

Modelling the Impact for the Application of an Antiretroviral Drug Therapy on HBV Infection

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ABSTRACT

In this paper, a mathematical model that considered the rate of antiretroviral drug therapy on hepatitis B virus (HBV) infection with a logistic growth, the immune response, the cure rate of an infected cells and a standard incidence function for the proliferation of the hepatocytes cells was developed. The results from the numerical simulations of the model shows that only 100% average usage of the antiretroviral therapy can guarantee a maximum number of healthy hepatocytes cells, a liver free from viruses and the absence of infected (unhealthy) hepatocytes cells from the liver.

Keywords: Hepatitis B Virus (HBV), Infection, logistic growth, antiretroviral drug therapy, hepatocytes cells

1.0 INTRODUCTION

Hepatitis B Virus (HBV) which is a major global caused of liver infection is one of the major threats to human lives particularly in Sub-Saharan Africa, some parts of the Arabian Peninsula, Asia, South Pacific, arctic North America, and the tropical South America. The disease usually caused liver infection that can lead to a Cirrhosis (scarring of the liver) and/or liver cancer. There is a high risk of spreading the virus by injecting several people with an unsterilized needle, or having a sexual contact with an infected person (Eikenberry *et al.*, 2009).

Almost one (1) out of every four (4) adults that have been diagnosed with the chronic infection of the virus from their childhood die from the liver Cirrhosis or Cancer (Dong *et al.*, 2010). More so, the rate at which individuals get infected with the virus is 50 to 100 times more compared to that of human immunodeficiency virus (HIV), with more than five hundred (500) million new cases being diagnosed yearly. Currently lamivudine or Interferon are the drugs been used for the treatment of patients with chronic HBV (WHO, 2020).

Most people who become patients of the disease and recover from it can never be infected again (they possesses permanent immunity). However, few of them who become infected early will have it as chronic infection (long period of infection) which make them became carriers (can pass it to others). Hepatitis B is one of the world's most common and serious infectious diseases. Over 350 million people worldwide are currently believed to be persistent carriers of the hepatitis B virus (HBV), with 1 million people dying as a result of chronic HBV infection. This paper created a nonlinear mathematical model for the transmission dynamics of HBV (Firaol *et al.*, 2023).

Hepatitis B remains enormous concern for the people affected by this viral infection. Understanding the intricate dynamics of Hepatitis B transmission is crucial for developing effective clinical control and evaluating clinical impact of immunity strategies (James 2025). To the declaration of July 28 as the world's hepatitis day with the aim to raise awareness worldwide of Hepatitis B and hepatitis C in order to encourage prevention, diagnosis and treatment. This campaign has been championed by the World Hepatitis Alliance since 2007 which produced a result in May 2010, during which the World Health Organization (WHO) grant it a global confirmation and from then up till now the July 28th of every year is declared as a World Hepatitis Day. In March 2015, WHO issued its first guidelines for the treatment of chronic hepatitis B. This condition according to WHO, is affecting some 240 million people world-wide. These guidelines are the prevention, care and treatment of person living with chronic hepatitis B (WHO, 2022).

Viral hepatitis B is a blood-borne infection that is most typically passed from mother to child during childbirth, but it can also be spread through sexual contact, sharing needles, syringes, or other drug injections, or being exposed to sharp devices. The acute stage of hepatitis B virus disease occurs within the first six months after infection. At this point, only a few infected persons are capable of resisting and clearing the infection; for others, the infection progresses to a chronic or lifelong condition and becomes a significant health issue. Hepatitis B disease gained in maturity produces chronic hepatitis in less than 5% of cases, whereas disease acquired in infancy and early childhood causes chronic hepatitis in approximately 95% of cases (Malede 2023).

A mathematical model is vital for understanding disease transmission and spread. Many mathematical

models have been used to investigate the dynamics of hepatitis B illness. Among these models, Khan et al. created a deterministic compartmental model of hepatitis B with a migrating class impact. The system simulation demonstrated that the flow rate from the migrated class is directly proportional to the number of infected people. We investigated a compartmental model of hepatitis B disease by using vaccination and treatment control techniques. Pontryagin's maximum approach and forward-backward iterative scheme were used to calculate the essential criteria for optimal values and numerically solve the optimal system.

Hepatitis B remains a significant global and national public health challenge. The virus Hepatitis B (HBV) infects the liver, and chronic HBV infection sharply increases the risk of serious long-term complications such as cirrhosis, liver failure, and primary liver cancer (hepatocellular carcinoma). Globally, HBV is one of the main causes of viral hepatitis and contributes substantially to liver-related morbidity and mortality (Busayo *et al.*, 2019). In Nigeria, the burden of HBV is particularly high compared to global averages, making the disease a pressing public health concern. A recent comprehensive meta-analysis of studies conducted between 2010 and 2019 estimated a pooled national prevalence of 9.5 % (95% CI: 8.1–11.0), which falls into the “high endemicity” category. This high prevalence translates into a substantial number of people living with chronic HBV: applying 9.5% prevalence to Nigeria’s large population suggests millions of individuals may be infected.

Sub-group and regional analyses reveal significant heterogeneity in HBV prevalence across Nigeria. For instance, prevalence tends to be higher in rural settings (about 10.7 %) compared to urban areas (about 8.2 %), and among certain subpopulations (e.g., blood donors). (WHO, 2021). Moreover, population groups such as pregnant women, incarcerated persons, and other high-risk groups have sometimes exhibited even higher prevalence rates in individual studies (Babayemi 2025).

Despite the availability of effective interventions notably vaccination many gaps remain. In Nigeria, widespread infant immunization against HBV was introduced, yet coverage remains sub-optimal, especially when it comes to timely birth-dose vaccination. Additionally, screening and diagnosis among adults remain low, and many individuals living with chronic HBV are unaware of their status. WHO supported the country in developing policy and guidance documents to prevent and treat viral hepatitis across the 5 core intervention areas (e.g., vaccination, prevention of mother to child transmission, blood and injection safety, harm reduction and hepatitis B and C testing and treatment (WHO, 2024).

Complicating matters further, recent research has drawn attention to the phenomenon of “occult” HBV infection where individuals carry HBV DNA but test negative for the standard surface antigen (HBsAg). Such infections can be especially problematic in contexts like blood transfusion or organ transplantation. These factors high and heterogeneous prevalence, suboptimal vaccination and screening coverage, presence of occult infections, and limited public awareness combine to make HBV a persistent and under-addressed public health threat in Nigeria (Sonderup *et al.*, 2022).

Given this background, there is an urgent need for strategic, evidence-based public health planning. Here, mathematical modelling offers a powerful and flexible tool. By formalizing the transmission dynamics of HBV accounting for acute and chronic infection states, perinatal/childhood transmission, adult infection, vaccination, possible occult infection, and other epidemiological factors models can help estimate key parameters (e.g., reproduction number), project future HBV prevalence under different intervention scenarios, and evaluate the impact of strategies such as expanded vaccination, increased screening, and treatment programs (Hsu *et al.*, 2023).

Immunopathogenesis of HBV when effectively grasped can lead to an efficient way to prevent and treat the disease. Fundamental insight into the fine details of the interaction between HBV and immune system have been provided through molecular procedure. However, many biologically important questions are not primarily concerned with population dynamics of the immune reaction. Mathematical models are often needed to answer these questions. Unlike many other viruses like HIV where cell culture structures allow in vitro infection and passaging of virus are not obtainable for HBV, which is noncytopathic and hepatotropic. Studies conducted on pathogenesis in animal models recently demonstrated that the enhanced recruitment of virus-specific CTLs into the cells of liver is critical for the pathogenesis of HBV infection and hepatocellular carcinoma.

Despite the reality that human being is blessed with numerous researches works and availability of secure, effective drugs and vaccinations, HBV carried on to be a serious threat to human being globally because of the increase in its morbidity and mortality rate. In Nigeria for instance, the prevalent rate of 12% which was reported in 2006 has risen to 13.6 % in 2013 (Musa *et al.*, 2015). In fact, latest statistics released by the Federal Ministry of Health indicated that one out of every ten Nigerians has Hepatitis (SOGHIN, 2016). The aim of this research is to investigate the use of antiretroviral drug therapy at different percentage (%) levels, in order to find out at what percentage of the

drug therapy administration has higher effect for eradicating the Hepatitis B virus completely.

2.0 MODEL FORMULATION

A mathematical model of HBV infection was developed improving on the existing models. The model incorporated the use of antiretroviral drug therapy with standard incidence function and a logistic term for proliferation of hepatocytes. The developed model contains five (5) variables described as: uninfected (healthy or target) hepatocytes (U), Exposed hepatocytes (E), Virus producing (infected) hepatocytes (I), Total host (T) and Free virus (V).

The utmost aim of the therapy with the antiviral drug is to curtail the infection, which can be accomplished through inhibition of viral replication or improvement of immunological reactions or both. There are lots of existing drugs for treating HBV infection, some of which includes peginterferon Alfa -2a, lamivudine, adefovir dipivoxil to mentioned but a few. Lamivudine (which is a Bacteriostatic antiretroviral drug therapy) for instance stops the virus from replicating.

When there is no treatment, the healthy (uninfected) hepatocytes become unhealthy (infected) at the rate β by the free virus. We then introduced a function $(1 - \varepsilon_b) \psi$ at this point, with ε_b as the efficacy of the drug therapy in blocking the potentiality of HBV to successfully contaminate a cell as in [14, Dahari *et al.*, (2009)] and ψ represents a control parameter for standard utilization rate of antiretroviral drug treatment with values that ranges from 0.25 - 1.0 (i.e. 25% - 100%).

The following assumptions are considered in the construction of the model:

- i. There exists a homogeneous mixing of the cells and the virus, where all cells are equally likely to be infected by the virus.
- ii. The liver is considered as a boundary space instead of being an unbounded space.
- iii. The antiretroviral drug treatment blocks infection of cells in the company of the virus that reduces the production rate of the virus by increasing an infected cells that turn-on the effectiveness of the drugs.

The uninfected hepatocytes (U) proliferate according to a signal that takes on the logistic form, with a homeostatic liver size T_{\max} , with a maximum per capita rate of proliferation ρ , with the reality that the hepatocytes cannot increase unboundedly. The healthy hepatocytes die at a rate μ_1 . Uninfected hepatocytes become latently infected by free virus at a rate β . Exposed cells moved to infectious class at the rate of σ . Infected hepatocytes (I) may be healed and return to uninfected stage class at a rate γ . This stage is described as noncytolytic loss of infected hepatocytes through loss of all covalently closed circular DNA (cccDNA) from the nucleus (Hattaf and Yousfi, 2011). N is the average number of viruses produced by infected cells. Furthermore, the Exposed cells die naturally at a rate μ_2 , the virus producing hepatocytes (I) die naturally at a rate μ_3 , while the free virus reduces at a rate μ_4 as a result of natural death. The description of the variables and parameters are explained in Table 1.

Table 1: Description of the Variables and Parameters of the Model

Variables/Parameters	Description
$U(t)$	Uninfected (healthy or target) hepatocytes at time t
$E(t)$	Exposed hepatocytes at time t
$I(t)$	Virus producing (infected) hepatocytes at time t
$V(t)$	Free virus at time t
$T(t)$	Total host at time t
ρ	Rate of proliferation of hepatocytes
β	Effective contact rate between V and U .
μ	Natural death rate of hepatocytes
μ_1	Virus-induced death rate of exposed hepatocytes
μ_2	Virus-induced death rate of Virus producing hepatocytes
μ_3	Natural death rate of free virus
σ	Progression rate from E to I .

γ	Rate of reversion from I to U due to clearance of virus by the immune response
N	Average number of viruses produced by infected cells.
T^m	Maximum number of hepatocytes
ε_b	Efficacy of antiretroviral drug in blocking infection
ε_p	Efficacy of antiretroviral drug in reducing viral production by virus producing cells
ψ	Compliance to drug usage

And the schematic diagram of the developed model is shown in Figure 1.

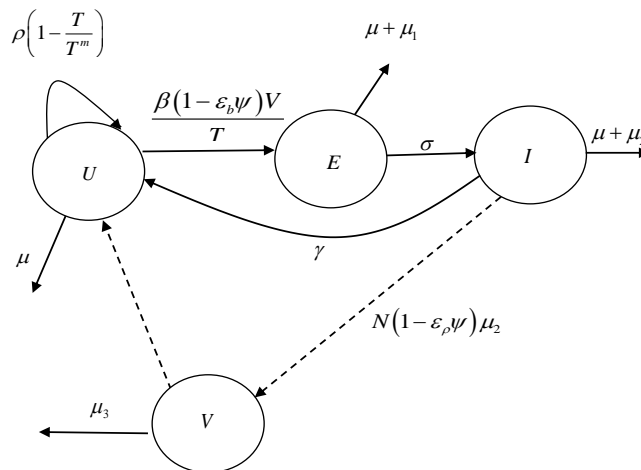


Fig. 1: The schematic diagram of the developed HBV infection model

Going by the above-mentioned assumptions and formulation, we arrived at the following nonlinear-coupled system of differential equations:

$$\begin{aligned}
 \frac{dU}{dt} &= \rho U \left(1 - \frac{T}{T^m}\right) - \frac{\beta(1 - \varepsilon_b \psi) V U}{T} + \gamma I - \mu U \\
 \frac{dE}{dt} &= \frac{\beta(1 - \varepsilon_b \psi) V U}{T} - (\sigma + \mu + \mu_1) E \\
 \frac{dI}{dt} &= \sigma E - (\gamma + \mu + \mu_2) I \\
 \frac{dV}{dt} &= N(1 - \varepsilon_p \psi) \mu_2 I - \frac{\beta(1 - \varepsilon_b \psi) V U}{T} - \mu_3 V
 \end{aligned} \tag{1}$$

where:

$$T = U + E + I \tag{2}$$

so that:

$$\frac{dT}{dt} = \rho U \left(1 - \frac{T}{T^m}\right) - \mu T - (\mu_1 E + \mu_2 I) \tag{3}$$

2.1 Positivity and Boundedness of Solutions

Since the model is dealing with populations, it is evident that all the variables and parameters under consideration in this model are positive. Thus, the closed set

$$\Omega = \Omega_1 \times \Omega_2 \subset \mathbb{R}_+^3 \times \mathbb{R}_+^1 \tag{4}$$

with;

$$\Omega_1 = \left\{ \left(U, E, I \in \mathbb{R}_+^3 : T \leq \frac{\rho UT^m}{(\rho U + \mu T^m)} \right) \right\} \quad (5)$$

for hepatocytes, and

$$\Omega_2 = \{V : V \geq 0\} \quad (6)$$

for free virus population, is positively-invariant set under the flow described by equations (1), so that no solution path leaves through any boundary of Ω . Hence, it is sufficient to consider the dynamics of the model in Ω_1 and Ω_2 . Thus, the model under consideration is mathematically and epidemiological well posed.

3.0 REPRODUCTION NUMBER AND THE STABILITY OF VIRUS-FREE EQUILIBRIUM (e^0)

The model (1) has a virus-free equilibrium states, given by;

$$e^0 = (U^0, E^0, I^0, V^0) = \left(\frac{(\rho - \mu)T^m}{\rho}, 0, 0, 0 \right) \quad (7)$$

3.1 Effective Reproduction Number (R_C) and the Local Stability of the Disease-free Equilibrium state

Using the notations stated in van den Driessche and Watmough (2002), also same in Tasiu *et al.*, (2024a) the next generation operator method is applied on the system (1) to show the local stability of the HBV-free equilibrium. Thus, the matrices \mathcal{F} and \mathcal{V} for the new infection terms and the surviving transfer terms are respectively given by;

$$\mathcal{F} = \begin{pmatrix} 0 & 0 & \frac{\beta(1-\varepsilon_b\psi)U^0}{T^0} \\ 0 & 0 & 0 \\ 0 & N(1-\varepsilon_\mu\psi)\mu_2 & 0 \end{pmatrix} \quad (8)$$

and

$$\mathcal{V} = \begin{pmatrix} -(\sigma + \mu + \mu_1) & 0 & 0 \\ \sigma & -(\gamma + \mu + \mu_2) & 0 \\ 0 & 0 & -(\beta + \mu_3) \end{pmatrix} \quad (9)$$

And from van den Driessche and Watmough [10], it follows that the control reproduction number is;

$$R_C = \rho(\mathcal{F}\mathcal{V}^{-1}) = \frac{\beta(1-\varepsilon_b\psi)N(1-\varepsilon_\mu\psi)\sigma\mu_2U^0}{(\sigma + \mu + \mu_1)(\gamma + \mu + \mu_2)(\beta + \mu_3)T^0} \quad (10)$$

i.e.

$$R_C = \frac{\beta(1-\varepsilon_b\psi)N(1-\varepsilon_\mu\psi)\sigma\mu_2}{(\sigma + \mu + \mu_1)(\gamma + \mu + \mu_2)(\beta + \mu_3)} \quad (11)$$

Thus, by Theorem 2 of [10], we established the following Theorem.

4.0 EFFECTS OF APPLYING ANTIRETROVIRAL DRUG TREATMENT ON HBV INFECTION

The utmost goal of treatment with antiretroviral drug therapy is to decrease the infection, which can be attained through the inhibition of viral replication or improvement of immunological reactions or both. There are lots of drugs for treating HBV infection that comprise of; adefovir dipivoxil, peginterferon Alfa -2a and lamivudine to mention but a few. Lamivudine for example pause the virus from reproducing. The consequence of the antiretroviral drug treatment was simulated and well explained in the following graphs depicted.

Table 2: Variables Values of HBV In-vivo Dynamics

Symbols	Values	Source
$U(0)$	1.1×10^{11} cells	Eikenberry <i>et al.</i> (2009)
$L(0)$	0 cells	Assumed
$I(0)$	0 cells	Assumed
$T(0)$	1.1×10^{11} cells	Eikenberry <i>et al.</i> (2009)
$V(0)$	1.0×10^3 virons	Assumed

Table 3: Parameters Values of HBV In-vivo Dynamics

Symbols	Range Values	Sources	Used Values
ρ	0 – 1	Hews <i>et al.</i> (2009)	0.3
μ	0.002 – 0.053	Eikenberry <i>et al.</i> (2009), Long <i>et al.</i> (2008), Dong <i>et al.</i> (2010), Zheng <i>et al.</i> (2009)	0.0039
μ_1	0.001 – 0.002	Eikenberry <i>et al.</i> (2009), Long <i>et al.</i> (2008), Dong <i>et al.</i> (2010), Zheng <i>et al.</i> (2009)	0.0015
μ_2	0.001 - 0.003	Eikenberry <i>et al.</i> (2009), Long <i>et al.</i> (2008), Dong <i>et al.</i> (2010), Zheng <i>et al.</i> (2009), Hattaf and Yousfi (2011)	0.002
μ_3	0.58 – 0.693	Eikenberry <i>et al.</i> (2009), Long <i>et al.</i> (2008), Dong <i>et al.</i> (2010), Zheng <i>et al.</i> (2009), Hattaf and Yousfi (2011), Hews <i>et al.</i> (2009)	0.67
T^m	2×10^{11}	Eikenberry <i>et al.</i> (2009), Hews <i>et al.</i> (2009)	2×10^{11}
N	> 0	Eikenberry <i>et al.</i> (2009), Long <i>et al.</i> (2008), Hattaf, Tasiu <i>et al.</i> (2024b)	Varies
β	0 – 1	Eikenberry <i>et al.</i> (2009), Long <i>et al.</i> (2008), Hattaf and Yousfi (2011), Hews <i>et al.</i> (2009), Tasiu <i>et al.</i> (2024b)	Varies
γ	0 – 1	Tasiu <i>et al.</i> (2025)	Varies
ε_b	0 – 1	Long <i>et al.</i> (2008)	0.95
ε_p	0 – 1	Assumed	0.95
ψ	0 – 1	Assumed	0, 0.25, 0.5, 0.7, 1

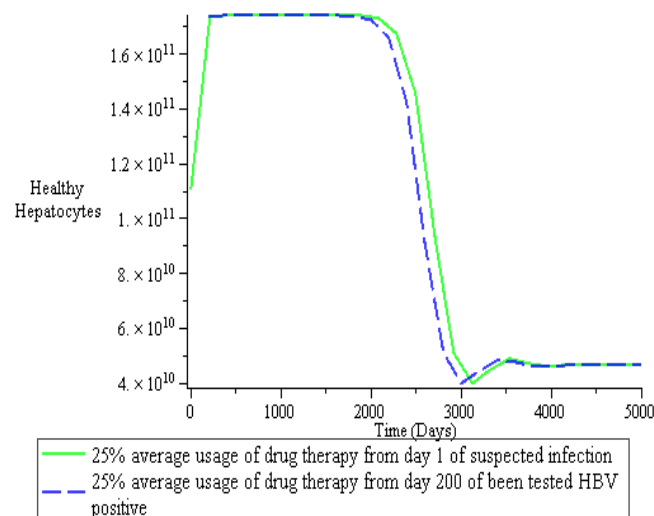
**Fig. 2: Comparison between the effects of 25% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested hepatitis B virus positive on healthy hepatocyte with $\psi = 0.25$.**

Fig. 2 explains the utilization rate of drug treatment between two individuals. It obviously appears that healthy (uninfected) hepatocytes increase up to the highest level for the individual whose drug intake started at the beginning of the infection, and later maintained a maximum stable level before 3000 days, and then slowly drop to nearly zero in 3000 days, and then continue to behave in an oscillatory behaviour. The decrease in the number of healthy (uninfected) cells in the first individual is far below that of the second individual whose drug intake 200 days after being infected. Although both of them later shared identical level. This mean that the second individual loses healthier (uninfected) cells for quite some times. Summarizing the results for both individuals indicates that 25% drug utilization rate is dangerous as lower healthy (uninfected) cells level means that there is a continuance of the disease.

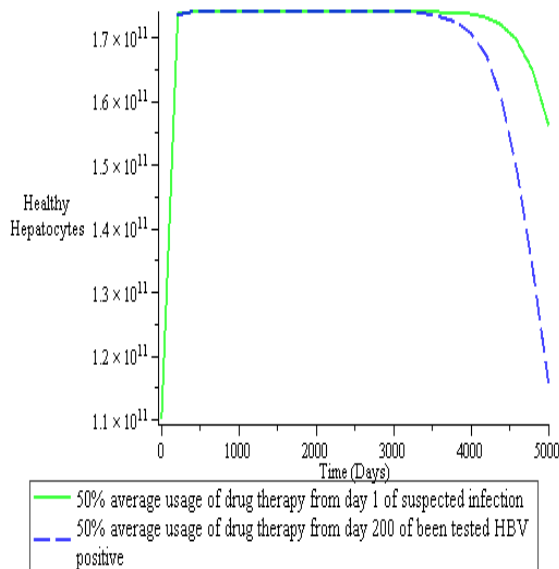


Fig. 3: Comparison between the effects of 50% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested hepatitis B virus positive on healthy hepatocyte with $\psi = 0.5$.

Clearly, the individual whose drug intake started immediately when the infection was noticed has a high level of having healthy (uninfected) cells compared to the individual whose drug intake is after 200 days of noticing the infection, even though both of them shared the same highest stable level close to 3500 days, with the healthy (uninfected) cells of the later decreases more than that of the former. The general implication for both is that there is a decrease in the number of healthy (uninfected) cells at the higher level in Fig. 3 compared to that of Fig. 2 with 50% drug usage, which make the treatment ineffective.

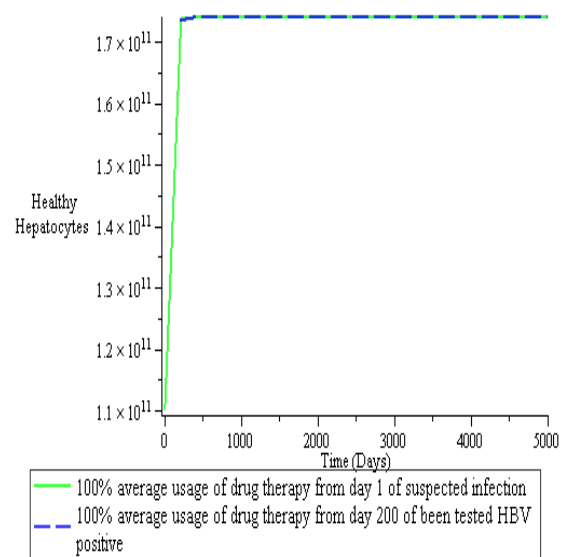


Fig. 4: Comparison between the effects of 100% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested hepatitis B virus positive on healthy hepatocyte with $\psi = 1.0$.

In this case, the individual whose drug intake start immediately when the infection is discovered has an advantage compared to the one who start the drug intake after 200 days, even though both of them maintained the same level of healthy (uninfected) cells at the highest stable level. Thus, Fig. 4 vividly show that a 100% average drug usage is the only choice of having a highest number of healthy (uninfected) cells that implies a total treatment to the infection.

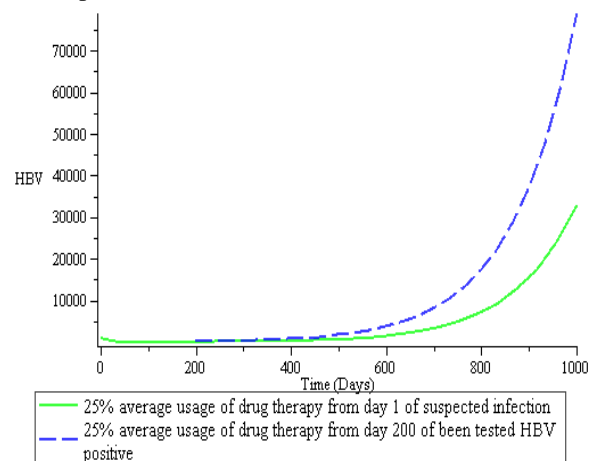


Fig. 5: Comparison between the effects of 25% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested positive on the hepatitis B virus with $\psi = 0.25$.

Fig. 5 divulges the consequences of the 25% drug rate usage on the viruses. Here, the virus rapidly increases for the individual whose drug intake is after 200 days of noticing the infection, compared to the one who

started on the first day of infection at the same 25% drug usage. The results of both individuals show rapid reappearance of the virus at different rate, which also signifies that the 25% drug usage does not eradicate the presence of the viruses.

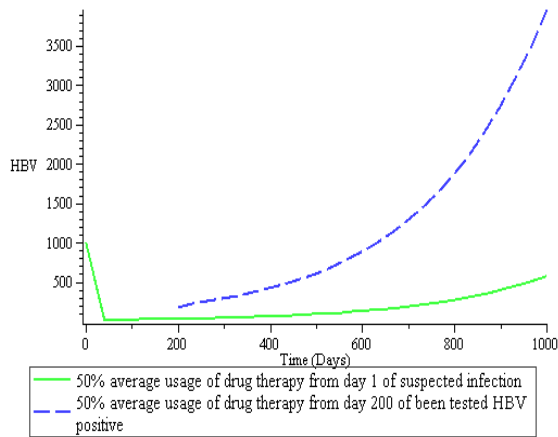


Fig. 6: Comparison between the effects of 50% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested positive on the hepatitis B virus with $\psi = 0.5$.

Like in Fig. 5, it is evident that the number of viruses associated with the individual whose drug intake is on the first day of noticing the infection is lower than the one who started the medication after 200 days. However, the number of viruses for both individuals here are less compared to that of the Fig. 5, because of the 50% drug rate usage. The result also show that 50% drug rate usage is not adequately sufficient to eliminate the viruses completely.

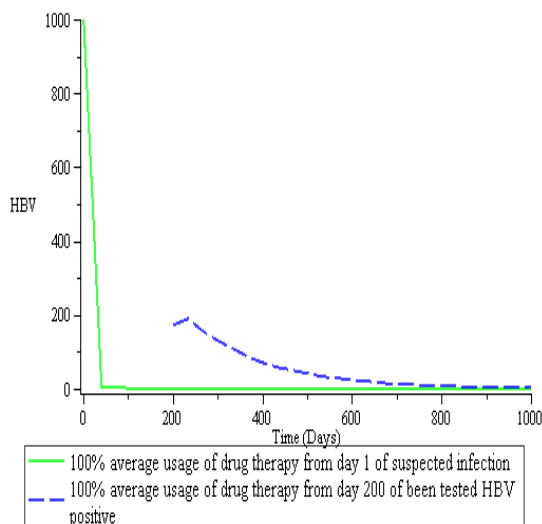


Fig. 7: Comparison between the effects of 100% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested positive on the hepatitis B virus with $\psi = 1.0$.

Fig 7 outline the importance of 100% average drug intake, which show that the level of viruses decreases faster initially for the individual whose drug intake is on the first day of noticing the virus infection that the other individual who started after 200 days. This imply that the viruses are eliminated faster when the drug usage is started earlier. It also indicates that the viruses are finally eliminated on both individuals because of the 100% average intake of the drugs, which signifies virus free of the liver.

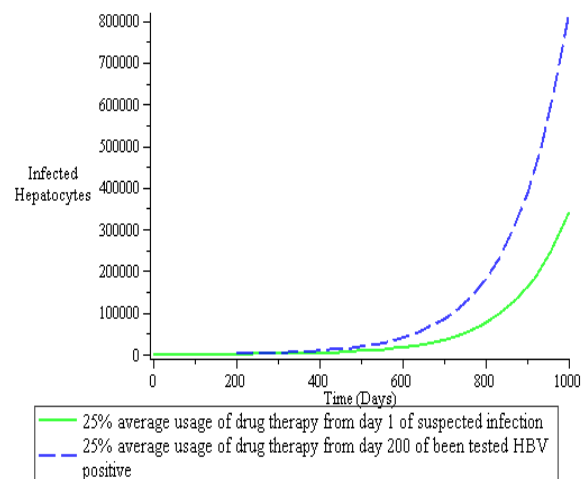


Fig. 8: Comparison between the effects of 25% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested hepatitis B virus positive on infected hepatocytes with $\psi = 0.25$

Fig. 8 have the same interpretation as Fig. 5 because they are similar in nature. The vital concept to consider for both Figures is that each has a higher number of viruses, indicating a higher number of infected (unhealthy) cells. In this case, for both the two individuals there is a slow grow for the number of infected (unhealthy) hepatocytes cells that occur at different rate, but the individual whose drug intake started at first day of detecting the infection has a lesser number of viruses compared to the second individual whose drug intake started after 200 days. We can as well easily distinguish an increase of viruses in both cases, which happened due to the 25% drug intake.

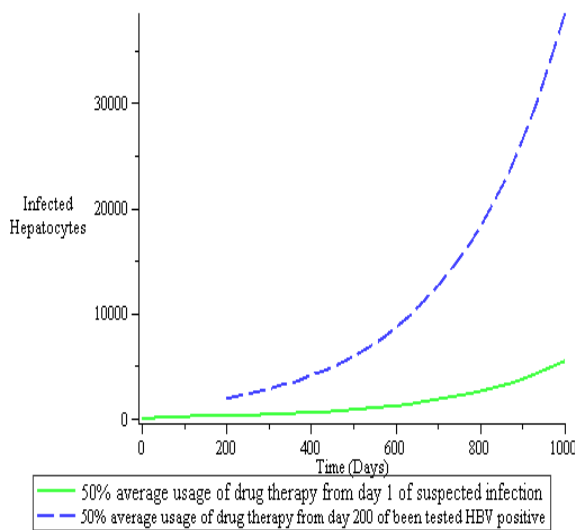


Fig. 9: Comparison between the effects of 50% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested hepatitis B virus positive on infected hepatocytes with $\psi = 0.5$.

The result obtained here is almost the same with what was obtained in Fig.6 with a little difference. The individual whose drug intake started at the first day of detecting the infection has a lower number of infected (unhealthy) hepatocytes, compared to the individual whose drug intake started after 200 days of detection. Because of the higher amount of drug intake in this situation which is 50%, the result is better than what was obtained in Fig.8, even though there exists a notable number infected (unhealthy) hepatocytes within the individuals, which definitely translates that the 50% drug administration cannot eliminate the infection completely.

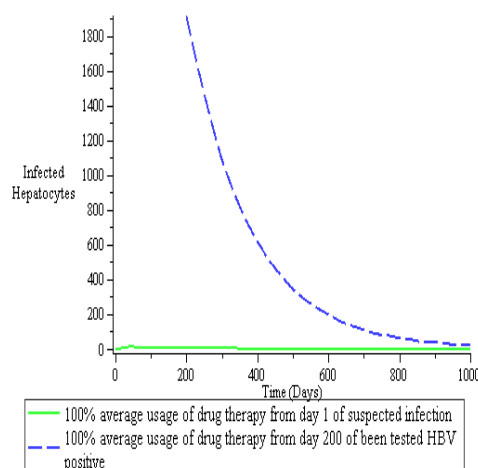


Fig. 10: Comparison between the effects of 100% average usage of drug therapy from day 1 of suspected infection and day 200 of been tested hepatitis B virus positive on infected hepatocytes with $\psi = 1.0$.

Referencing to Figures 7 and 8, the individual whose drug intake rate is 100% at the first day of detecting the infection is free from the virus from the beginning, compared to the individual who have the same drug administration rate but started after 200 days of noticing the infection, with both individual finally having no infected (unhealthy) cells. This implies that with 100% drug administration rate, there is an absence of infected (unhealthy) hepatocytes.

5.0 CONCLUSION

In this study within host mathematical model of hepatocytes virus was developed by considering the immune response (implicitly), the logistic hepatocytes cell growth, the standard incidence function, the function of the antiretroviral drug treatment and the cure rate of the infected (unhealthy) hepatocytes cells. The basic reproduction rate (R_c) of the model was obtained, where the effective reproduction number represent the average number of cases generated by the infected (unhealthy) cells if introduce into a susceptible (healthy) population of hepatocytes cells, with a control policy. The control policy is the utilization of the antiretroviral drug therapy.

Based on the research carried out in this paper, the following findings were made:

- i. If the policy for the prevention of viral reproduction within the host is adopted, and by embracing the use of an antiretroviral therapy, it will definitely go a long way in controlling the threat paused by the disease.
- ii. When there is an infection, a 100% average utilization of the antiretroviral drug is the only convinced way of managing the disease successfully, in order to minimize the mortality rate.
- iii. Once the infection is identified, it is advisable to begin the treatment on the first day of identifying the infection, then waiting for a long period of time before the administration of the antiretroviral drug therapy started.

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