\dot{P}_4 = i_1\left((1 - d_2)\tau_2 + \frac{h_2 P_1}{N_2} - (\zeta\alpha_2 + d_4 + d_0)\right)P_4. \tag{67}$$

Since the state variables of the model when the 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 $E^0{}_2 = (\Lambda/d_0, 0, 0)$ with $R^2{}_{eff} < 1$, $\dot{W}_2$ can be simplified as
$\dot{W}_2 \leq i_1((1 - d_2)\tau_2 + h_2(P_1{}^0/N_2{}^0) - (\zeta\alpha_2 + d_4 + d_0))P_4$.
Substitution of i_1 and simplification gives

$$\dot{W}_2 \leq (R^2{}_{eff} - 1)P_4. \tag{68}$$

Inequality (68) shows that $\dot{W}_2 < 0$ when $R^2{}_{eff} < 1$. Furthermore, $\dot{W}_2 = 0$ if and only if $P_4 = 0$ or $R^2{}_{eff} = 1$ and $(\Lambda/d_0, 0, 0)$

is the only singleton set in $\{(P_1, P_4, P_7) \in \mathbb{R}^3{}_+ \in \Omega_2 : \dot{W}_2 = 0\}$. Thus, by LaSalle's invariance principle, the DFE $E^0{}_2 = (\Lambda/d_0, 0, 0)$ is globally asymptotically stable if $R^2{}_{eff} < 1$. □

3.2.6. Existence of Endemic Equilibrium Point of HIV/AIDS-Only Submodel.
Suppose that $E^*{}_2 = (P^*{}_1, P^*{}_4, P^*{}_7)$ be an arbitrary endemic equilibrium point of HIV/AIDS-only submodel (50), which occurs when the disease persists in the society. After long steps we obtained the endemic equilibrium point

$$E^*{}_2 = \left(\frac{\Lambda(\zeta\alpha_2 + d_0)}{d_0((\zeta\alpha_2 + d_4 + d_0)(R^2{}_{eff} - 1) + \zeta\alpha_2 + d_0)},\right.$$
$$\frac{\Lambda(R^2{}_{eff} - 1)}{(\zeta\alpha_2 + d_4 + d_0)(R^2{}_{eff} - 1) + \zeta\alpha_2 + d_0},$$
$$\left.\frac{\Lambda\zeta\alpha_2(R^2{}_{eff} - 1)}{d_0((\zeta\alpha_2 + d_4 + d_0)(R^2{}_{eff} - 1) + \zeta\alpha_2 + d_0)}\right). \tag{69}$$

The associated force of infection of the dynamical system (50) is given by

$$\lambda_2{}^* = \frac{h_2 P_4{}^*}{N_2{}^*}, \tag{70}$$

where

$$N_2{}^* = \frac{h_2\Lambda(\zeta\alpha_2 + d_0)}{d_0(\zeta\alpha_2 + d_4 + d_0)((\zeta\alpha_2 + d_4 + d_0)(R^2{}_{eff} - 1) + \zeta\alpha_2 + d_0)}. \tag{71}$$

We can show the existence of the endemic equilibrium point of the model by substituting $P^*{}_4$ and $N_2{}^*$ in (70). After substitution we got

$$\lambda_2{}^* = \frac{d_0(\zeta\alpha_2 + d_4 + d_0)(R^2{}_{eff} - 1)}{\zeta\alpha_2 + d_0}. \tag{72}$$

Clearly, $\lambda_2{}^* > 0$ if $R^2{}_{eff} > 1$. Hence, an endemic equilibrium point $E^*{}_2 = (P^*{}_1, P^*{}_4, P^*{}_7)$ of the dynamical system (50) exists whenever $R^2{}_{eff} > 1$

3.2.7. Local Stability of the Endemic Equilibrium Point of HIV/AIDS-Only Submodel

Theorem 13. *The endemic equilibrium $E^*{}_2 = (P^*{}_1, P^*{}_4, P^*{}_7)$ of the model (50) is locally asymptotically stable if $R^2{}_{eff} > 1$.*

Proof. The Jacobian matrix of the model (50) at the endemic equilibrium point $E^*{}_2$ is

$$J_2(E^*{}_2) = \begin{pmatrix} -(F_1 + d_0) & -F_2 & 0 \\ F_1 & F_3 & 0 \\ 0 & \zeta\alpha_2 & -d_0 \end{pmatrix}, \tag{73}$$

where

$F_1 = ((d_0(\zeta\alpha_2 + d_4 + d_0)(R^2{}_{eff} - 1))/\zeta\alpha_2 + d_0)$, $F_2 = (\zeta\alpha_2 + d_4 + d_0)$, and $F_3 = (1 - d_2)\tau_2$

The corresponding characteristic equation of (73) is

$$\lambda^3 + g_2\lambda^2 + g_1\lambda + g_0 = 0, \tag{74}$$

where

$$g_2 = F_1 + 2d_0 - F_3, g_1 = F_1F_2 + (F_1 + d_0)(d_0 - F_3) - F_3d_0, \tag{75}$$

and

$$g_0 = F_1F_2d_0 - F_3d_0(F_1 + d_0). \tag{76}$$

The sign of the coefficients g_0, g_1, and g_2 of the characteristic polynomial $\lambda^3 + g_2\lambda^2 + g_1\lambda + g_0 = 0$ is positive if $R^2{}_{eff} > 1$. In addition to that, $g_1g_2 > g_0$. Thus, by Routh–Hurwitz stability criterion the endemic equilibrium $E^*{}_2$ is stable if $R^2_{eff} > 1$ and unstable if $R^2_{eff} < 1$ □

3.2.8. Global Stability of the Endemic Equilibrium Point of HIV/AIDS-Only Submodel

Theorem 14. *The endemic equilibrium $E^*{}_2 = (P^*{}_1, P^*{}_4, P^*{}_7)$ of the dynamical system (50) is globally asymptotically stable if $R^2{}_{eff} > 1$.*

Proof. Consider the following LaSalle-Lyapunov candidate function:

$$\mathscr{L}_2(P_1, P_4) = h_3(P_1 - P_1{}^*)^2 + h_4(P_4 - P_4{}^*)^2, h_3 > 0, h_4 > 0. \tag{77}$$

Differentiating $\mathscr{L}_2(P_2, P_4)$ with respect to time gives us

$$\begin{aligned}
\dot{\mathscr{L}}_2(P_1, P_4) &= 2h_3(P_1 - P_1{}^*)\dot{P}_1 + 2h_4(P_4 - P_4{}^*)\dot{P}_4 \\
&= 2h_3(P_1 - P_1{}^*)\left(\Lambda - \left(\frac{h_2P_4}{N_2} + d_0\right)P_1\right) \\
&\quad + 2h_4(P_4 - P_4{}^*)\left((1 - d_2)\tau_2P_4 + \frac{h_2P_4}{N_2}P_1 \right. \\
&\quad \left. - (\zeta\alpha_2 + d_4 + d_0)P_4\right).
\end{aligned} \tag{78}$$

Substituting $\Lambda = ((h_2P^*{}_4/N_2{}^*) + d_0)P_1{}^*$ and $(1 - d_2)\tau_2 + (\zeta\alpha_2 + d_4 + d_0) = h_2P^*{}_4/N_2{}^*$ with $N_2 \approx N_2{}^*$ in (78) yields:

$$\begin{aligned}
\dot{\mathscr{L}}_2(P_1, P_4) &= 2h_3(P_1 - P_1{}^*)\left(\frac{h_2P^*{}_4}{N_2{}^*}P_1{}^* + d_0P_1{}^* - \frac{h_2P_4}{N_2}P_1 - d_0P_1\right) \\
&\quad + 2h_4(P_4 - P_4{}^*)\left(\frac{h_2P_4}{N_2}P_1 - \frac{h_2P_4}{N_2{}^*}P_1{}^*\right) \\
&= -2h_3d_0(P_1 - P_1{}^*)^2 - \frac{2h_2h_3}{N_2}P_4(P_1 - P_1{}^*)^2 \\
&\quad + \frac{2h_2}{N_2}(P_4{}^* - P_4)(P_1 - P_1{}^*)(h_3P_1{}^* - h_4P_4) \\
&\leq 0 \text{ if } P_4 \geq \frac{h_3P_1{}^*}{h_4}, P_1{}^* \leq P_1, P^*{}_4 \geq P_4.
\end{aligned} \tag{79}$$

This implies $\dot{\mathscr{L}}_2(P_1, P_4) \leq 0$, and it vanishes if and only if $P_1{}^* = P_1$, $P_4{}^* = P_4$, and $P_1{}^* = h_4P_4/h_3$

This shows that $E^*{}_2 = (P^*{}_1, P^*{}_4, P^*{}_7)$ is the largest compact invariant singleton set in $\{(P^*{}_1, P^*{}_4, P^*{}_7) \in \Omega_2 : \dot{\mathscr{L}}_2 = 0\}$

Hence, by LaSalle's invariance principle [30], the endemic equilibrium $E^*{}_2$ is globally asymptotically stable in the invariant region whenever $R^2{}_{eff} > 1$.   □

4. Analysis of the HBV-HIV/AIDS Model at Equilibrium Points

4.1. Disease-Free Equilibrium Point of HBV-HIV/AIDS Coinfection Model. To find the DFE point, we equated the right-hand side of the full mode (2) to zero and evaluated it at $P_3 = P_4 = P_5 = 0$. Therefore, the DFE point represented by $E^0{}_3$ is equal to:

$$E^0{}_3 = \left(\frac{\Lambda(\omega + d_0(1 - \eta))}{d_0(\omega + d_0)}, \frac{\eta\Lambda}{\omega + d_0}, 0, 0, 0, 0, 0, 0, 0\right). \tag{80}$$

4.2. Effective Reproduction Number of the Full Model. We represented the effective reproduction number of the coinfected model by $R^3{}_{eff}$. To find $R^3{}_{eff}$, we used the next generation matrix method that was formulated in [28, 29]. In the same way that we have shown in sub section 3.1.3, $R^3{}_{eff}$ can be manipulated as follows:

$$F = \begin{bmatrix} (1 - d_1)\tau_1 + \dfrac{h_1(\omega + d_0(1 - \eta))}{\omega + d_0} & 0 & \dfrac{h_1(\omega + d_0(1 - \eta))}{\omega + d_0} \\ 0 & (1 - d_2)\tau_2 + h_2 & h_2 \\ 0 & 0 & 0 \end{bmatrix},$$

$$V = \begin{bmatrix} r_2 + \alpha_1 + d_3 + d_0 & 0 & 0 \\ 0 & \zeta\alpha_2 + d_4 + d_0 & -r_1 \\ 0 & 0 & r_1 + \alpha_3 + d_5 + d_0 \end{bmatrix}. \tag{81}$$

This implies the next-generation matrix FV^{-1} becomes

$$FV^{-1} = \begin{bmatrix} \dfrac{(1 - d_1)\tau_1 + ((\omega + d_0(1 - \eta))h_1/\omega + d_0)}{r_2 + \alpha_1 + d_3 + d_0} & 0 & \dfrac{(\omega + d_0(1 - \eta))h_1}{(\omega + d_0)(r_1 + \alpha_3 + d_5 + d_0)} \\ 0 & \dfrac{(1 - d_2)\tau_2 + h_2}{\zeta\alpha_2 + d_4 + d_0} & \dfrac{h_2}{r_1 + \alpha_3 + d_5 + d_0} + \dfrac{r((1 - d_2)\tau_2 + h_2)}{(r_1 + \alpha_3 + d_5 + d_0)(\zeta\alpha_2 + d_4 + d_0)} \\ 0 & 0 & 0 \end{bmatrix}. \tag{82}$$

The spectral radius (the largest eigenvalue) of (82) is the effective reproduction number of the full model. Hence, after some steps, the spectral radius of equation (82) becomes

$$R^3{}_{eff} = \max\left\{\frac{h_1(\omega + d_0(1 - \eta)) + (\omega + d_0)(1 - d_1)\tau_1}{(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0)}, \frac{h_2 + (1 - d_2)\tau_2}{\zeta\alpha_2 + d_4 + d_0}\right\}. \tag{83}$$

4.3. Local Stability Analysis of the Disease-Free Equilibrium Point of the HBV-HIV/AIDS Coinfection Model

Theorem 15. *The disease-free equilibrium point $E^0_3 = ((h_2 P^*_4/N^*_2/d_0(\omega + d_0)), (\eta\Lambda/\omega + d_0), 0, 0, 0, 0, 0, 0, 0, 0)$ of the dynamical system (2) is locally asymptotically stable if $R^3_{eff} < 1$.*

Proof. The Jacobean matrix of the dynamical system (2) at the DFE point E^0_3 is

$$J(E^0_3) = \begin{bmatrix} -d_0 & \omega & -h_1 k_8 & -h_2 k_8 & -(h_1+h_2)k_8 & 0 & 0 & 0 & 0 & r_3 \\ 0 & -k_1 & 0 & -\dfrac{h_2 d\eta}{\omega+d} & -\dfrac{h_2 d\eta}{\omega+d} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & h_1 k_8 + k_2 & 0 & h_1 k_8 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & h_2 + k_3 & h_2 + r_1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -k_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \alpha_1 & 0 & 0 & -k_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \zeta\alpha_2 & 0 & 0 & -k_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \alpha_3 & 0 & 0 & -k_7 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \theta_1 & \theta_2 & \theta_3 & -d_0 & 0 \\ 0 & 0 & r_2 & 0 & 0 & 0 & 0 & 0 & 0 & -(r_3+d_0) \end{bmatrix}.$$

(84)

where $k_1 = (\omega + d_0)$, $k_2 = (1 - d_1)\tau_1 - (r_2 + \alpha_1 + d_3 + d_0)$, $k_3 = (1 - d_2)\tau_2 - (\zeta\alpha_2 + d_4 + d_0)$, $k_4 = (r + \alpha_3 + d_5 + d_0)$, $k_5 = (\theta_1 + d_0)$, $k_6 = (\theta_2 + d_0)$, $k_7 = (\theta_3 + d_0)$, $k_8 = ((\omega + d_0(1 - \eta))/\omega + d_0)$, $k_9 = (r_3 + d_0)$

The characteristic equation of (84) is

$$(d_0 + \lambda)^2(k_1 + \lambda)(k_4 + \lambda)(k_5 + \lambda)(k_6 + \lambda)(k_7 + \lambda) \\ \cdot (k_9 + \lambda)(t_2\lambda^2 + t_1\lambda + t_0) = 0$$

(85)

where

$$t_2 = 1, t_1 = (r_2 + \alpha_1 + d_3 + d_0)(1 - R^1_{eff}) \\ + (\zeta\alpha_2 + d_4 + d_0)(1 - R^2_{eff}),$$

$$t_0 = (r_2 + \alpha_1 + d_3 + d_0)(1 - R^1_{eff})(\zeta\alpha_2 + d_4 + d_0)(1 - R^2_{eff}).$$

(86)

Thus, the first seven eigenvalues from the characteristic equation (85) are $\lambda_1 = \lambda_2 = -d_0$, $\lambda_3 = -(\omega + d_0)$, $\lambda_4 = -(\theta_3 + d_0)$, $\lambda_5 = -(\theta_2 + d_0)$, $\lambda_6 = -(d_0 + \theta_1)$, $\lambda_7 = -(r_1 + \alpha_3 + d_0 + d_5)$, and $\lambda_8 = -(r_3 + d_0)$. But we determined the remaining two eigenvalues from the characteristic polynomial $t_2\lambda^2 + t_1\lambda + t_0 = 0$ using the Routh-Hurwitz stability criteria. Clearly, $t_2 = 1 > 0$ and both t_1 and t_0 are positive if $R^1_{eff} < 1$ and $R^2_{eff} < 1$. This implies t_1 and t_0 are positive, if $R^3_{eff} = \max \{R^1_{eff}, R^2_{eff}\} < 1$. This shows $\lambda_8 < 0$ and $\lambda_9 < 0$. From these all, we see that all eigenvalues are negative.

Hence, by some algebraic manipulations together with Routh-Hurwitz stability criteria, the disease-free equilibrium point E^0_3 of the dynamical system (2) is locally asymptotically stable if $R^3_{eff} = \max \{R^1_{eff}, R^2_{eff}\} < 1$ □

4.4. Global Stability Analysis of the Disease-Free Equilibrium Point of the HBV-HIV/AIDS Coinfection Model

Theorem 16. *The disease-free equilibrium point $E^0_3 = ((\Lambda(\omega + d_0(1 - \eta))/d_0(\omega + d_0)), (\eta\Lambda/\omega + d_0), 0, 0, 0, 0, 0, 0, 0, 0)$*

of the model (2) is globally asymptotically stable in the feasible region Ω if the effective reproduction number $R^3_{eff} < 1$.

Proof. Consider the following LaSalle-Lyapunov candidate function:

$$\mathscr{L} = m_1 P_3 + m_2 P_4 + P_5,$$

(87)

where

$$m_1 = \frac{1/2(r_1 + \alpha_3 + d_5 + d_0)}{r_2 + \alpha_1 + d_3 + d_0},$$

(88)

and

$$m_2 = \frac{1/2(r_1 + \alpha_3 + d_5 + d_0)}{\zeta\alpha_2 + d_4 + d_0}.$$

(89)

The time derivative of (87) along the solution path yields

$$\dot{\mathscr{L}} = m_1\left((1 - d_1)\tau_1 P_3 + h_1(P_3 + P_5)\frac{P_1}{N} - h_2\frac{(P_4 + P_5)P_3}{N} \right. \\ \left. - (r_2 + \alpha_1 + d_3 + d_0)P_3\right) \\ + m_2\left((1 - d_2)\tau_2 P_4 + h_2(P_4 + P_5)\frac{(P_1 + P_2)}{N} + rP_5 \right. \\ \left. - h_1\frac{(1 - \pi)(P_3 + P_5)P_4}{N} - (\zeta\alpha_2 + d_4 + d_0)P_4\right) \\ + h_1\frac{(1 - \pi)(P_3 + P_5)P_4}{N} + h_2\frac{(P_4 + P_5)P_3}{N} \\ - (r_1 + \alpha_3 + d_5 + d_0)P_5.$$

(90)

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 $E^0_3 = ((\Lambda(\omega + d_0(1 - \eta))/d_0(\omega + d_0)), (\eta\Lambda/\omega + d_0), 0, 0, 0, 0, 0, 0, 0, 0)$ with $R^3_{eff} = \max \{(h_1(\omega + d_0(1 - \eta)) + (\omega + d_0)(1 - d_1)\tau_1/(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0)), (((\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0))/\zeta\alpha_2 + d_4 + d_0)\}$, $\dot{\mathscr{L}}$ can be simplified as follows:

$$\dot{\mathscr{L}} \leq m_1\left((1 - d_1)\tau_1 P_3 + h_1 P_3\frac{P_1^0}{P_1^0 + P_2^0} \right. \\ \left. + h_1 P_5\frac{P_1^0}{P_1^0 + P_2^0} - (r_2 + \alpha_1 + d_0 + d_3)P_3\right) \\ + m_2\left((1 - d_2)\tau_2 P_4 + h_2 P_4\frac{(P_1^0 + P_2^0)}{N} \right. \\ \left. + h_2 P_5\frac{(P_1^0 + P_2^0)}{N} - (\zeta\alpha_2 + d_4 + d_0)P_4\right) \\ - (r_1 + \alpha_3 + d_5 + d_0))P_5.$$

(91)

After simplifying (91) we got

$$
\begin{aligned}
\dot{\mathscr{L}} \leq\; & m_1(r_2 + \alpha_1 + d_0 + d_3)R^1{}_{eff} - 1)P_3 \\
& + m_2(\zeta\alpha_2 + d_4 + d_0)\left(R^2{}_{eff} - 1\right)P_4 \\
& + \left(m_1 R^1{}_{eff}(r_2 + \alpha_1 + d_0 + d_3) + m_2 R^2{}_{eff}(\zeta\alpha_2 + d_4 + d_0)\right. \\
& \left. - (r_1 + \alpha_3 + d_5 + d_0)\right)P_5.
\end{aligned}
\tag{92}
$$

Substituting m_1 and m_2 (92) gives us

$$
\begin{aligned}
\dot{\mathscr{L}} \leq\; & \frac{1}{2}(r_1 + \alpha_3 + d_5 + d_0)\left(R^3{}_{eff} - 1\right)P_3 \\
& + \frac{1}{2}(r_1 + \alpha_3 + d_5 + d_0)\left(R^3{}_{eff} - 1\right)P_4 \\
& + \left((r_1 + \alpha_3 + d_5 + d_0)\left(R^3{}_{eff} - 1\right)\right)P_5.
\end{aligned}
\tag{93}
$$

This implies $\dot{\mathscr{L}} \leq 0$ when $R^3{}_{eff} < 1$. Furthermore, $\dot{\mathscr{L}} = 0$ if and only if $P_3 = P_4 = P_5 = 0$. Thus, by LaSalle's invariance principle [30], the largest invariant set in Ω contained in {

$(P_1, P_2, P_3, P_4, P_5, P_6, P_7, P_8, P_9, P_{10}) \in \mathbb{R}^{10}{}_{+}$} is reduced to the DFE. This proves the global asymptotic stability of the DFE $E^0{}_3$ on Ω if the effective reproduction number $R^3{}_{eff} < 1$ and unstable otherwise. □

4.5. Existence and Stability of Endemic Equilibrium of HBV-HIV/AIDS Coinfection Model.

The endemic equilibrium point of a dynamical system (2) which exists when the disease persists in the community is denoted by $E^*{}_3 = (P_1{}^*, P_2{}^*, P^*{}_3, P^*{}_4, P^*{}_5, P^*{}_6, P^*{}_7, P^*{}_8, P^*{}_9, P^*{}_{10})$. From the analysis of HBV-only submodel (18) and HIV/AIDS-only submodel (50), there exists an endemic equilibrium point for HBV-only submodel and HIV/AIDS-only submodel if $R^1{}_{eff} > 1$ and $R^2{}_{eff} > 1$, respectively.

This implies that the endemic equilibrium point $E^*{}_3$ for HBV- HIV/AIDS coinfection exists because the effective reproduction number for HBV- HIV/AIDS coinfection is the maximum of $R^1{}_{eff}$ and $R^2{}_{eff}$, which is greater than one. After some steps we got $E^*{}_3 = (P_1{}^*, P_2{}^*, P^*{}_3, P^*{}_4, P^*{}_5, P^*{}_6, P^*{}_7, P^*{}_8, P^*{}_9, P^*{}_{10})$. Where

$$
P_1{}^* = \frac{\Lambda(\omega + (1-\eta)(\lambda^*{}_2 + d_0))((r_2 + \alpha_1 + \lambda^*{}_2 + d_3 + d_0) - (1 - d_1)\tau_1)}{(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))},
$$

$$
P^*{}_2 = \frac{\eta\Lambda}{\lambda^*{}_2 + \omega + d_0},
$$

$$
P^*{}_3 = \frac{\lambda^*{}_1\Lambda(\omega + (1-\eta)(\lambda^*{}_2 + d_0))}{(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))},
$$

$$
\begin{aligned}
P^*{}_4 =\; & \frac{\lambda^*{}_2\Lambda(\lambda^*{}_2 + \omega + d_0)(\omega + (1-\eta)(\lambda^*{}_2 + d_0))((r_2 + \alpha_1 + \lambda^*{}_2 + d_3 + d_0) - (1 - d_1)\tau_1)}{(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\lambda^*{}_1 + \lambda^*{}_2 + d_0) + r_2(\lambda^*{}_2 + d_0))(\lambda^*{}_1(1-\zeta)(\alpha_3 + d_5 + d_0) + (\zeta\alpha_2 + d_4 + d_0)(r_1 + \alpha_3 + d_5 + d_0))} \\
& + \frac{\lambda^*{}_2\eta\Lambda(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))(r_1 + \alpha_3 + d_5 + d_0)}{(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))(\lambda^*{}_1(1-\zeta)(\alpha_3 + d_5 + d_0) + (\zeta\alpha_2 + d_4 + d_0)(r_1 + \alpha_3 + d_5 + d_0))} \\
& + \frac{\lambda^*{}_1\lambda^*{}_2 r_1\Lambda(\omega + (1-\eta)(\lambda^*{}_2 + d_0))}{(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))(\lambda^*{}_1(1-\zeta)(\alpha_3 + d_5 + d_0) + (\zeta\alpha_2 + d_4 + d_0)(r_1 + \alpha_3 + d_5 + d_0))},
\end{aligned}
$$

$$
P^*{}_5 = \frac{(1-\zeta)\lambda^*{}_1 P^*{}_4(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(r_2 + \alpha_1 + \lambda^*{}_2 + d_3 + d_0) - r_2\lambda^*{}_1) + \lambda^*{}_1\lambda^*{}_2\Lambda(\omega + (1-\eta)(\lambda^*{}_2 + d_0))}{(r_1 + \alpha_3 + d_5 + d_0)(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))},
$$

$$
P^*{}_6 = \frac{\lambda^*{}_1\alpha_1\Lambda(\omega + (1-\eta)(\lambda^*{}_2 + d_0))}{(\phi + \mu)(\lambda^*{}_H + q + \mu)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))}, \quad P^*{}_7 = \frac{\zeta\alpha_2 P^*{}_4}{\theta_2 + d_0},
$$

$$
P^*{}_8 = \frac{\alpha_3\lambda^*{}_1((1-\zeta)(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))P^*{}_4 + \Lambda\lambda^*{}_2((1-\eta)(\lambda^*{}_2 + d_0) + \omega))}{(\theta_3 + d_0)(r_1 + \alpha_3 + d_5 + d_0)(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_1 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))},
$$

$$
\begin{aligned}
P^*{}_9 =\; & \frac{\alpha_3\theta_3\lambda^*{}_1((1-\zeta)(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))P^*{}_4 + \Lambda\lambda^*{}_2((1-\eta)(\lambda^*{}_2 + d_0) + \omega))}{d_0(\theta_3 + d_0)(r_1 + \alpha_3 + d_5 + d_0)(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))} \\
& + \frac{\lambda^*{}_1\alpha_1\Lambda\theta_1((1-\eta)(\lambda^*{}_2 + d_0) + \omega)}{d_0(\theta_1 + d_0)(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))} + \frac{\theta_2\zeta\alpha_2 P^*{}_4}{d_0(\theta_2 + d_0)},
\end{aligned}
$$

$$
P^*{}_{10} = \frac{r_2\lambda^*{}_1\Lambda(\omega + (1-\eta)(\lambda^*{}_2 + d_0))}{(r_3 + d_0)(\lambda^*{}_2 + \omega + d_0)((\lambda^*{}_1 + \lambda^*{}_2 + d_0)(\alpha_1 + \lambda^*{}_2 + d_3 + d_0) + r_2(\lambda^*{}_2 + d_0))}.
\tag{94}
$$

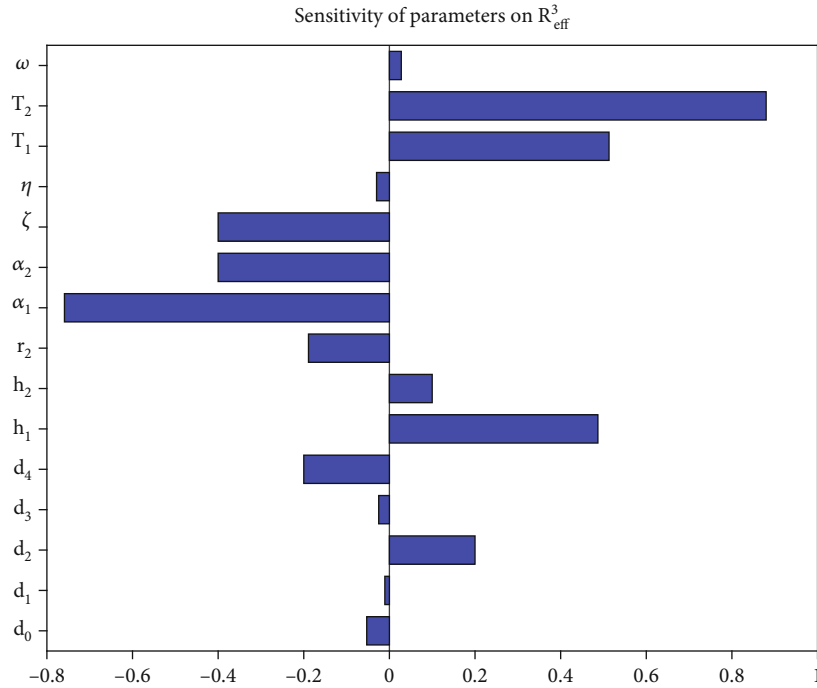Figure 2: Magnitude and direction of sensitivity analysis result of parameters for the full model.

5. Sensitivity Analysis

The reason why this section is important is that it tells us which parameters deserve the most numerical attention. That is, it highlights which parameters should be prioritized in prevention and controlling strategies. On the basic parameters, we carried out sensitivity analysis using the techniques outlined by [36, 37]. As the magnitude and direction of the sensitivity analysis result of the model parameters in Figure 2 indicates, the most sensitive parameters are τ_2 and α_1, which are the rate of vertical transmission of HIV and treatment rate for HBV infected class, respectively. The graphical representation of sensitivity indices of R^3_{eff} is given as follows:

The sign of the sensitivity index of each parameter value in Figure 2 shows that what will happen to R^3_{eff} if the parameter is increased or decreased. R^3_{eff} increases when sensitivity indices with positive signs increase, while R^3_{eff} decreases when sensitivity indices with negative signs increase and vice versa. The indices in the figure also shows that the percentage change of R^3_{eff} for each increase value of parameter for 1%. As the figure shows, the sensitivity indices of τ_2 and α_1 are 0.88 and −0.759, respectively. These parameters deserve the most numerical attention. For instance, increasing τ_2 for 10% will increases R^3_{eff} for approximately

8.8%. On the other hand, increasing α_1 for 10% will decreases R^3_{eff} for 7.59%. Biologically, this means that, increasing treatment rate by 10% for HBV infected individuals decreases a patient's chance of infecting others by approximately 7.59% during his/her infectious period. On the other hand, a single HIV/AIDS infectious individual gets approximately 8.8% chance to infect healthy individuals during his/her infectious period if the rate of MTCT of HIV increased by 10%. Hence, increasing treatment rate for HBV infected individuals and decreasing the rate of MTCT of HIV are very important to eliminate HBV- HIV/AIDS coinfection.

6. Numerical Simulation

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

Initially, $P_1(0) =100,000$, $P_2(0) =1000$, $P_3(0) =10,000$, $P_4(0) =3000$, $P_5(0) =1000$, $P_6(0) =500$, $P_7(0) =500$, $P_8(0) = 100$, $P_9(0) =200$, and $P_{10}(0) =750$.
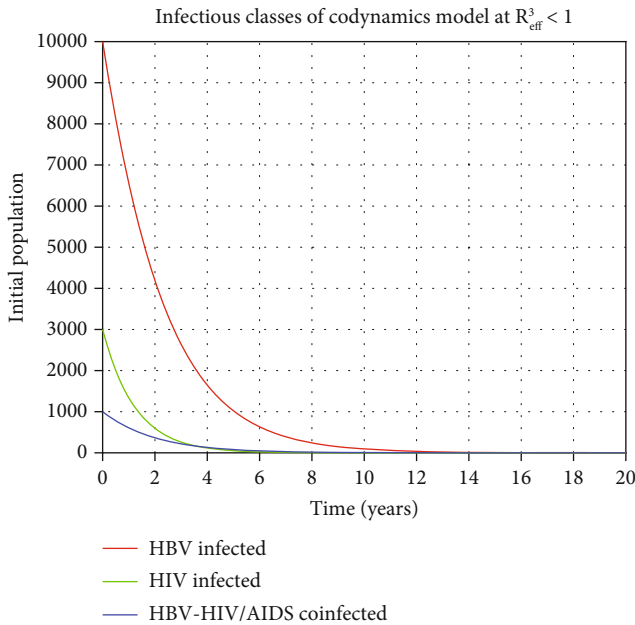
Numerical simulations for all models here are manipulated using MATLAB numerical solver (ode45). We choose ODE45 for the reason that the state of being exact and the speed at which the result of numerical process of calculating complicated system faster.

7. Results and Discussions

In this section, numerical results are manipulated for the submodels and the coinfection model using MATLAB numerical solver (ode45). We chose ODE45 for the reason that the state of being exact and the speed at which the result

Table 2: Parameter values used in numerical simulations.

| Parameters	Value	Source
Λ	$0.04^{*}N_0$	[26]
α_1	0.3	[26]
d_5, α_3, r_1	0.001, 0.015, 0.001 respectively	[26]
r_2	0.075	[26]
h_2	0.03	[26]
$\theta_1, \theta_2, \theta_3$	0.014, 0.013, and 0.012, respectively	[26]
d_0	0.01	[31]
d_4	0.333	[31]
τ_1, d_1, d_3, r_3	0.4, 0.01, 0.1, 0.2, respectively	Assumption
τ_2	0.3	[32]
d_2	0.2	[32]
α_2	0.6	[33]
ζ	0.396	[33]
η	0.65	[34]
h_1	0.4	[34]
ω	0.1	[34, 35]

Figure 3: Population Profile of infected classes of the coinfection model at $R^3_{eff} = 0.49$, $h_1 = 0.09$, and $d_0 = 0.5$.

of numerical process of calculating complicated system faster. Figure 3 has been plotted under consideration of the effective reproduction number of the full model less than unity. It is about the behavior of infected classes of the coinfection model at $R^3_{eff} = 0.49$, $h_1 = 0.09$, $d_0 = 0.5$, and all other associated parameters as listed in Table 2. As the figure clearly shows, each infectious class of HBV- HIV/AIDS

coinfection model (2) converges to the disease-free equilibrium point of the model. The convergence of all infectious classes to the disease-free equilibrium point of the model shows that the disease-free equilibrium of the model is globally asymptotically stable, which indicates the absence of HBV- HIV/AIDS coinfection in the society.

Figures 4(a), 4(b), and 4(c) are demonstrate the stability of endemic equilibrium of HBV- HIV/AIDS coinfection model for three different initial conditions keeping all other associated parameters as listed in Table 2. In each case, the effective reproduction number is greater than one and the simulation results show the convergence of the solutions of the model to the endemic equilibrium point. The convergence of the solutions of the model to the endemic equilibrium point for different initial conditions indicates that the endemic equilibrium point of the model is locally asymptotically stable (i.e., the disease is spreading in the society). Effect of the rate of MTCT of HBV on the effective reproduction number of HB-only submodel and HBV-HIV/AIDS coinfection model, respectively, are shown in Figures 5(a) and 5(b). The figures show that if the rate of MTCT of HBV (τ_1) is less than 0.02, the reproduction number of HB-only submodel and HBV-HIV/AIDS coinfection model are less than one, which indicates both infections die out in the community. Whereas, if the rate of MTCT of HBV (τ_1) is greater than 0.02, the reproduction number of HB-only submodel and HBV-HIV/AIDS coinfection model are greater than one, which shows the infections are spreading in the community. On the other hand, Figure 6(a) is demonstrating the effect of the rate of MTCT of HIV on the effective reproduction number of HIV/AIDS-only submodel. The simulation results in Figure 6(a) show that if the rate of MTCT of HIV (τ_2) is less than 0.68, the reproduction number of HIV-only submodel is less than one, which indicates HIV infection dies out in the community. Whereas, if the rate of MTCT of HIV (τ_2) is greater than 0.68, the effective reproduction number of HIV/AIDS-only submodel is greater than one, which shows the infection is spreading in the community.

Figure 5(c) shows the profile of HB-only infected individuals for different values of τ_1. Three different values were considered with increasing τ_1 whose values were 0.04, 0.6, and 0.9, respectively. It was observed that when $\tau_1 = 0.9$, there was a rapid growth in HB-only infected population from 10000 to 13225 up to one and a half years. Thereafter, there was a gradual decrease in the population. The gradual decrease in the population was due to enough immunization coverage and effective treatment applied to prevent the spread of the infection. That means with the current prevention and control lowering the number of patients is impossible within one and a half years when the transmission rate is high. Similarly, when $\tau_1 = 0.6$, the number of HB-only infected individuals rises rapidly from 10000 to 10836 within a year. Thereafter, there was a decline in HB-only infected individuals as time progresses as a result of effective prevention and control measures. On the other hand, we did not observe a gradual or rapid increase in HB-only infected individuals when $\tau_1 = 0.04$, but a decrease. This is due to the lowest vertical transmission rate of HBV and effective
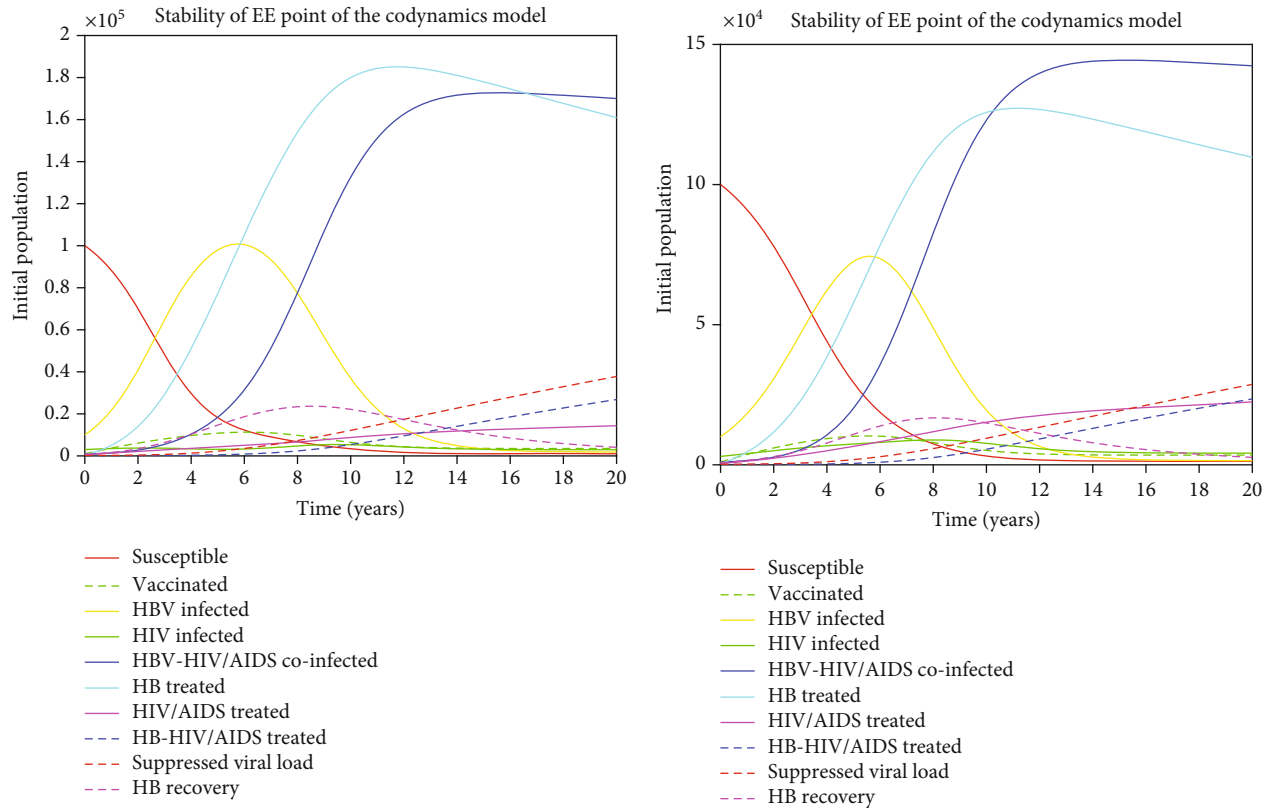
(a) Stability of EE of HBV-HIV/AIDS coinfection model at $R^3_{eff} = 3.14$, $h_1 = 0.9$, and $h_2 = 0.5$

(b) Stability of EE of HBV-HIV/AIDS coinfection model at $R^3_{eff} = 3.42$, $h_1 = 0.7$, and $h_2 = 0.6$
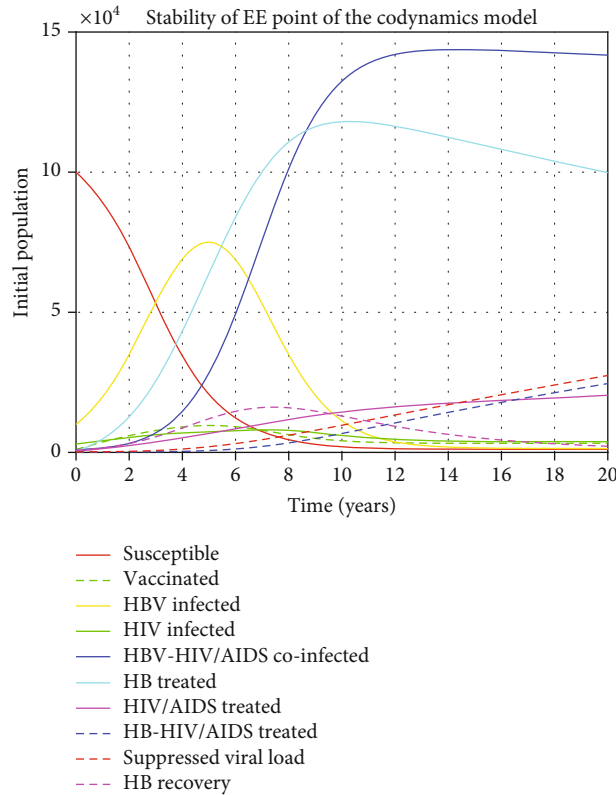
(c) Stability of EE of HBV-HIV/AIDS coinfection model at $R^3_{eff} = 4.0$, $h_1 = 0.8$, and $h_2 = 0.65$

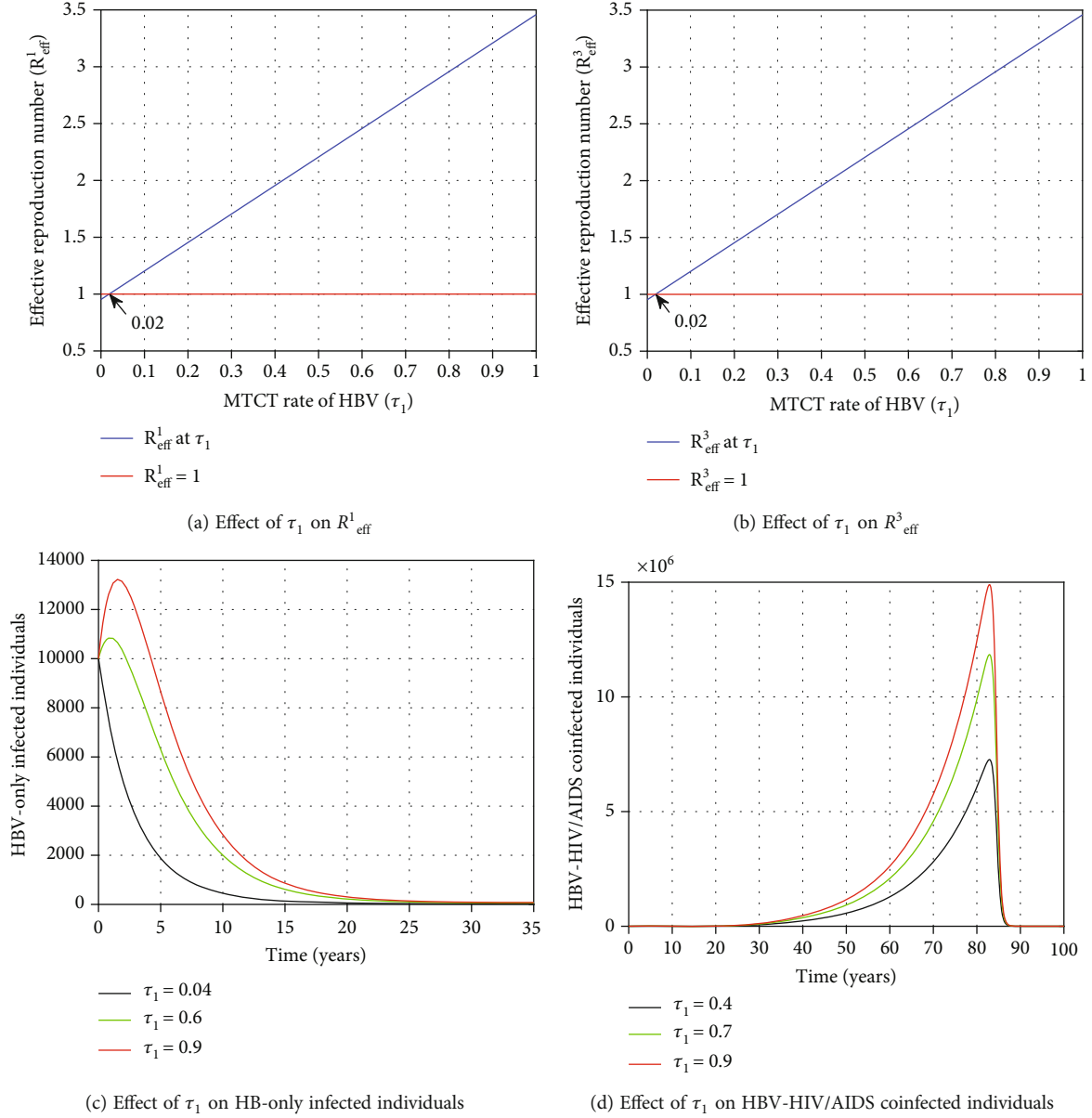Figure 4: Stability of EE of HBV-HIV/AIDS coinfection model for three initial conditions.

(a) Effect of τ_1 on $R^1{}_{eff}$

(b) Effect of τ_1 on $R^3{}_{eff}$

(c) Effect of τ_1 on HB-only infected individuals

(d) Effect of τ_1 on HBV-HIV/AIDS coinfected individuals

FIGURE 5: Effect of MTCT of HBV on $R^1{}_{eff}$, $R^3{}_{eff}$, HB-only infected individuals, and HBV-HIV/AIDS coinfected individuals.

prevention and control measures applied to forestall the spread of the infection. That is when the transmission rate is low, it is possible to manage the number of HB infected individuals with the current prevention and control.

Figure 5(d) is demonstrating the effect of τ_1 on HBV-HIV/AIDS coinfected individuals. In this figure, for τ_1 = 0.4, τ_1 = 0.7, and τ_1 = 0.9, there was a slow increase in HBV-HIV/AIDS coinfected individuals up to thirty years due to enough immunization coverage and effective treatment applied to prevent the spread of the infection. However, for the same values of τ_1, there was a rapid growth of coinfected individuals to different peaks from thirty to eighty years. This indicates that the current prevention and control should be modified after thirty years. After eighty years, there was a rapid decrease in the population. This is due to the effective preven-

tion and control measures applied to forestall the spread of the infection and disease-induced deaths.

Figure 6(b) is about the profile of HIV/AIDS-only infected individuals for three different values of τ_2. The figure shows that when τ_2 =0.3 and 0.6, the number of HIV/AIDS-only infected individuals did not increase but decreased due to the effective control measure applied to forestall the spread of HIV infection. But there was a slight increase when τ_2 =0.9 within one and a half years. Thereafter, there was a decline in HIV/AIDS-only infected individuals as time progresses as a result of treatment and becomes near to zero after twenty-four years. The effect of τ_2 on HBV-HIV/AIDS coinfected individuals is examined in Figure 6(c). As the figure shows when τ_2 =0.3 and 0.6, the number of HIV/AIDS-only infected individuals did not
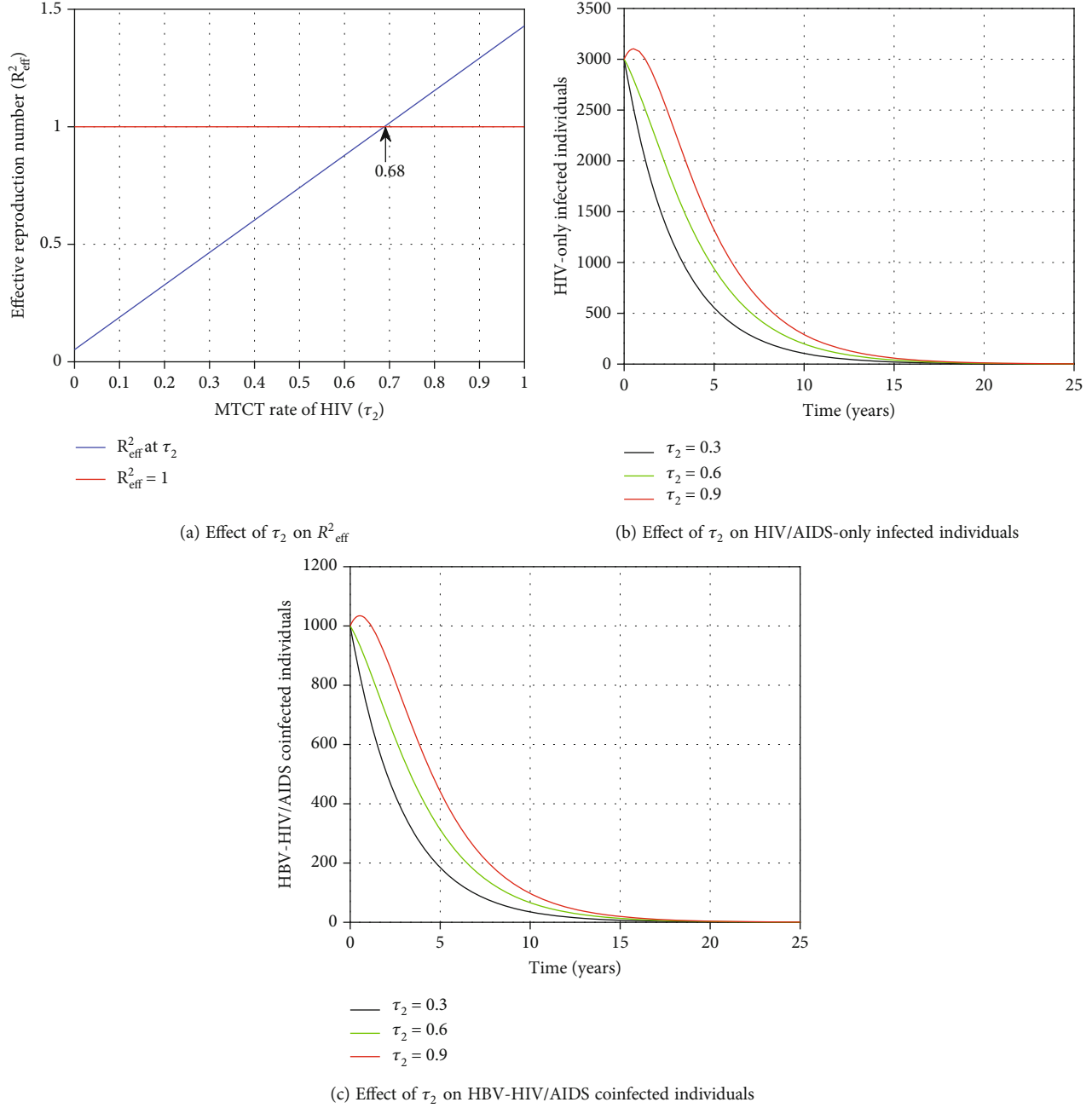
(a) Effect of τ_2 on R^2_{eff}

(b) Effect of τ_2 on HIV/AIDS-only infected individuals

(c) Effect of τ_2 on HBV-HIV/AIDS coinfected individuals

FIGURE 6: Effect of MTCT of HIV on R^2_{eff}, HIV/AIDS-only infected individuals, and HBV-HIV/AIDS coinfected individuals.

increase but decreased due to the effective control measure applied to forestall the spread of HIV infection. But there was a slight increase when $\tau_2 = 0.9$ within one and a half years. Thereafter, there was a decline in HIV/AIDS-only infected individuals as time progresses as a result of treatment and becomes near to zero after twenty-four years.

8. Conclusion

The importance of epidemiological models lies in their ability to provide meaningful biological interpretations and pos-

sible disease prevention and control measures. In this study, we improved the model in [26] to show that the combined effect of MTCT of hepatitis B virus and HIV on their codynamics model, which were not considered in [26]. We derived the effective reproduction number (R^3_{eff}) of the improved model and compared it with the effective reproduction number (R^{BH}_{eff}) in [26]. The effective reproduction number of the improved model is $R^3_{eff} = \max\{(h_1(\omega + d_0(1-\eta)) + (\omega + d_0)(1-d_1)\tau_1/(\omega + d_0)(r_2 + \alpha_1 + d_3 + d_0)), (h_2 + (1-d_2)\tau_2/\zeta\alpha_2 + d_4 + d_0)\}$. Based on the data given in Table 2, we evaluated the numerical value of R^3_{eff}. As the

numerical value indicates, $R^3_{eff} = \max\{2, 0.5\} = 2$ which is greater than $R^{BH}_{eff} = \max\{0.94, 0.28\} = 0.94$ in [26]. This tells us that the transmission possibility of the infection in the improved model is high due to MTCT of both infections, which was not observed in the previous work. This is because, in the improved model, MTCT of hepatitis B virus and HIV causes HBV-HIV/AIDS coinfection to spread and may cause an epidemic, whereas, this infection will not spread, there will be a decline in the number of cases, and will eventually die out because one infectious case will infect less than one person on average in his/her infectious period in [26]. We proved that the disease-free equilibrium points of the models are locally and globally asymptotically stable if the associated reproduction numbers 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 are greater than one. From the sensitivity analysis calculated to show the impact of different parameters on R^3_{eff}, the most sensitive parameters are τ_2 and α_1, which are the rate of MTCT of HIV and treatment rate for HBV infected class, respectively. As shown in Section 8, if the rate of MTCT of HBV (τ_1) and HIV (τ_2) are less than 0.02 and 0.3, respectively, the coinfection of the two viruses will not spread in the community, there will be a decline in the number of cases, and will eventually dies out in the community. Hence, an increase in the rates of MTCT of HBV and HIV exacerbated HBV-HIV/AIDS coinfection, while a decrease in the rates of MTCT of these infections would decline the number of cases, minimize the spread, and help to eliminate the coinfection of HBV and HIV from the community gradually. From the numerical results, we recommend that public policymakers and other concerned bodies must focus on decreasing the rates of MTCT of HBV and HIV in addition to the recommendation given in the study [26] to control the spread of HBV-HIV/AIDS coinfection. Last but not least, it should be noted that this study did not take into account the importance of screening in the dynamics of HBV-HIV/AIDS coinfection. It may affect the transmission dynamics of HBV-HIV/AIDS coinfection in a population. We leave this for future consideration.

Data Availability

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.

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