Mathematical Modelling and Numerical Simulation with Applications, 2024, 4(5), 139–164

https://dergipark.org.tr/en/pub/mmnsa
ISSN Online: 2791-8564 / Open Access
https://doi.org/10.53391/mmnsa.1528691

RESEARCH PAPER

Mathematical modelling of the impact of vaccination, treatment and media awareness on the hepatitis B epidemic in Burkina Faso

Adama Kiemtoré [1,*,‡], **Wenddabo Olivier Sawadogo** [1,2,‡], **Pegdwindé Ousséni Fabrice Ouédraogo** [3,‡], **Fatima Aqel** [4,‡], **Hamza Alaa** [5,‡], **Kounpielime Sosthène Somda** [6,‡] **and Abdel Karim Sermé** [6,‡]

[1]LANIBIO Laboratory University Joseph Ki-Zerbo, 03 BP 7021, Ouagadougou, Burkina Faso, [2]LARED Laboratory University of Bernard Lédéa Ouédraogo 01 BP 346 Ouahigouya, Burkina Faso, [3]LaST Laboratory, University of Thomas Sankara, 12 BP 417 Ouagadougou 12, Burkina Faso, [4]Computer, Networks, Mobility and Modeling Laboratory (IR2M), Faculty of Sciences and Technics, Hassan First University, Settat 26000, Morocco, [5]LAMAI Laboratory, Faculty of Sciences and Technology, University of Cadi Ayyad, Abdelkrim, Khattabi, Marrakech 40000, Morocco, [6]Department of Gastroenterology, Yalgado Ouedraogo University Hospital, 01 BP 5234, Ouagadougou 01, Burkina Faso

*Corresponding Author
‡kiemtoreadama@gmail.com (Adama Kiemtoré); wenddabo@81gmail.com (Wendddabo Olivier Sawadogo); fabropof@gmail.com (Pegdwindé Ousséni Fabrice Ouédraogo); fatima.aqel@uhp.ac.ma (Fatima Aqel); hamza.alaa@ced.uca.ma (Hamza Alaa); sosthenesomda@gmail.com (Kounpielime Sosthène Somda); serme_abdel@yahoo.fr (Abdel Karim Sermé)

Abstract

Infection with the hepatitis B virus (HBV) remains a global public health issue. Particularly in Burkina Faso, HBV is a major public health concern due to its high prevalence and associated mortality. However, universal vaccination, treatment of chronic carriers, and awareness campaigns are currently employed means in Burkina Faso to combat the spread of HBV. Therefore, this paper aims to study the impact of these control measures on the expansion of this virus. This paper presents a mathematical model of vertically transmitted HBV that takes into account the progression to chronicity as a function of the age of the infected person, as well as vaccination, treatment of chronic carriers, and media awareness. After formulating the model and carrying out the mathematical analysis, we simulated the proposed model in Matlab, taking into account the various involved parameters. Finally, we presented the results of sensitivity analysis and numerical simulation. According to our model, with vaccination coverage of 30%, a 50% success rate of awareness campaigns and 20% effectiveness for the 10% of treated chronic carriers, the prevalence of hepatitis B infection could decrease down to 2% within thirty years in Burkina Faso.

Keywords: Hepatitis B; effective reproduction number; sensitivity analysis; vaccination

AMS 2020 Classification: 92D30; 37N25; 92B05

1 Introduction

The hepatitis B virus (HBV) is a significant global health threat, and its elimination by 2030 is a priority for the World Health Organization (WHO) [1]. Hepatitis B is not easily curable, and the majority of infected individuals do not exhibit any symptoms. These individuals, known as asymptomatic carriers, harbor the virus without displaying any clinical signs and play a crucial role in its transmission. Consequently, serological testing is essential for detection. Many infected people with HBV are unaware of their status and can unknowingly transmit the virus to others [2]. According to the WHO, approximately 2.5 billion people are or have been infected with HBV, representing about 30% of the global population [3]. Among them, 257 million suffer from chronic hepatitis B, including 60 million in Africa, with 136,000 deaths recorded in 2015 [2]. HBV infection is the second leading human carcinogen after tobacco [3]. The virus primarily targets the liver, causing damage through acute or chronic infections. Chronic infection can lead to severe complications such as cirrhosis and hepatocellular carcinoma (HCC) [2, 4]. Approximately 25% of individuals with chronic hepatitis B develop cirrhosis or liver cancer, which can result in premature death if untreated [4, 5].

The distribution of the prevalence of Hepatitis B surface Antigen (HBsAg) carrier status allows the division of the world into three prevalence zones, corresponding to different modes of transmission and risk levels: Low endemicity (prevalence below 2%): Infection risk below 20%, contamination at all ages (Western Europe, North America, Australia); Medium endemicity (prevalence between 2% and 7%): Infection risk between 20% and 60%, contamination at all ages (Middle East, Asia, China, Eastern Europe); High endemicity (prevalence above 8%): Infection risk above 60%, contamination primarily at birth or during early childhood (Sub-Saharan Africa, Southeast Asia) [5].

The distribution of HBV infection prevalence across Burkina Faso's regions is shown in Figure 1.

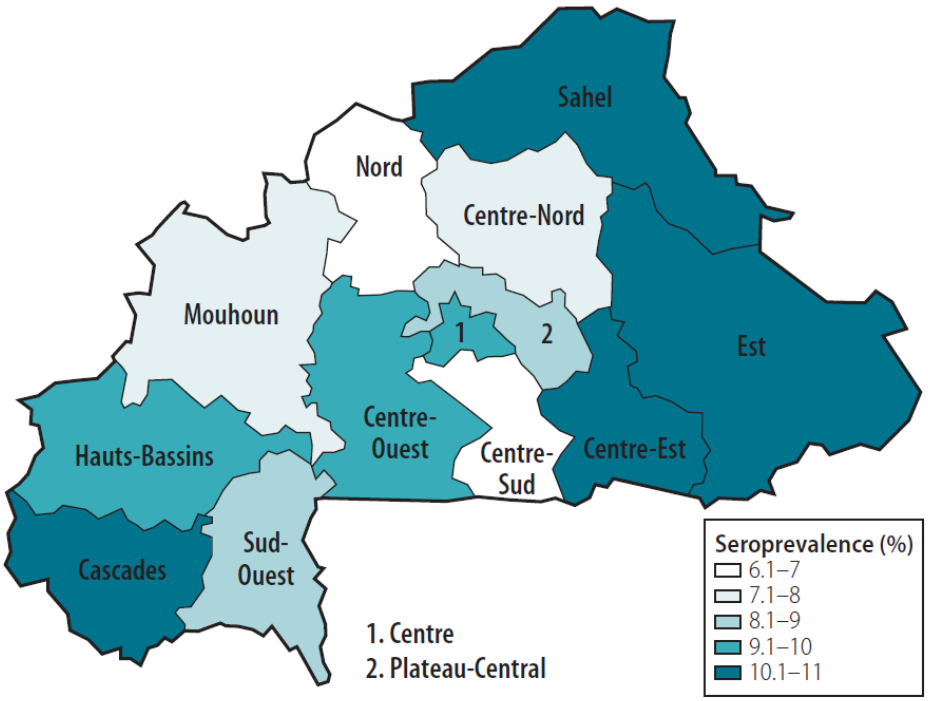

Figure 1. Burkina Faso distribution of HBV prevalence [7]

➤ **Received:** 08.08.2024 ➤ **Revised:** 18.12.2024 ➤ **Accepted:** 27.12.2024 ➤ **Published:** 31.12.2024

In Burkina Faso, the severity of this pathology is underscored by over 5,000 deaths per year [6]. The country is classified as highly endemic, with a seroprevalence rate over 8% [5]. A 2011 Demographic Health Survey estimated a seroprevalence of 9.1% [7, 8], while a study conducted in Ouagadougou, Burkina Faso's Capital showed a prevalence of 14.5% in the general population [9]. This indicates nearly 2 million people in Burkina Faso are infected with HBV [2, 8]. However, due to the asymptomatic nature of the disease, the actual number of infections may be higher.
In Burkina Faso, the situation concerning the hepatitis B epidemic remains alarming due to low vaccination coverage and a lack of information and education about the disease, which results in low screening rates and a faster spread of the hepatitis B virus (HBV). Over 90% of infected individuals are unaware of their status, serving as a significant reservoir for HBV transmission [2]. Additionally, the high costs of post-diagnostic medical tests discourage patients from seeking regular medical follow-ups. Finally, despite a significant reduction in treatment costs in recent years, access to care remains limited for chronic patients due to prices that are still unaffordable for many.
In high-endemic areas such as Burkina Faso, the primary mode of HBV transmission is mother-to-child transmission during childbirth or close contact in early childhood, referred to as vertical transmission [10]. Hepatitis B virus (HBV) is frequently transmitted from infected mothers to their newborns during childbirth, especially when the mother tests positive for the hepatitis B surface antigen (HBsAg) and the hepatitis B e antigen (HBeAg) [10]. Perinatal transmission or infection during early childhood has a high probability of resulting in chronic disease [2, 3, 11].
Additionally, HBV transmission can occur through high risky behaviors, including injecting drug use, unprotected sex with infected partners, or using unsterilized equipment for tattoos or piercings. Household transmission is also possible through shared personal items like razors, toothbrushes, or nail clippers that cause minor injuries. Unsanitary conditions and promiscuity further contribute to HBV spread, although these are not the only risk factors [10, 11].
The primary objectives of treating chronic hepatitis B are to enhance patient longevity and quality of life and to prevent disease evolvement into cirrhosis, liver cancer, and mortality. Effective treatment aims at eliminating HBsAg and achieve sustained suppression of viral load [10, 11]. In addition to vaccination, prevention efforts include the screening and immunization of susceptible children, adolescents, and adults. Awareness campaigns, particularly through modern media platforms, are crucial for preventing new infections.
In this paper, We here suggest a deterministic mathematical model of the type SVLACTR ($S = Susceptible$, $V = Vaccinated$, $L = Latent$, $A = Acute$, $C = Chronic$, $T = Treated$, $R = Recovered$), to study the impact of control strategies against the hepatitis B epidemic in Burkina Faso in order to issue recommendations. This is an extension of the model of hepatitis B virus transmission with vertical transmission in Burkina Faso with differential susceptibility and infectivity developed by Kiemtore et al. [12].
The novel aspects are:

- The inclusion of the ρ proportion of vertical transmission, which translates into the proportion of newly-infected infants who will develop acute hepatitis B only, and a $1-\rho$ proportion of newly-infected infants who will become chronic carriers.
- The parameter of awareness through media m, which will concern only susceptible S_3 people over 15 years of age.
- Chronic carriers treated by the T_r compartment.
- Unlike many hepatitis B models, our model will take into account a parameter τ representing the reactivation of the virus in cured chronic carriers, particularly in cases of immunosuppression, such as during dialysis or chemotherapy.

- Vaccination with a V compartment with loss of immunity.

The structure of this paper is detailed as below:

- The mathematical model is presented and described in Section 2.
- Section 3 focuses on the mathematical analysis of the model: Firstly, the existence, uniqueness, positivity, and boundedness of solutions will prove that the model is mathematically and epidemiologically well-posed. Secondly, the effective reproduction number $\mathcal{R}e$, which governs the spread of the hepatitis B virus, is calculated along with the equilibrium points. Then, when $\mathcal{R}e$ is less than 1, the equilibrium point without hepatitis B is globally stable, and the epidemic disappears, meaning that control strategies are effective. When $\mathcal{R}_e$ is more than 1, the endemic equilibrium point is globally stable, and the disease persists in the population.
- Section 4 is dedicated to sensitivity analysis. Thus, the sensitivity analysis of the effective reproduction rate $\mathcal{R}_e$ confirmed that universal vaccination of newborns and the treatment of chronic carriers are very effective in slowing down the spread of the hepatitis B virus in Burkina Faso.
- Section 5 discusses numerical results. Indeed, Numerical simulations have shown that universal vaccination, media-based awareness campaigns, and the treatment of chronic patients are effective means of controlling the spread of the hepatitis B virus. According to our model, with a vaccination coverage of 30%, a 50% success rate for awareness campaigns (television, radio, social networks), and 20% effectiveness for the 10% of treated chronic carriers, the prevalence of hepatitis B infection could decrease to 2% within thirty years, setting Burkina Faso in a zone of low endemicity.
- Section 6 concludes the study.

2 Model formulation

The total population $N(t)$ is categorized in ten different classes:

- $S_1(t)$: indicates the population of susceptible newborns aged 0 to 1 year.
- $S_2(t)$: Represents susceptible children aged 1 to 15 years.
- $S_3(t)$: Represents susceptible individuals aged over 15 years.
- $V(t)$: Refers to individuals of all ages who have been vaccinated and are therefore considered immunized from infection.
- $L_A(t)$: Represents latently infected individuals, who are infected but will not progress to acute infection.
- $L_C(t)$: Describes latently infected individuals who will eventually develop a chronic infection.
- $A(t)$: Describes acutely infected individuals.
- $C(t)$: are chronic carriers of the infection ;
- $Tr(t)$: It is the chronic patients who receive treatment.
- $R(t)$: Refers to recovered individuals who have recovered from the infection and may have developed immunity.

This is a vertical transmission model. In fact, despite systematic vaccination at birth only effective since January 2022 [2], vertical transmission remains persistent. We will limit our analysis to three distinct age groups. This division of susceptible individuals into three classes gives a reasonable representation of the epidemiological dynamics of the infection [2, 4, 13]. We have $\lambda\, N$ the total number of births. Here, vertical transmission will reduce births by $\lambda_1 A + \lambda_2 C + \lambda_3 T_r$. Thus, some of these virus-infected newborns will remain in the acute phase, while others will become chronic. Then, in the respective latent compartments L_A and L_C, $\rho(\lambda_1 A + \lambda_2 C + \lambda_3 T_r)$ and $(1-\rho)(\lambda_1 A + \lambda_2 C + \lambda_3 T_r)$ will appear. Table 1 gives a description of the parameters.

Table 1. Parameters used in the model

Parameters	Description
λ	population birth rate
λ_1	rate of newborns contaminated by acutely infected mothers
λ_2	rate of newborns contaminated by their chronic carrier mothers
λ_3	rate of newborns contaminated by their chronically ill mothers under treatment
ρ	rate of newborns contaminated by their mothers who will develop acute hepatitis B only
$1-\rho$	rate of newborns contaminated by their mothers who go on to develop chronic hepatitis B.
m	rate of efficiency of the media coverage within the community
θ_1	rate of vaccinated individuals in class S_1
θ_2	rate of vaccinated individuals in class S_2
θ_3	rate of vaccinated individuals in class S_2
ϕ_1	immunization failure rate of vaccinated individuals from S_1
ϕ_2	failure rate of immunisation of vaccinated individuals from S_2
ϕ_3	failure rate of immunisation of vaccinated individuals from S_3
p_1	proportion of infants aged 0 to 1 who are still susceptible to entering S_2.
p_2	proportion of infants aged 1 to 15 who are still susceptible to entering S_3
$\beta_{i,1}, i \in \{1,2,3\}$	transmission rate relative to acute individual
$\beta_{i,2}, i \in \{1,2,3\}$	transmission rate relative to chronic carriers
$\beta_{i,3}, i \in \{1,2,3\}$	transmission rate relative to treated chronic patients
α_1	probability that a susceptible S_1 is infected and enters a latent state L_A to become acute
$1-\alpha_1$	probability that a susceptible S_1 is infected and enters a latent state L_C to become chronic
α_2	probability that a susceptible S_2 is infected and enters a latent state L_A to become acute
$1-\alpha_2$	probability that a susceptible S_2 is infected and enters a latent state L_C to become chronic
α_3	probability that a susceptible S_3 is infected and enters a latent state L_A to become acute
$1-\alpha_3$	probability that a susceptible S_3 is infected and enters a latent state L_C to become chronic
δ_1	transfer rate of individuals from class L_A to class A
δ_2	transfer rate of individuals from class L_C to class C
ω_1	rate of chronic patients receiving treatment
ω_2	treatment failure rate for chronic patients
τ	probability of virus reactivation after the individual has recovered
γ_a	recovery rate for individuals with acute hepatitis B
γ_c	recovery rate for individuals with chronic hepatitis B
γ_{tr}	recovery rate for people with chronic hepatitis B who are treated
μ	rate of natural mortality
μ_a	death rate induced by acute hepatitis B
μ_c	death rate induced by chronic hepatitis B
μ_{tr}	mortality rate due to HBV in chronically treated patients

For a better formulation of the model, we have considered the following statements:

(1) Each susceptibility class S_1, S_2 and S_3 divided into two groups: The first group moves into the acute latent state L_A and eventually develops acute hepatitis with probability $\alpha_i, i = 1,2,3$ The second group moves into the chronic latent state L_C becoming chronic C carriers with probability $(1-\alpha_i), i = 1,2,3$ [12, 14, 15].

(2) The recruitment rate is $\lambda N - \lambda_1 A - \lambda_2 C - \lambda_3 T_r$ [12].

(3) Vertical transmission occurs via acute infections, chronic carriers and treated patients, with one proportion $\rho(\lambda_1 A + \lambda_2 C + \lambda_3 T_r)$ only becoming acute, and the other proportion $(1-\rho)(\lambda_1 A +$

$\lambda_2 C + \lambda_3 T_r$) becoming chronic.

(4) The rate of contact required for a person to become infected with the virus will depend on all the infectious individuals: A, C, T_r which gives [14, 16–18]:
$\beta_1 = \frac{\beta_{1,1}A + \beta_{1,2}C + \beta_{1,3}T_r}{N}$, $\beta_2 = \frac{\beta_{2,1}A + \beta_{2,2}C + \beta_{2,3}T_r}{N}$, $\beta_3 = \frac{\beta_{3,1}A + \beta_{3,2}C + \beta_{3,3}T_r}{N}$ are respectively the adequate contact rates for a susceptible S_1 (susceptible individual to age $0-1$ years), S_2(susceptible individual to age 1 to 15) and S_3(susceptible individual over 15 years of age) to be infected by the hepatitis B virus [16].

(5) Acute hepatitis includes fulminant hepatitis, which is extremely rare [3].

(6) All those who do not die of hepatitis B, i.e. those belonging to the compartments S_1, S_2, S_3, V, L_A, L_C, R have a natural mortality rate of μ [12].

(7) In Burkina Faso, The prognosis for the evolution of this infection towards chronicity is inversely proportional to the age at which a person becomes infected [3, 9, 12, 14].

(8) The HBV vaccine must be stored at low temperature, faillure to do so can undermine the effectiveness of the vaccine at its administration [2, 3].

(9) Hepatitis B treatment will only affect chronic carriers [2, 19].

(10) Viral reactivation will concern chronically recovered carriers [20], i.e. this phenomenon is neglected in patients who have recovered from the acute form.

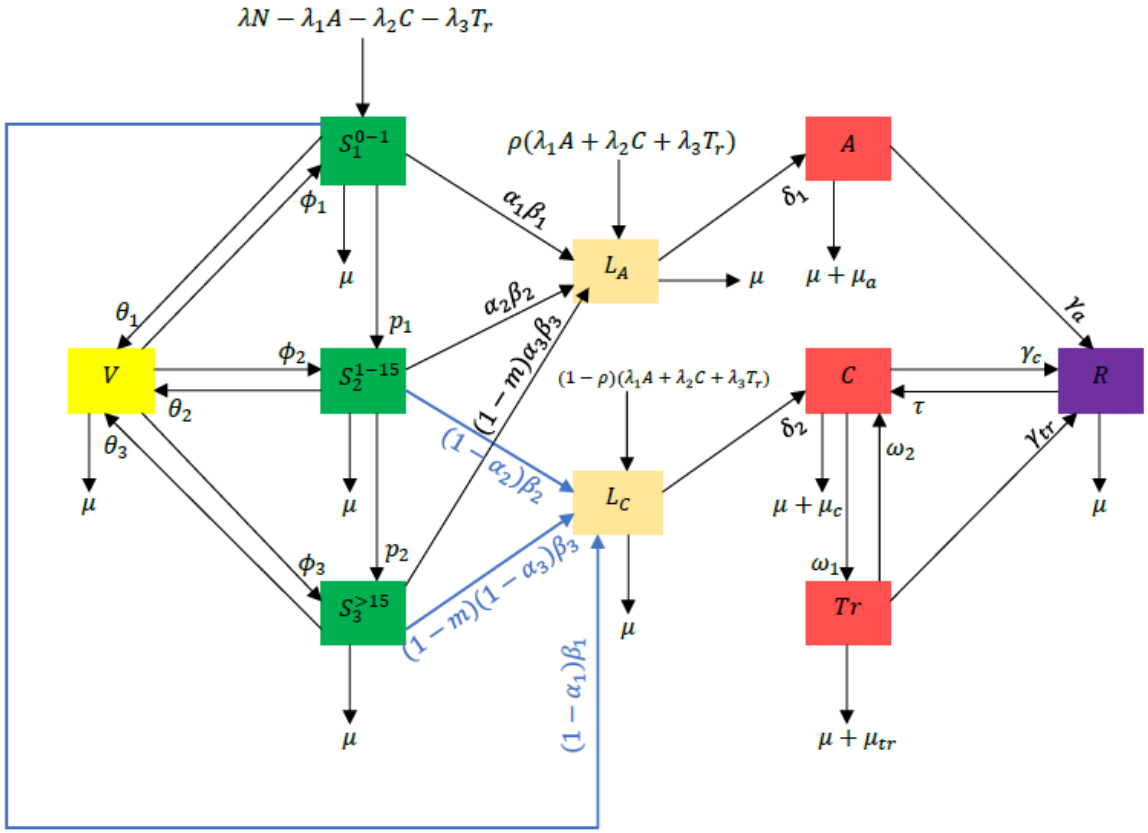

Figure 2. HVB model with vertical transmission, vaccination, treatment and awareness

Based on all the information provided above and the Table 1, we have drawn the compartmental diagram illustrated in Figure 2. The following system of differential equations is obtained:

$$
\begin{cases}
\dfrac{dS_1}{dt} = \lambda\, N - \lambda_1 A - \lambda_2 C - \lambda_3 T_r + \phi_1 V - \left(\dfrac{\beta_{1,1}A + \beta_{1,2}C + \beta_{1,3}T_r}{N}\right) S_1 - (\theta_1 + p_1 + \mu)\, S_1, \\
\dfrac{dS_2}{dt} = p_1 S_1 + \phi_2 V - \left(\dfrac{\beta_{2,1}A + \beta_{2,2}C + \beta_{2,3}T_r}{N}\right) S_2 - (\theta_2 + p_2 + \mu)\, S_2, \\
\dfrac{dS_3}{dt} = p_2 S_2 + \phi_3 V - (1-m)\left(\dfrac{\beta_{3,1}A + \beta_{3,2}C + \beta_{3,3}T_r}{N}\right) S_3 - (\theta_3 + \mu)\, S_3, \\
\dfrac{dV}{dt} = \theta_1 S_1 + \theta_2 S_2 + \theta_3 S_3 - (\phi_1 + \phi_2 + \phi_3 + \mu)\, V, \\
\dfrac{dL_A}{dt} = \alpha_1 \left(\dfrac{\beta_{1,1}A + \beta_{1,2}C + \beta_{1,3}T_r}{N}\right) S_1 + \alpha_2 \left(\dfrac{\beta_{2,1}A + \beta_{2,2}C + \beta_{2,3}T_r}{N}\right) S_2 \\
\qquad + \rho(\lambda_1 A + \lambda_2 C + \lambda_3 T_r) + \alpha_3\,(1-m) \left(\dfrac{\beta_{3,1}A + \beta_{3,2}C + \beta_{3,3}T_r}{N}\right) S_3 - (\delta_1 + \mu)\, L_A, \\
\dfrac{dL_C}{dt} = (1-\alpha_1) \left(\dfrac{\beta_{1,1}A + \beta_{1,2}C + \beta_{1,3}T_r}{N}\right) S_1 + (1-\alpha_2) \left(\dfrac{\beta_{2,1}A + \beta_{2,2}C + \beta_{2,3}T_r}{N}\right) S_2 \\
\qquad + (1-\rho)(\lambda_1 A + \lambda_2 C + \lambda_3 T_r) + (1-\alpha_3)\,(1-m) \left(\dfrac{\beta_{3,1}A + \beta_{3,2}C + \beta_{3,3}T_r}{N}\right) S_3 - (\delta_2 + \mu)\, L_C, \\
\dfrac{dA}{dt} = \delta_1 L_A - (\mu_a + \gamma_a + \mu)\, A, \\
\dfrac{dC}{dt} = \delta_2 L_C + \omega_2 T_r + \tau R - (\gamma_c + \omega_1 + \mu + \mu_c)\, C, \\
\dfrac{dT_r}{dt} = \omega_1 C - (\gamma_{tr} + \omega_2 + \mu + \mu_{tr})\, T_r, \\
\dfrac{dR}{dt} = \gamma_a A + \gamma_c C + \gamma_{tr} T_r - (\tau + \mu)\, R.
\end{cases}
\tag{1}
$$

Let :

$$
\begin{aligned}
&\kappa_1 = \theta_1 + p_1 + \mu,\ \kappa_2 = \theta_2 + p_2 + \mu,\ \kappa_3 = \theta_3 + \mu,\ \kappa_4 = \phi_1 + \phi_2 + \phi_3 + \mu,\ \kappa_5 = \delta_1 + \mu, \\
&\kappa_6 = \delta_2 + \mu,\ \kappa_7 = \mu + \mu_a + \gamma_a,\ \kappa_8 = \gamma_c + \omega_1 + \mu + \mu_c,\ \kappa_9 = \gamma_t + \omega_2 + \mu + \mu_t,\ \kappa_{10} = \tau + \mu.
\end{aligned}
\tag{2}
$$

The total population is:

$$
N(t) = S_1(t) + S_2(t) + S_3(t) + V(t) + L_A(t) + L_C(t) + A(t) + C(t) + T_r(t) + R(t).
$$

Let also:

$$
s_1 = \frac{S_1}{N},\ s_2 = \frac{S_2}{N},\ s_3 = \frac{S_3}{N},\ v = \frac{V}{N},\ l_a = \frac{L_A}{N},\ l_c = \frac{L_C}{N},\ a = \frac{A}{N},\ c = \frac{C}{N},\ t_r = \frac{T_r}{N},\ r = \frac{R}{N}.
$$

Normalizing model (1), we get:

$$
\begin{cases}
\frac{ds_1}{dt} = \lambda - \lambda_1 a - \lambda_2 c - \lambda_3 t_r + \phi_1 v - (\beta_{1,1} a + \beta_{1,2} c + \beta_{1,3} t_r)\, s_1 - \kappa_1 s_1, \\
\frac{ds_2}{dt} = p_1 s_1 + \phi_2 v - (\beta_{2,1} a + \beta_{2,2} c + \beta_{2,3} t_r)\, s_2 - \kappa_2 s_2, \\
\frac{ds_3}{dt} = p_2 s_2 + \phi_3 v - (1-m)\,(\beta_{3,1} a + \beta_{3,2} c + \beta_{3,3} t_r)\, s_3 - \kappa_3 s_3, \\
\frac{dv}{dt} = \theta_1 s_1 + \theta_2 s_2 + \theta_3 s_3 - \kappa_4 v, \\
\frac{dl_a}{dt} = \alpha_1 (\beta_{1,1} a + \beta_{1,2} c + \beta_{1,3} t_r)\, s_1 + \alpha_2 (\beta_{2,1} a + \beta_{2,2} c + \beta_{2,3} t_r)\, s_2 \\
\qquad +\rho(\lambda_1 a + \lambda_2 c + \lambda_3 t_r) + \alpha_3 (1-m)\,(\beta_{3,1} a + \beta_{3,2} c + \beta_{3,3} t_r)\, s_3 - \kappa_5 l_a, \\
\frac{dl_c}{dt} = (1-\alpha_1)\,(\beta_{1,1} a + \beta_{1,2} c + \beta_{1,3} t_r)\, s_1 + (1-\alpha_2)(\beta_{2,1} a + \beta_{2,2} c + \beta_{2,3} t_r) s_2 \\
\qquad +(1-\rho)(\lambda_1 a + \lambda_2 c + \lambda_3 t_r) + (1-\alpha_3)\,(1-m)\,(\beta_{3,1} a + \beta_{3,2} c + \beta_{3,3} t_r)\, s_3 - \kappa_6 l_c, \\
\frac{da}{dt} = \delta_1 l_a - \kappa_7 a, \\
\frac{dc}{dt} = \delta_2 l_c + \omega_2 t_r + \tau r - \kappa_8 c, \\
\frac{dt_r}{dt} = \omega_1 c - \kappa_9 t_r, \\
\frac{dr}{dt} = \gamma_a a + \gamma_c c + \gamma_{tr} t_r - \kappa_{10} r, \\
s_1(0) = s_{1,0},\ s_2(0) = s_{2,0},\ s_3(0) = s_{3,0},\ v(0) = v_0,\ l_a(0) = la_0, \\
l_c(0) = lc_0, a(0) = a_0,\ c(0) = c_0,\ t_r(0) = t_{r,0},\ r(0) = r_0.
\end{cases}
\tag{3}
$$

3 Model analysis

All thirty-two parameters : λ, γ_a, γ_c, γ_{tr}, λ_1, λ_2, λ_3, p_1, p_2, δ_1, δ_2, ϕ_1, ϕ_2, ϕ_3, θ_1, θ_2, θ_3, μ, μ_a, μ_c, μ_{tr}, ω_1, $\omega_2, \beta_{1,1}$, $\beta_{1,2}$, $\beta_{1,3}$, $\beta_{2,1}$, $\beta_{2,2}$, $\beta_{2,3}$, $\beta_{3,1}$, $\beta_{3,2}$, $\beta_{3,3}$ used for the hepatitis B model are non-negative, since the model describes human population dynamics.

Positivity, boundedness and global existence of solutions

Theorem 1 *For all initial condition* $(s_{1,0}, s_{2,0}, s_{3,0}, v_0, la_0, lc_0, a_0, c_0, tr_0, r_0) \in \mathbb{R}^{10}_+$,

$$\text{such that} \quad s_{1,0} + s_{2,0} + s_{3,0} + v_0 + la_0, lc_0 + c_0 + tr_0 + r_0 = 1.$$

There is therefore a unique, non-negative, bounded global solution for system (3).
In addition, for all $t \geq 0$,

(i)

$$\Omega = \left\{ (s_1(t), s_2(t), s_3(t), v(t), la(t), lc(t), a(t), c(t), t_r(t), r(t)) \mid\ 0 \leq \Theta(t) \leq \frac{\lambda}{\mu} + \Theta(0) \right\}, \tag{4}$$

where

$$\Theta(t) = s_1(t) + s_2(t) + s_3(t) + v(t) + la(t) + lc(t) + a(t) + c(t) + t_r(t) + r(t).$$

(ii) *Furthermore, if*

$$\begin{aligned}
s_{1,0} &\leq \left[\frac{\lambda}{\kappa_1} + \frac{\phi_1\lambda\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)}{\kappa_1\left[\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3\right]}\right], \\
s_{2,0} &\leq \left[\frac{\lambda p_1}{\kappa_1\kappa_2} + \frac{\left(p_1\phi_1 + \kappa_1\phi_2\right)\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)\lambda}{\kappa_1\kappa_2\left[\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3\right]}\right], \\
s_{3,0} &\leq \left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3} + \frac{\left(p_1 p_2\phi_1 + p_2\kappa_1\phi_2 + \kappa_1\kappa_2\phi_3\right)\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)\lambda}{\kappa_1\kappa_2\kappa_3\left[\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3\right]}\right], \\
v_0 &\leq \frac{\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)\lambda}{\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3}.
\end{aligned} \tag{5}$$

then

$$s_1(t) \leq s_{1,0},\ \ s_2(t) \leq s_{2,0},\ \ s_3(t) \leq s_{3,0},\ \ v(t) \leq v_0. \tag{6}$$

Proof

(i) To guarantee the existence of a local solution, all functions in system (3) are locally continuous and satisfy the Lipschitz condition. Consequently, there is a unique local solution for $t \in [T_0, T_{max})$ [21].
By showing that the elements of the solution vector $(s_1(t),\ s_2(t),\ s_3(t),\ v(t),\ l_a(t),\ l_c(t),\ a(t),\ c(t),\ t_r(t),\ r(t))$ are uniformly bounded on any bounded interval $[0, T_{max})$, we know that $T_{max} = \infty$.
Noting the elements of Ω by $x = (s_1,\ s_2,\ s_3,\ v,\ l_a,\ l_c,\ a,\ c,\ t_r,\ r)^t$ we rewrite model (3) as the following autonomous differential system:

$$\frac{dx_i}{dt} = f_i(x); \quad i=1;\ 2;\ldots;\ 10.$$

We notice that the components of the vector f_i quasi-positive. So, given that the initial conditions are non-negative, this entails that the components of the solution remain non-negative for all $t \in [T_0, T_{max})$ [12, 21, 22].
Now, consider defining the function Θ as:

$$\Theta(t) = s_1(t) + s_2(t) + s_3(t) + v(t) + l_a(t) + l_c(t) + a(t) + c(t) + tr(t) + r(t).$$

Adding up the ten equations of system (3), we find that

$$\begin{cases} \dfrac{d\Theta}{dt} \leq \lambda - \mu\Theta(t), \\ \Theta(0) = 1. \end{cases} \tag{7}$$

Integrating Eq. (7) over the interval $[0, t]$, for all $t_0 < t < T$, one gets

$$0 \leq \Theta(t) \leq \frac{\lambda}{\mu} + 1.$$

Consequently, we show that for $T_{max} = \infty$ and the existence of a unique, non-negative and bounded global solution is established.

(ii) Note that s_1 satisfies:

$$\begin{cases} \dfrac{ds_1}{dt} \leq \lambda + \phi_1 \left[\dfrac{\lambda\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)}{\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3} \right] - \kappa_1 s_1(t), \\ s_1(0) = s_{1,0}. \end{cases} \tag{8}$$

Let us integrate Eq. (8) over $[0, t]$ for all $t > 0$.
One obtains:

$$s_1(t)\exp(\kappa_1(t)) \leq \frac{\lambda + \phi_1 \left[\dfrac{\lambda\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)}{\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3} \right]}{\kappa_1} \times \left(\exp(\kappa_1(t)) - 1\right) + s_{1,0}.$$

Since

$$s_{1,0} \leq \frac{\lambda + \phi_1 \left[\dfrac{\lambda\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)}{\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3} \right]}{\kappa_1},$$

which implies that $s_1(t) \leq s_{1,0}$. We apply the same reasoning to $s_2(t)$, $s_3(t)$ and $v(t)$.

Thus, model (3) is mathematically and epidemiologically well-posed inside of Ω.

Hepatitis B free equilibrium point $\mathcal{E}_0$

Hepatitis B free equilibrium point is a theoretical state in the mathematical model of hepatitis B where no individuals in the population are infected with the hepatitis B virus. At this equilibrium, all model compartments associated with infected individuals—such as latent infected, acute infected, chronic carriers, and chronic cases under treatment—have a population size equal to zero, while the susceptible population remains constant. This equilibrium depends on the effective reproduction number $\mathcal{R}_e$. If $\mathcal{R}_e < 1$, the disease-free equilibrium is globally stable, meaning that hepatitis B cannot spread within the population [23].
Model (3) admits a unique Hepatitis B free equilibrium point given by:

$$\mathcal{E}_0 = \left(s_{1,0},\ s_{2,0},\ s_{3,0},\ v_0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0\right),$$

where

$$s_{1,0} = \left[\frac{\lambda}{\kappa_1} + \frac{\phi_1\lambda\left(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2\right)}{\kappa_1\left[\kappa_2\kappa_3\left(\kappa_1\kappa_4 - \theta_1\phi_1\right) - \phi_1 p_1\left(\theta_2\kappa_3 + \theta_3 p_2\right) - \theta_3\kappa_1\left(\phi_2 p_1 + \phi_3\kappa_2\right) - \theta_2\phi_2\kappa_1\kappa_3\right]} \right],$$

$$s_{2,0} = \left[\frac{\lambda p_1}{\kappa_1\kappa_2} + \frac{(p_1\phi_1 + \kappa_1\phi_2)(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right],$$

$$s_{3,0} = \left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3} + \frac{(p_1 p_2\phi_1 + p_2\kappa_1\phi_2 + \kappa_1\kappa_2\phi_3)(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\kappa_3[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right],$$

$$v_0 = \frac{(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)\lambda}{\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3}.$$

Effective reproduction number $\mathcal{R}_e$

The effective reproduction number $\mathcal{R}_e$ represents the average number of secondary cases generated by a single infectious individual at a specific point in time, in a population where some individuals may already be immune or where control measures are in place (e.g. vaccination, media awareness, treatment, etc) [24–27]. However, if $\mathcal{R}_e > 1$, hepatitis B continues to spread but if $\mathcal{R}_e < 1$, hepatitis B transmission declines, and the epidemic is likely to be contained.
Let us consider model (3), The effective reproduction number calculated using the "next generation matrix" method of Driessche and Watmough [23] is given by:

$$\mathcal{R}_e = \frac{1}{2}\left[\frac{\delta_1\eta_1}{\kappa_5\kappa_7} + \frac{\delta_2\kappa_9\eta_5 + \delta_2\omega_1\eta_6}{\kappa_6(\kappa_8\kappa_9 - \omega_1\omega_2)} + \left(\left(\frac{\delta_1\eta_1}{\kappa_5\kappa_7} - \frac{\delta_2\kappa_9\eta_5 + \delta_2\omega_1\kappa_6}{\kappa_6(\kappa_8\kappa_9 - \omega_1\omega_2)}\right)^2 + \frac{4(\delta_2\kappa_9\eta_2 + \delta_2\omega_1\eta_3)\delta_1\eta_4}{\kappa_5\kappa_6\kappa_7(\kappa_8\kappa_9 - \omega_1\omega_2)}\right)^{\frac{1}{2}}\right].$$

Where κ_1, κ_2, κ_3, κ_4, κ_5, κ_6, κ_7, κ_8, κ_9 are given by (2) and η_1, η_2, η_3, η_4, η_5, η_6 are given by:

$$\begin{aligned}\eta_1 = {} & \alpha_1\beta_{1,1}\left[\frac{\lambda}{\kappa_1} + \frac{\phi_1\lambda(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)}{\kappa_1[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right] \\ & + \alpha_2\beta_{2,1}\left[\frac{\lambda p_1}{\kappa_1\kappa_2} + \frac{(p_1\phi_1 + \kappa_1\phi_2)(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right] \\ & + \rho\lambda_1 + \alpha_3(1-m)\beta_{3,1} \\ & \times\left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3} + \frac{(p_1 p_2\phi_1 + p_2\kappa_1\phi_2 + \kappa_1\kappa_2\phi_3)(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\kappa_3[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right],\end{aligned}$$

$$\begin{aligned}\eta_2 = {} & \alpha_1\beta_{1,2}\left[\frac{\lambda}{\kappa_1} + \frac{\phi_1\lambda(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)}{\kappa_1[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right] \\ & + \alpha_2\beta_{2,2}\left[\frac{\lambda p_1}{\kappa_1\kappa_2} + \frac{(p_1\phi_1 + \kappa_1\phi_2)(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right] \\ & + \rho\lambda_2 + \alpha_3(1-m)\beta_{3,2} \\ & \times\left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3} + \frac{(p_1 p_2\phi_1 + p_2\kappa_1\phi_2 + \kappa_1\kappa_2\phi_3)(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\kappa_3[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right],\end{aligned}$$

$$\eta_3 = \alpha_1\beta_{1,3}\left[\frac{\lambda}{\kappa_1} + \frac{\phi_1\lambda(\theta_1\kappa_2\kappa_3 + \theta_2\kappa_3 p_1 + \theta_3 p_1 p_2)}{\kappa_1[\kappa_2\kappa_3(\kappa_1\kappa_4 - \theta_1\phi_1) - \phi_1 p_1(\theta_2\kappa_3 + \theta_3 p_2) - \theta_3\kappa_1(\phi_2 p_1 + \phi_3\kappa_2) - \theta_2\phi_2\kappa_1\kappa_3]}\right]$$

$$+\alpha_2\beta_{2,3}\left[\frac{\lambda p_1}{\kappa_1\kappa_2}+\frac{(p_1\phi_1+\kappa_1\phi_2)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right]$$
$$+\rho\lambda_3+\alpha_3(1-m)\beta_{3,3}$$
$$\times\left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3}+\frac{(p_1p_2\phi_1+p_2\kappa_1\phi_2+\kappa_1\kappa_2\phi_3)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\kappa_3\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right],$$

$$\eta_4=(1-\rho)\lambda_1+(1-\alpha_1)\beta_{1,1}$$
$$\times\left[\frac{\lambda}{\kappa_1}+\frac{\phi_1\lambda(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)}{\kappa_1\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right]+(1-\alpha_2)$$

$$\times\beta_{2,1}\left[\frac{\lambda p_1}{\kappa_1\kappa_2}+\frac{(p_1\phi_1+\kappa_1\phi_2)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right]$$
$$+(1-\alpha_3)(1-m)\beta_{3,1}\times$$
$$\times\left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3}+\frac{(p_1p_2\phi_1+p_2\kappa_1\phi_2+\kappa_1\kappa_2\phi_3)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\kappa_3\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right],$$

$$\eta_5=(1-\rho)\lambda_2+(1-\alpha_1)\beta_{1,2}$$
$$\times\left[\frac{\lambda}{\kappa_1}+\frac{\phi_1\lambda(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)}{\kappa_1\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right]+(1-\alpha_2)$$

$$\times\beta_{2,2}\left[\frac{\lambda p_1}{\kappa_1\kappa_2}+\frac{(p_1\phi_1+\kappa_1\phi_2)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right]$$
$$+(1-\alpha_3)(1-m)\beta_{3,2}$$
$$\times\left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3}+\frac{(p_1p_2\phi_1+p_2\kappa_1\phi_2+\kappa_1\kappa_2\phi_3)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\kappa_3\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right],$$

$$\eta_6=(1-\rho)\lambda_3+(1-\alpha_1)\beta_{1,3}$$
$$\times\left[\frac{\lambda}{\kappa_1}+\frac{\phi_1\lambda(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)}{\kappa_1\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right]+(1-\alpha_2)$$

$$\times\beta_{2,3}\left[\frac{\lambda p_1}{\kappa_1\kappa_2}+\frac{(p_1\phi_1+\kappa_1\phi_2)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right]$$
$$+(1-\alpha_3)(1-m)\beta_{3,3}$$
$$\times\left[\frac{\lambda\, p_1 p_2}{\kappa_1\kappa_2\kappa_3}+\frac{(p_1p_2\phi_1+p_2\kappa_1\phi_2+\kappa_1\kappa_2\phi_3)(\theta_1\kappa_2\kappa_3+\theta_2\kappa_3 p_1+\theta_3 p_1 p_2)\lambda}{\kappa_1\kappa_2\kappa_3\left[\kappa_2\kappa_3(\kappa_1\kappa_4-\theta_1\phi_1)-\phi_1 p_1(\theta_2\kappa_3+\theta_3 p_2)-\theta_3\kappa_1(\phi_2 p_1+\phi_3\kappa_2)-\theta_2\phi_2\kappa_1\kappa_3\right]}\right].$$

Global stability analysis of $\mathcal{E}_0$

If $\mathcal{R}_e<1$ and with Eqs. (5)-(6), then $\mathcal{E}_0$ is global asymptotically stable.
The global stability of $\mathcal{E}_0=(s_{1,0},\ s_{2,0}, s_{3,0},\ v_0,\ 0,\ 0,\ 0,\ 0,\ 0,\ 0)$ of model (3) would be explored in this subsection and as in [29–32], the Castillo Chavez method is used for this purpose. Therefore, by applying this method of Castillo Chavez, firstly the given problem (3) is converted into the

following to sub models given as :

$$\begin{cases} \dfrac{dX_1}{dt} = F(X_1, X_2), \\ \dfrac{dX_2}{dt} = G(X_1, X_2), \\ G(X_1, 0) = 0. \end{cases}$$

Where X_1 and X_2 indicates the population of uninfected individuals, and infected individuals, respectively. The following conditions (A_1) and (A_2) must be satisfied to guarantee the global asymptotic stability.

A_1 : If $\dfrac{dX_1}{dt} = F(X_1, 0)$, X_0 is global asymptotically stable.
A_2 : $G(X_1, X_2) = AX_2 - \bar{G}(X_1, X_2)$, here $\bar{G}(X_1, X_2) \geq 0$.

Where $\bar{G}(X_1, X_2) \geq 0$ for $(X_1, X_2) \in \Omega$, the matrix A is a M-matrix whose off diagonal elements are non-negative and Ω is the region in which the model has biological sense. In the system (3) $X_1 = (s_1, s_2, s_3, v, r) \in \mathbf{R}^5$ and $X_2 = (l_a, l_c, a, c, t_r) \in \mathbf{R}^5$.
To ensure the global asymptotic stability of the Hepatitis B-free equilibrium point $\mathcal{E}_0$, the results mentioned above were used as in [29–32].

The endemic equilibrium point $\mathcal{EE}$

The endemic equilibrium in an epidemiological model represents a stable situation where the disease persists consistently within a population over the long term, with a fixed average number of new cases and infected carriers.

Existence of $\mathcal{EE}$

System (3) admits a unique endemic equilibrium
$\mathcal{EE} = (s_1^*,\ s_2^*,\ s_3^*,\ v^*,\ l_a^*,\ l_c^*,\ a^*,\ c^*,\ t_r^*,\ r^*)$ whenever $\mathcal{R}_e > 1$.
By putting the right hand side of system (3) to zero, and keeping each state variable different from zero $(s_1 \neq 0,\ s_2 \neq 0,\ s_3 \neq 0,\ v \neq 0,\ l_a \neq 0,\ l_c \neq 0,\ a \neq 0,\ c \neq 0,\ t_r \neq 0,\ r \neq 0)$ then one obtains:

$$s_1^* = \frac{\lambda - \lambda_1 a^* - \lambda_2 c^* - \lambda_3 t_r^* + \phi_1 v^*}{\kappa_1 + \beta_{1,1} a^* + \beta_{1,2} c^* + \beta_{1,3} t_r^*}, \quad s_2^* = \frac{p_1 s_1^* + \phi_2 v^*}{\kappa_2 + \beta_{2,1} a^* + \beta_{2,2} c^* + \beta_{2,3} t_r^*},$$

$$s_3^* = \frac{p_2 s_2^* + \phi_3 v^*}{\kappa_3 + (1-m)\left(\beta_{3,1} a^* + \beta_{3,2} c^* + \beta_{3,3} t_r^*\right)}, \quad v^* = \frac{\theta_1 s_1^* + \theta_2 s_2^* + \theta_3 s_3^*}{\kappa_4},$$

$$l_a^* = \frac{\rho\left(\lambda_1 a^* + \lambda_2 c^* + \lambda_3 t_r^*\right) + \alpha_1 s_1^*\left(\beta_{1,1} a^* s_1^* - \beta_{1,2} c^* s_1^* + \beta_{1,3} t_r^*\right)}{\kappa_5 - \alpha_1 \beta_{1,1} s_1^* - \alpha_2 \beta_{2,1} s_2^* - \alpha_3 (1-m)\beta_{3,1} s_3^*}$$

$$+ \frac{\alpha_2 s_2^*\left(\beta_{2,1} a^* - \beta_{2,2} c^* - \beta_{2,3} t_r^*\right)}{\kappa_5 - \alpha_1 \beta_{1,1} s_1^* - \alpha_2 \beta_{2,1} s_2^* - \alpha_3 (1-m)\beta_{3,1} s_3^*} + \frac{\alpha_3 s_3^*\left(\beta_{3,1} a^* - \beta_{3,2} c^* + \beta_{3,3} t_r^*\right)}{\kappa_5 - \alpha_1 \beta_{1,1} s_1^* - \alpha_2 \beta_{2,1} s_2^* - \alpha_3 (1-m)\beta_{3,1} s_3^*},$$

$$l_c^* = \frac{(1-\rho)\left(\lambda_1 a^* + \lambda_2 c^* \lambda_3 t_r^*\right) + (1-\alpha_1) s_1^*\left(\beta_{1,3} a^* - \beta_{1,2} c^* - \beta_{1,3} t_r^*\right)}{\kappa_6 - \alpha_1 \beta_{1,1} s_1^* - \alpha_2 \beta_{2,1} s_2^* - \alpha_3 (1-m)\beta_{3,1} s_3^*}$$

$$+\frac{(1-\alpha_2)s_2^*\left(\beta_{2,3}a^*-\beta_{2,2}c^*-\beta_{2,3}t_r^*\right)}{\kappa_6-\alpha_1\beta_{1,1}s_1^*-\alpha_2\beta_{2,1}s_2^*-\alpha_3\left(1-m\right)\beta_{3,1}s_3^*}+\frac{(1-\alpha_3)s_3^*\left(\beta_{3,1}a^*-\beta_{3,2}c^*-\beta_{3,3}t_r^*\right)}{\kappa_6-\alpha_1\beta_{1,1}s_1^*-\alpha_2\beta_{2,1}s_2^*-\alpha_3\left(1-m\right)\beta_{3,1}s_3^*},$$

$$a^*=\frac{\delta_1 l_a^*}{\kappa_7},\quad c^*=\frac{\delta_2 l_c^*+\omega_2 t_r*+\sigma r*}{\kappa_8},\quad t_r^*=\frac{\omega_1 c^*}{\kappa_9},\quad r^*=\frac{\gamma_a a^*\gamma_c c^*+\gamma_{tr}t_r^*}{\kappa_{10}}.$$

Remember that $\kappa_1,\ \kappa_2,\ \kappa_3,\ \kappa_4,\ \kappa_5,\ \kappa_6,\ \kappa_7,\ \kappa_8,\ \kappa_9,\ \kappa_{10}$ are given in (2) It is obvious that $\mathcal{R}_e>1$ then there exists a unique endemic equilibrium [12, 22, 33].

Global stability of endemic equilibrium point $\mathcal{EE}$

In this subsection, we will demonstrate the global asymptotic stability of the endemic equilibrium point $\mathcal{EE}$ by developing a suitable Lyapunov function.

Theorem 2 *If $\mathcal{R}_e>1$, the endemic equilibrium point $\mathcal{EE}$ of model (3) is globally asymptotically stable.*

Proof If $\mathcal{R}_e>1$, the following Lyapunov function is defined as in [34–38]

$$\begin{aligned}\mathcal{L}=&(s_1-s_1^*)+(s_2-s_2^*)+(s_3-s_3^*)+(v-v^*)+(l_a-l_a^*)+(l_c-l_c^*)+(a-a^*)\\&+(c-c^*)+(t_r-t_r^*)+(r-r^*)-(s_1^*+s_2^*+s_3^*+v^*+l_a^*+l_c^*+a^*+c^*+t_r^*+r^*)\\&\times ln\left(\frac{s_1+s_2+s_3+v+l_a+l_c+a+c+t_r+r}{s_1^*+s_2^*+s_3^*+v^*+l_a^*+l_c^*+a^*+c^*+t_r^*+r^*}\right).\end{aligned}$$

We have:

$\Theta=s_1+s_2+s_3+v+l_a+l_c+a+c+t_r+r$ and $\Theta^*=s_1^*+s_2^*+s_3^*+v^*+l_a^*+l_c^*+a^*+c^*+t_r^*+r^*,.$

The Lyapunov function can then be reformulated as follows:

$$\mathcal{L}=\Theta-\Theta^*-\Theta^*ln\frac{\Theta}{\Theta^*}.$$

Since $s_1(t)>0$, $s_2(t)>0$, $s_3(t)>0$, $v(t)>0$, $l_a(t)>0$, $l_c(t)>0$, $a(t)>0, c(t)>0$, $t_r(t)>0$, $r(t)>0$, we can deduce the following:

$$\mathcal{L}=\Theta-\Theta^*-\Theta^*ln\frac{\Theta}{\Theta^*}>0. \tag{9}$$

Thus, the derivative of the Lyapunov function $\mathcal{L}$ is expressed as follows:

$$\frac{d\mathcal{L}}{dt}=\left(1-\frac{\Theta^*}{\Theta}\right)\frac{d\Theta}{dt}. \tag{10}$$

Note that according to system (3)

$$\frac{d\Theta}{dt}=\lambda-\mu_a a-\mu_c c-\mu_{tr}t_r-\mu\Theta. \tag{11}$$

As at the endemic equilibrium point $\frac{d\Theta}{dt}=0$ then one obtains

$$\lambda=\mu_a a^*+\mu_c c^*+\mu_{tr}t_r^*+\mu\Theta^*. \tag{12}$$

According to (9)- (12), and considering that

$$a - a^* \geq 0, \quad c - c^* \geq 0, \quad t_r - t_r^* \geq 0, \quad \Theta - \Theta^* \geq 0.$$

We have:

$$\frac{d\mathcal{L}}{dt} \leq 0.$$

From (3) and by using the fact that $\frac{d\mathcal{L}}{dt} = 0$ if and only if

$$s_1 = s_1^*,\ s_2 = s_2^*,\ s_3 = s_3^*,\ v = v^*,\ l_a = l_a^*,\ l_c = l_c^*,\ a = a^*,\ c = c^*, t_r = t_r^*,\ r = r^*,$$

then $\frac{d\mathcal{L}}{dt}$ converges in positive region Ω as t $\longrightarrow \infty$.
Due to the theorem of LaSalle's invariance principle [37], the endemic equilibrium point $\mathcal{EE}$ is considered globally asymptotically stable when $\mathcal{R}_e > 1$ within the region Ω [39, 40].

4 Sensitivity analysis

In the sensitivity analysis of the effective number of reproduction $\mathcal{R}_e$, the impact of each parameter on the endemic threshold is examined in detail. Sensitivity analysis, an essential technique for complex systems, is employed to assess the relative importance of model parameters. Thus, parameters with higher sensitivity index magnitudes exert greater influence than those with smaller magnitudes. The sign of the $\mathcal{R}_e$ sensitivity indices for the various parameters reveals their positive or negative effect. Here we calculate the sensitivity indices for each parameter, using the values listed in Table 2 above. The standard formula for calculating the sensitivity index of a parameter Υ for $\mathcal{R}_e$ is given by [35, 41, 42]:

$$\chi_{\Upsilon}^{\mathcal{R}_e} = \frac{\partial \mathcal{R}_e}{\partial \Upsilon} \times \frac{\Upsilon}{\mathcal{R}_e}.$$

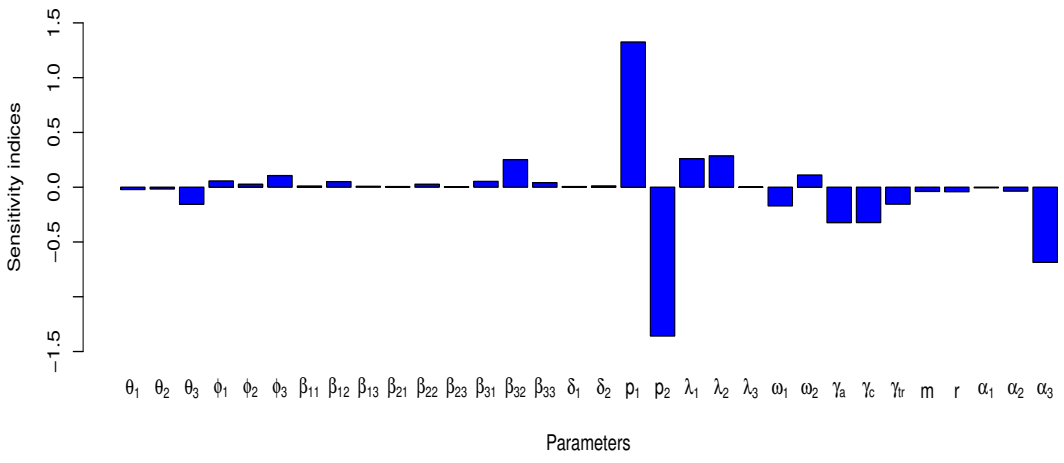

Figure 3. Sensitivity indices of model parameters

Considering the intricacy of the formula for $\mathcal{R}_e$, numerical differentiation has been employed. So, the numerical values of the sensitivity indices are provided in the Table 2. In Table 2, the estimated parameters were obtained using model (3) and the Grey Wolf Optimizer (GWO) algorithm on hepatitis B case data in Burkina Faso from 2016 to 2020 [16, 43]. Figure 3 clearly illustrates the influence of each parameter on the endemic threshold. Indeed, the positive sensitive parameters are $\chi_{\phi_1}^{\mathcal{R}_e} = 0.057246$, $\chi_{\phi_2}^{\mathcal{R}_e} = 0.027495$, $\chi_{\phi_3}^{\mathcal{R}_e} = 0.105957$, $\chi_{\beta_{11}}^{\mathcal{R}_e} = 0.010753$, $\chi_{\beta_{12}}^{\mathcal{R}_e} = 0.050853$, $\chi_{\beta_{13}}^{\mathcal{R}_e} = 0.008282$, $\chi_{\beta_{21}}^{\mathcal{R}_e} = 0.005765$, $\chi_{\beta_{22}}^{\mathcal{R}_e} = 0.027265$, $\chi_{\beta_{23}}^{\mathcal{R}_e} = 0.004441$, $\chi_{\beta_{31}}^{\mathcal{R}_e} = 0.053165$, $\chi_{\beta_{32}}^{\mathcal{R}_e} = 0.251426$, $\chi_{\beta_{33}}^{\mathcal{R}_e} = 0.040949$, $\chi_{\delta_1}^{\mathcal{R}_e} = 0.005823$, $\chi_{\delta_2}^{\mathcal{R}_e} = 0.011859$, $\chi_{\omega_2}^{\mathcal{R}_e} = 0.111042$, $\chi_{p_1}^{\mathcal{R}_e} = 1.325427$. The parameters that contribute to increase the value of $\mathcal{R}_e$ are therefore ϕ_1, ϕ_2, ϕ_3, β_{11}, β_{12}, β_{13}, β_{21}, β_{22}, β_{23}, β_{31}, β_{32}, β_{33}, δ_1, δ_2, p_1. It is noted that the most positively sensitive parameter is p_1, meaning that the lack of immunization among children aged 0 to 1 year strongly influences the increase in the value of $\mathcal{R}_e$, thus leading to the significant spread of the Hepatitis B virus in the population. The negative sensitive parameters are $\chi_{\theta_1}^{\mathcal{R}_e} = -0.021232$, $\chi_{\theta_2}^{\mathcal{R}_e} = -0.016215$, $\chi_{\theta_3}^{\mathcal{R}_e} = -0.155457$, $\chi_{\omega_1}^{\mathcal{R}_e} = -0.170449$, $\chi_{\gamma_a}^{\mathcal{R}_e} = -0.323745$, $chi_{\gamma_c}^{\mathcal{R}_e} = -0.322896$, $\chi_{\gamma_{tr}}^{\mathcal{R}_e} = -0.154835$, $\chi_m^{\mathcal{R}_e} = -0.038393$, $\chi_r^{\mathcal{R}_e} = -0.041173$, $\chi_{\alpha_1}^{\mathcal{R}_e} = -0.005260$, $\chi_{\alpha_2}^{\mathcal{R}_e} = -0.035991$, $\chi_{\alpha_3}^{\mathcal{R}_e} = -0.685630$, $\chi_{p_2}^{\mathcal{R}_e} = -1.358268$, the parameters responsible for decreasing the value of $\mathcal{R}_e$ are θ_1, θ_2, θ_3, ω_1, γ_a, γ_c, γ_{tr}, m, ρ, α_1, α_2, α_3, p_2. It is noted that the most negatively sensitive parameter is p_2, meaning that when there is a high proportion of the population growing up without being infected, this significantly contributes to the decrease in the value of $\mathcal{R}_e$, thus leading to the disappearance of the epidemic. We also note the importance of the parameters for the recovery $(\gamma_a, \gamma_c, \gamma_{tr})$ of acute infected, chronic patients, and chronic patients receiving treatment, highlighting the role of treatment in reducing, even if slightly, the value of $\mathcal{R}_e$.

5 Numerical results and discussions

Influence of the variation in vaccination coverage rate and the success rate of awareness campaigns on $\mathcal{R}_e$

According to Figure 4, one notes that:

- Without vaccination and a media awareness campaign, even with treatment, $\mathcal{R}_e = 2.0112$ the epidemic will continue to spread through the population.
- $\mathcal{R}_e$ decreases as the success rate of raising public awareness through the media increases.
- $\mathcal{R}_e$ decreases as the rate of vaccination coverage increases and starts to fall below 1, meaning that the epidemic will die out as the proportion of the population immunised by the vaccine increases. Hence the effectiveness of universal vaccination against hepatitis B as a means of combating the disease.

These last remarks provide information on the impact of systematic vaccination of children at birth and vaccination of the general population, as well as the effectiveness of campaigns by the media in combating the spread of HBV in Burkina Faso and elsewhere in highly endemic areas. The other figures below show the evolution of prevalence and infected populations.

Numerical simulation

We used the 4th-order Runge Kutta method for the numerical simulation. The details of this approximation and its implementation under Matlab. We set the initial condition to $N = 20818036$: the total population of Burkina Faso in 2020 [44]. The parameter values used are given in the Table 2.

Table 2. Parameters used in the model

Parameters	Values	References	Sensitivity index	values
N	20818036	[44]		
λ	0.0394	[44]		
λ_1	0.3	Estimated	$\chi_{\lambda_1}^{\mathcal{R}_e}$	0.259639
λ_2	0.07	Estimated	$\chi_{\lambda_2}^{\mathcal{R}_e}$	0.286503
λ_3	0.00144	Estimated	$\chi_{\lambda_3}^{\mathcal{R}_e}$	0.000960
ρ	0.1	Assumed	$\chi_{\rho}^{\mathcal{R}_0}$	−0.041173
m	0, 0.1, 0.2, 0.3, 0.4, 0.5	Assumed	$\chi_{m}^{\mathcal{R}_e}$	−0.038393
θ_1	0, 0.1, 0.3, 0.5, 0.7, 0.9	Assumed	$\chi_{\theta_1}^{\mathcal{R}_e}$	−0.021232
θ_2	0, 0.1, 0.3, 0.5, 0.7, 0.9	Assumed	$\chi_{\theta_1}^{\mathcal{R}_e}$	−0.016215
θ_3	0, 0.1, 0.3, 0.5, 0.7, 0.9	Assumed	$\chi_{\theta_1}^{\mathcal{R}_e}$	−0.155457
ϕ_1	0.03	Assumed	$\chi_{\phi_1}^{\mathcal{R}_e}$	0.057246
ϕ_2	0.04	Assumed	$\chi_{\phi_2}^{\mathcal{R}_e}$	0.027495
ϕ_3	0.05	Assumed	$\chi_{\phi_3}^{\mathcal{R}_e}$	0.105957
p_1	0.8	[16]	$\chi_{p_1}^{\mathcal{R}_e}$	1.325427
p_2	0.8	[16]	$\chi_{p_2}^{\mathcal{R}_e}$	−1.358268
$\beta_{1,1}$	0.1	Estimated	$\chi_{\beta_{1,1}}^{\mathcal{R}_e}$	0.010753
$\beta_{1,2}$	0.1	Estimated	$\chi_{\beta_{1,2}}^{\mathcal{R}_e}$	0.050853
$\beta_{1,3}$	0.1	Estimated	$\chi_{\beta_{1,3}}^{\mathcal{R}_e}$	0.008282
$\beta_{2,1}$	0.05	Estimated	$\chi_{\beta_{2,1}}^{\mathcal{R}_e}$	0.005765
$\beta_{2,2}$	0.05	Estimated	$\chi_{\beta_{2,2}}^{\mathcal{R}_e}$	0.027265
$\beta_{2,3}$	0.05	Estimated	$\chi_{\beta_{2,3}}^{\mathcal{R}_e}$	0.004441
$\beta_{3,1}$	0.1	Estimated	$\chi_{\beta_{3,1}}^{\mathcal{R}_e}$	0.053165
$\beta_{3,2}$	0.1	Estimated	$\chi_{\beta_{3,2}}^{\mathcal{R}_e}$	0.251426
$\beta_{3,3}$	0.1	Estimated	$\chi_{\beta_{3,3}}^{\mathcal{R}_e}$	0.040949
α_1	0.05	[4, 13]	$\chi_{\alpha_1}^{\mathcal{R}_e}$	−0.005260
α_2	0.7	[4, 13]	$\chi_{\alpha_2}^{\mathcal{R}_e}$	−0.035991
α_3	0.95	[4, 13]	$\chi_{\alpha_3}^{\mathcal{R}_e}$	−0.685630
δ_1	0.5	Estimated	$\chi_{\delta_1}^{\mathcal{R}_0}$	0.005823
δ_2	0.5	Estimated	$\chi_{\delta_2}^{\mathcal{R}_e}$	0.011859
γ_a	0.79	Estimated	$\chi_{\gamma_a}^{\mathcal{R}_e}$	−0.323745
γ_c	0.05	Estimated	$\chi_{\gamma_c}^{\mathcal{R}_e}$	−0.322896
γ_{tr}	0.2	Estimated	$\chi_{\gamma_{tr}}^{\mathcal{R}_e}$	−0.154835
ω_1	0.1	Estimated	$\chi_{\omega_1}^{\mathcal{R}_e}$	−0.170449
ω_2	0.4	Estimated	$\chi_{\omega_2}^{\mathcal{R}_e}$	0.111042
μ	0.009	[12, 44]		
μ_a	0.00461	[12, 45]		
μ_c	0.01	[12, 45]		
μ_{tr}	0.005	[16]		

So, each of subfigures (a), (b) and (c), present the evolution of the population of acutely infected, chronically infected, and all infected persons, respectively, and subfigure (d) shows the evolution

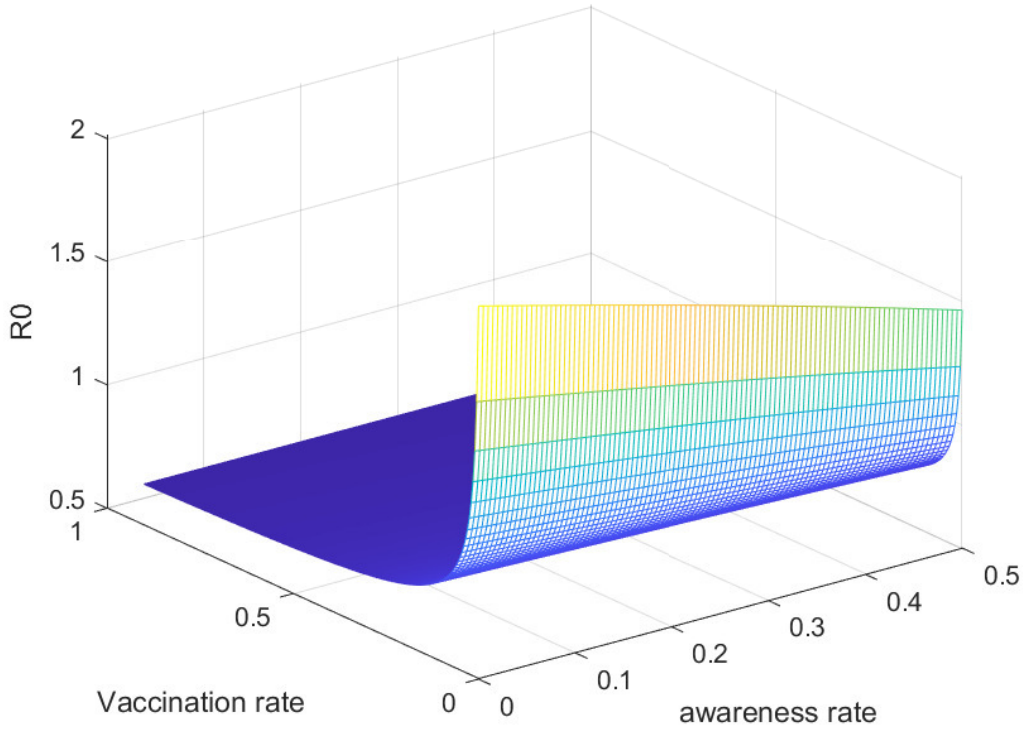

Figure 4. Evolution of the effective number of reproduction as a function of vaccination coverage and the effectiveness of media campaigns

of the prevalence, all depending on the vaccination coverage rate. In this Figure 5, sub-figure (a) illustrates the evolution of acute infections in the population in Burkina Faso as a function of vaccination coverage rate and in the absence of media awareness campaigns.
Indeed, without vaccination, the number of acute infections temporarily decreases (which could be explained by the exponential population growth in Burkina Faso) before increasing indefinitely. The same observation applies to a vaccination coverage rate of 10%, indicating that, up to 50 years, this 10% rate remains inefficient in reversing the current trend of hepatitis B. However, there is a continuous decrease in the number of acute infections starting from a vaccination coverage rate of 30%. This implies that with just a 30% vaccination coverage, the trend could begin to reverse in Burkina Faso starting from 10 years onwards.
This latter assertion is confirmed by sub-figures (b), (c), and (d). In sub-figure (d), a continuous decrease in prevalence is observed from a vaccination rate of 30%, with prevalence dropping below 5% in less than 10 years. Of course, for a vaccination coverage rate of 50%, one can observe over 50 years a prevalence dropping below 3%, and this decline is even more significant at vaccination coverage rates of 70% and 90%, where prevalence tends towards 1%. This indicates that we are moving towards the extinction of the epidemic.

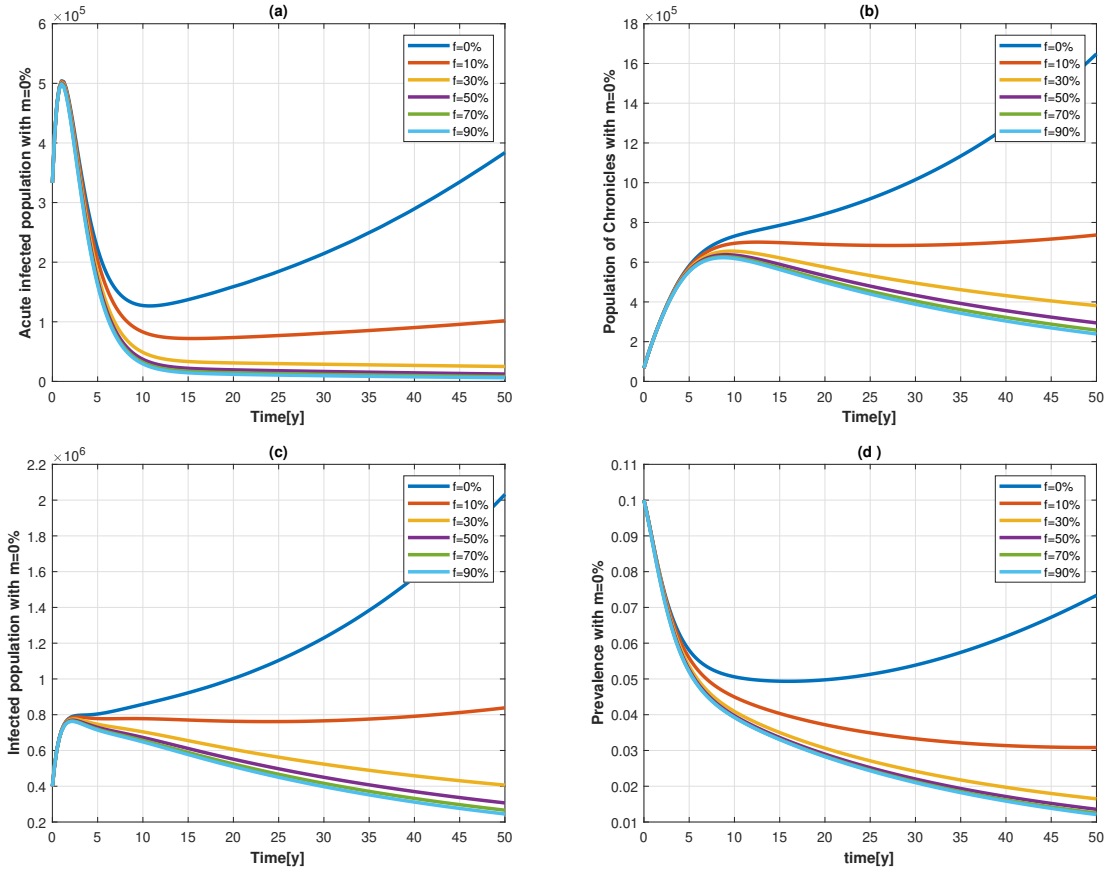

Figure 5. Subfigures (a)-(b)-(c)-(d) present the global dynamics of disease when there are no awareness campaigns i.e, $m = 0\%$

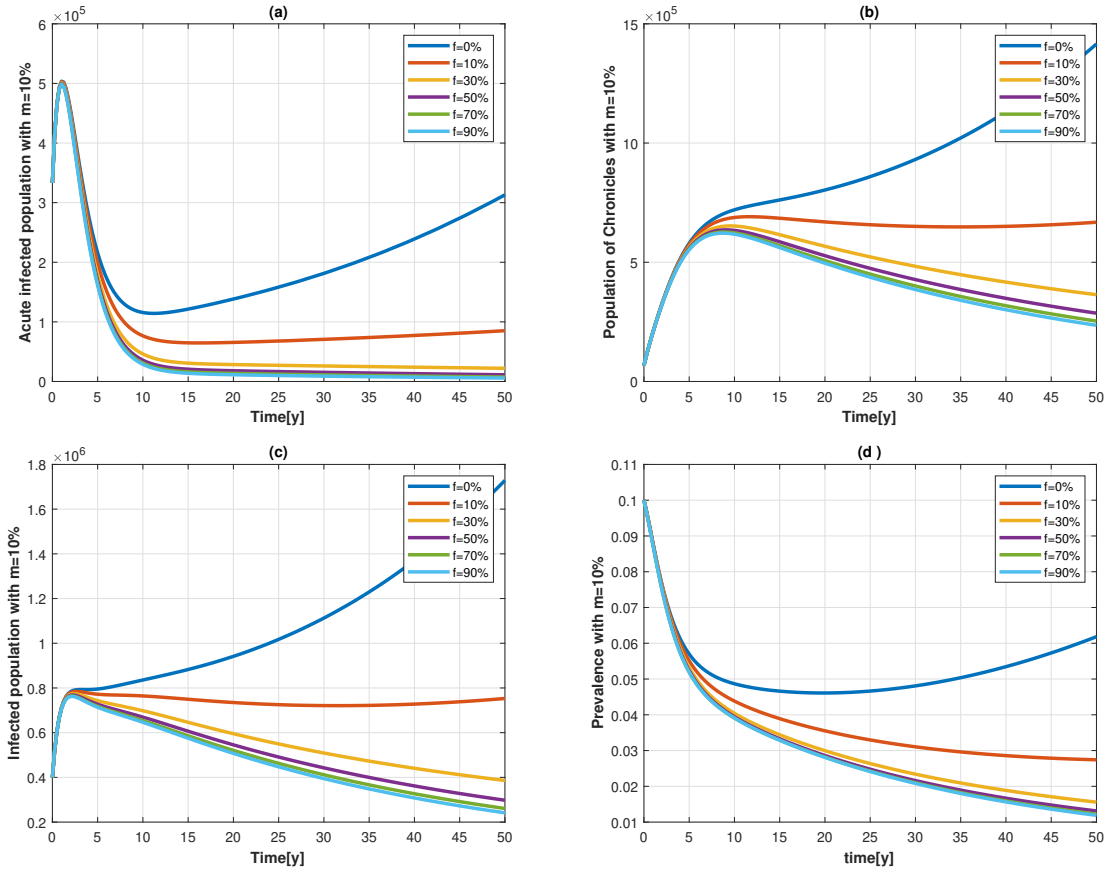

Figure 6. Subfigures (a)-(b)-(c)-(d) present the global dynamics of the disease for $m = 10\%$

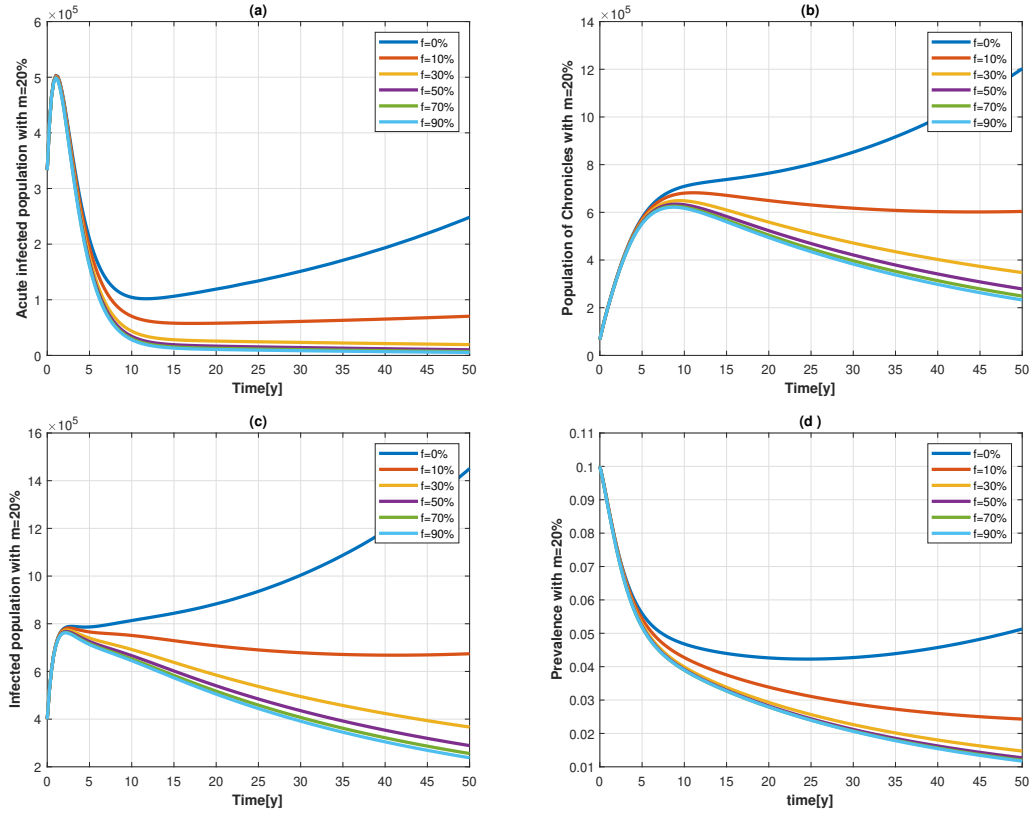

Figure 7. Subfigures (a)-(b)-(c)-(d) present the global dynamics of the disease for $m = 20\%$

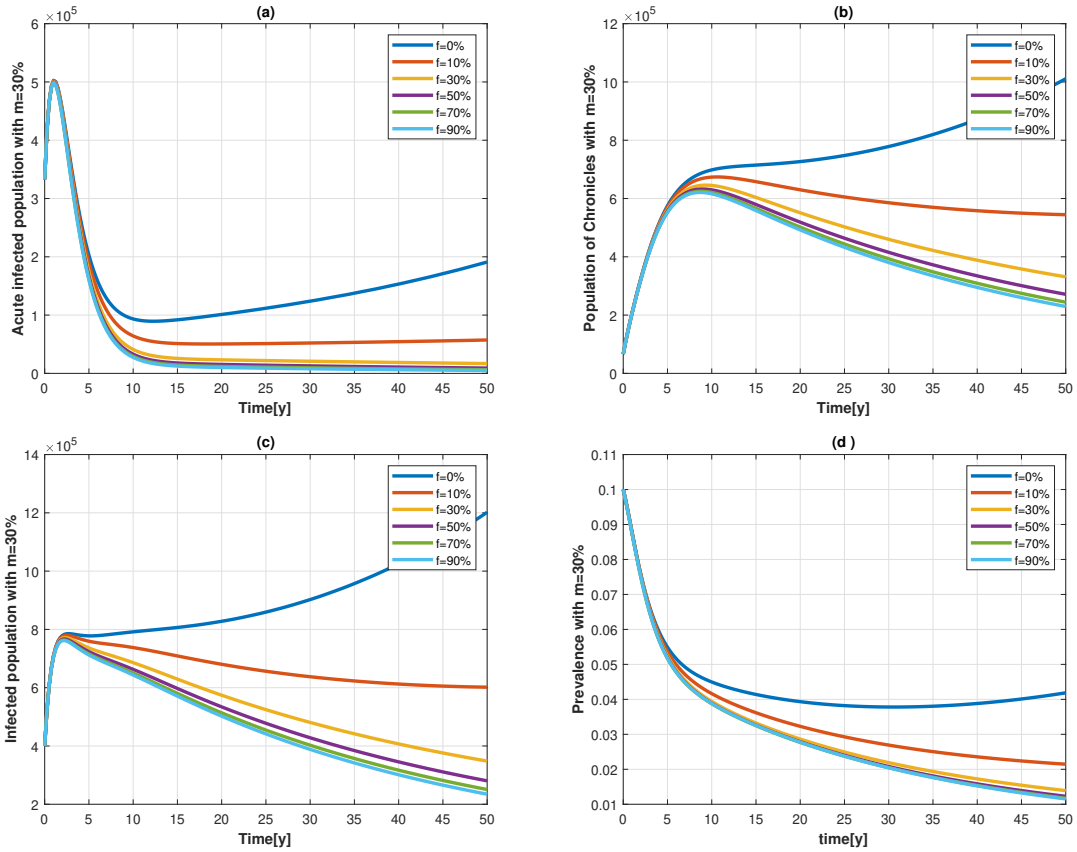

Figure 8. Subfigures (a)-(b)-(c)-(d) present the global dynamics of the disease for $m = 30\%$

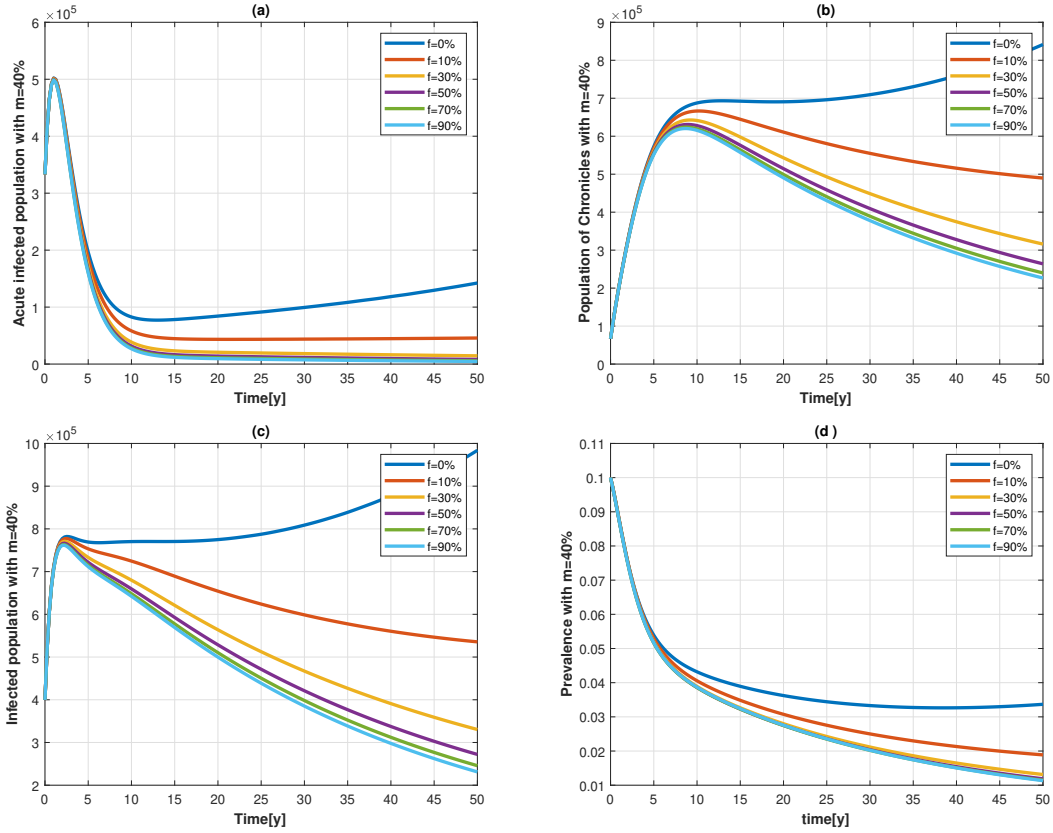

Figure 9. Subfigures (a)-(b)-(c)-(d) present the global dynamics of the disease for $m = 40\%$

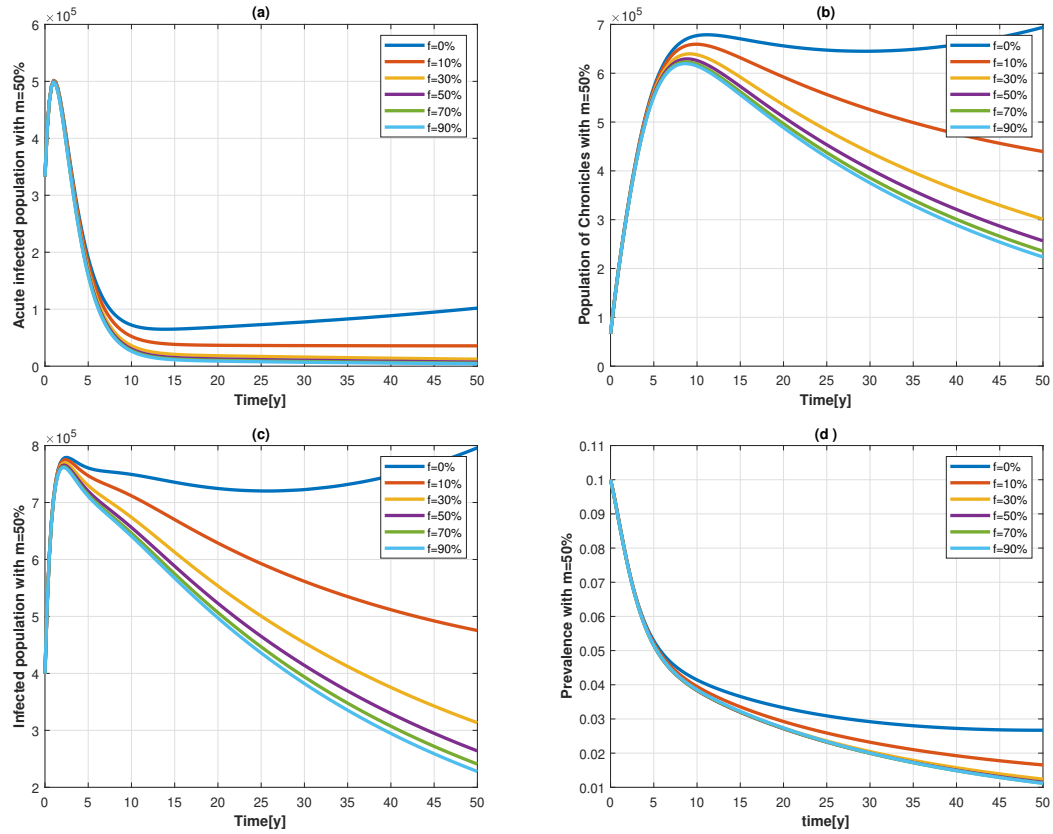

Figure 10. Subfigures (a)-(b)-(c)-(d) present the global dynamics of the disease for $m = 50\%$

In Figure 6, Figure 7, Figure 8, Figure 9 and Figure 10, it nicely almost the same dynamics in sub-figures (a)-(b)-(c)-(d) as in Figure 4 above. However, the impact of improving the success rate of media awareness campaigns is clearly visible. Indeed, from Figure 5 to Figure 9, as m increases, the number of acute infections, chronic infections, and overall infections increase more slowly for vaccination coverage rates of 0% and 10%. Additionally, the number of infections decreases more rapidly as m increases. Furthermore, prevalence decreases more rapidly with an increase in m. Thus, for $m = 50\%$, even without vaccination, the prevalence decreases significantly, and this decrease is much more substantial with the increase in vaccination coverage rate. Figure 6, Figure 7, Figure 8, Figure 9 and Figure 10 show the effectiveness of health education. As the saying goes, "prevention is better than cure." Indeed, according to the dynamics of the curves in these figures, with a vaccination coverage rate above 30%, the more educated and aware the population is about hepatitis B virus infection, its modes of transmission, preventive measures to avoid infection, and risky behaviors, the lower the prevalence becomes. Therefore, when 50% of the population is successfully educated through the media and 20% effectiveness for the 10% of treated chronic carriers, combined with a vaccination coverage rate above 30%, the prevalence of hepatitis B infection will be less than 2% from the time of the thirty years.

6 Conclusion

This article presents a deterministic model developed to analyze the impact of strategies aimed at controlling the hepatitis B epidemic in Burkina Faso through mathematical modeling. The model incorporates several key intervention measures: vaccination targeting three age groups among susceptible populations (infants aged 0–1 year, children aged 1–15 years, and individuals over 15 years old), treatment of chronic carriers, and awareness campaigns conducted through traditional media (radio, television) and social media platforms (Facebook, WhatsApp, Twitter (X), YouTube, etc.). A notable feature of this model is its consideration of viral reactivation in previously recovered individuals with immunodeficiency, an aspect often overlooked in most hepatitis B models.

Applied to the specific context of Burkina Faso, this model highlights critical epidemiological features of hepatitis B. Mathematical analysis reveals that the spread of the disease is determined by the effective reproduction number $\mathcal{R}_e$. When $\mathcal{R}_e$ is less than 1, the epidemic gradually dies out, allowing the model to stabilize at a hepatitis B-free equilibrium, which remains globally stable. Conversely, if $\mathcal{R}_e$ exceeds 1, the epidemic persists and evolves toward a globally stable endemic state.

A sensitivity analysis of $\mathcal{R}_e$ identified the most critical parameters influencing its increase or reduction. These results go along with the recommendations of the World Health Organization (WHO) and Burkina Faso's Ministry of Health, which emphasize universal newborn vaccination as the cornerstone of hepatitis B control strategies[2, 13]. Although the impact of treating chronic carriers is relatively limited, it contributes to reducing virus transmission, as treated and immunized carriers can no longer spread the disease.

Numerical simulations performed with this model confirm that increasing vaccination coverage and enhancing the effectiveness of awareness campaigns are significant factors in controlling the epidemic. For instance, achieving a 30% vaccination coverage, combined with a 50% success rate for awareness campaigns and 20% efficacy in treating 10% of chronic carriers, could reduce hepatitis B prevalence to 2% within 30 years.

These findings indicate that a combination of universal vaccination, targeted education on transmission modes and preventive measures, and treatment of chronic carriers make up a coherent and effective strategy that could potentially lead to the eradication of hepatitis B. This approach mirrors successes observed in countries like Taiwan, which transitioned from endemic situations

to significantly reduced disease prevalence through similar policies [46].
This study strongly supports the efforts of the Burkinabe Society of Hepatology and Digestive Endoscopy in advocating for increased accessibility to vaccination and treatment of the population. It also emphasizes the need to strengthen awareness campaigns leveraging both traditional and social networks media to maximize access to preventive messaging.
We recommend urgent action from the Burkinabe government to promote large-scale screening, vaccination, and treatment programs. Special attention should be paid to developing permanent awareness campaigns tailored to local contexts and improving access to these interventions, particularly in rural areas. These actions could significantly transform the fight against hepatitis B and lead Burkina Faso to eradicate this disease, joining then the rank of low-endemicity countries that have successfully achieved this goal through similar strategies.

Declarations

Use of AI tools

The authors declare that they have not used Artificial Intelligence (AI) tools in the creation of this article.

Data availability statement

All authors declare that data availability is not applicable to this article.

Ethical approval (optional)

The authors declare that this research complies with ethical standards. This research does not involve human participants or animals.

Consent for publication

Not applicable

Conflicts of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Funding

Not applicable

Author's contributions

A.K.: Conceptualization, Methodology, Software, Formal Analysis, Data Curation, Investigation, Writing - Original Draft, Writing - Review & Editing, Validation. W.O.S.: Conceptualization, Methodology, Investigation, Software, Data Curation, Writing - Review & Editing, Visualization, Supervision, Project Administration, Validation. P.O.F.O.: Writing - Review & Editing, Visualization, Suggestion, Validation. F.A.: Writing - Review & Editing, Visualization, Suggestion, Validation. H.A.: Writing - Review & Editing, Visualization, Suggestion, Validation. K.S.S.: Conceptualization, Investigation, Suggestion, Validation. A.K.S.: Conceptualization, Investigation, Suggestion, Validation. All authors discussed the results and contributed to the final manuscript.

Acknowledgements

The authors express their deepest thanks to the editor and anonymous referees for their comments and suggestions on the article.

References

[1] World Health Organization, Progress report on HIV, viral hepatitis and sexually transmitted infections 2019: accountability for the global health sector strategies, 2016-2021. *World Health Organization*, (2019).

[2] Ministère de la Santé. Normes et Protocoles de Prise en Charge des Hépatites Virales au Burkina Faso, (2019). *Ministère de la santé du Burkina Faso*, https://scge-cm.com/download.

[3] Masseta, M. *Incidental discovery of HBs antigen at the Y.O. University Hospital: socio-demographic, clinical and paraclinical aspects*, PhD Thesis, Joseph Ki-Zerbo University, (2018).

[4] Banach, M. (2022). Global, regional, and national burden of hepatitis B, 1990-2019: a systematic analysis for the Global Burden of Disease Study 2019. GBD 2019 Hepatitis B Collaborator. *The Lancet Gastroenterology and Hepatology*, 7(9), 796 -829, (2022). [CrossRef]

[5] Ott, J.J., Stevens, G.A., Groeger, J. and Wiersma, S.T. Global epidemiology of hepatitis B virus infection: new estimates of age-specific HBsAg seroprevalence and endemicity. *Vaccine*, 30(12), 2212-2219, (2012). [CrossRef]

[6] Ministry of Health, Burkina. *Hepatitis B, cirrhosis and primary liver cancer kill nearly 5,000 people a year by Editorial Board, Ouagadougou*, Fasozine, (2017). https://fasozine.com.

[7] Meda, N., Tuaillon, E., Kania, D., Tiendrebeogo, A., Pisoni, A., Zida, S., ... and Dujols, P. Hepatitis B and C virus seroprevalence, Burkina Faso: a cross-sectional study. *Bulletin of the World Health Organization*, 96(11), 750, (2018). [CrossRef]

[8] Zouré, A.A., Nadembega, C., Ouattara, A.K., Traoré, L., Tiemtoré, A., Sawadogo, O., ... and Simpore, J. State of the HIV, Hepatitis B and C Virus Pandemic from 2003 to 2022 in Burkina Faso: Evolution of Prevalence Trends and Strategic Recommendations to Achieve the WHO's Goal for Their Eradication by 2030. *Advances in Infectious Diseases* Scientific Research Publishing, 14(3) 628–643 (2024). [CrossRef]

[9] Tao, I., Compaoré, T.R., Diarra, B., Djigma, F., Zohoncon, T.M., Assih, M., ... and Simpore, J. Seroepidemiology of hepatitis B and C viruses in the general population of Burkina Faso. *Hepatitis Research and Treatment*, 2014(1), 781843, (2014). [CrossRef]

[10] Guingané, A.N., Kaboré, R., Shimakawa, Y., Somé, E.N., Kania, D., Pisoni, A., ... and Tuaillon, E. Screening for Hepatitis B in partners and children of women positive for surface antigen, Burkina Faso. *Bulletin of the World Health Organization*, 100(4), 256, (2022). [CrossRef]

[11] Somé, E.N., Kaboré, J., Kaboré, C., Somda, S., Somé, R., Somda, P. and Sombié, R. Burkina Faso and the global goal of eliminating Hepatitis B virus By 2030. Advances in Public Health, Community and Tropical Medicine, 2022(02), 1-5, (2022). [CrossRef]

[12] Kiemtoré, A., Sawadogo, W.O., Aquel, F., Alaa, H. and Somda, K.S. Mathematical modelling and Numerical Simulation of Hepatitis B Viral Infection: the case of Burkina Faso. *European Journal of Pure and Applied Mathematics*, 17(1), 59–92 (2024). [CrossRef]

[13] Feld, J., Janssen, H.L., Abbas, Z., Elewaut, A., Ferenci, P., Isakov, V., ... and LeMair, A. World gastroenterology organisation global guideline hepatitis B: September 2015. *Journal of Clinical Gastroenterology*, 50(9), 691–703, (2016). [CrossRef]

[14] Fall, A., Iggidr, A., Sallet, G. and Tewa, J.J. Epidemiological models and Lyapunov functions.

Mathematical Modelling of Natural Phenomena, 2(1), 62-83, (2007). [CrossRef]

[15] Bonzi, B., Fall, A.A., Iggidr, A. and Sallet, G. Stability of differential susceptibility and infectivity epidemic models. *Journal of Mathematical Biology*, 62(1), 39-64, (2011). [CrossRef]

[16] Kiemtore, A., Sawadogo, W.O., Zangré, I., Ouedraogo, P.O.F. and Mouaouia, I. Estimation of parameters for the mathematical model of the spread of hepatitis B in Burkina Faso using grey wolf optimizer. *International Journal of Analysis and Applications*, 22, 48-48, (2024). [CrossRef]

[17] Anderson, R.M. and May, R.M. *Infectious Diseases of Humans: Dynamics and Control.* Oxford University Press: New York, (1991).

[18] Edmunds, W.J., Medley, G.F., Nokes, D.J., O'callaghan, C.J., Whittle, H.C. and Hall, A.J. Epidemiological patterns of hepatitis B virus (HBV) in highly endemic areasr. *Epidemiology & Infection*, 117(2), 313-325, (1996). [CrossRef]

[19] Mahardika, D., Tjahjana, R. and Sunarsih, S. Dynamic modelling of Hepatitis B and use of optimal control to reduce the infected population and minimizing the cost of vaccination and treatment. In Proceedings, *AIP Conference Proceedings*, (Vol. 2331, No. 1), pp. 020013, Jakarta, Indonesia, (2021, April). [CrossRef]

[20] Sanz-Bueno, J., Vanaclocha, F. García-Doval, I., Torrado, R., Carretero, G., Daudén, E. et al. Risk of reactivation of hepatitis B virus infection in psoriasis patients treated with biologics: a retrospective analysis of 20 cases from the BIOBADADERM database. *Actas Dermo-Sifiliográficas (English Edition)*, 106(6), 477-482, (2015). [CrossRef]

[21] Aqel, F., Alaa, H. and Alaa, N.E. Mathematical model of Covid-19 transmissibility during the vaccination period. *Eurasian Journal of Mathematical and Computer Applications*, 11(1), 4-28, (2023). [CrossRef]

[22] Seidu, B., Bornaa, C.S. and Makinde, O.D. An Ebola model with hyper-susceptibility. *Chaos, Solitons & Fractals*, 138, 109938, (2020). [CrossRef]

[23] Van den Driessche, P. and Watmough, J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, 180(1-2), 29-48, (2002). [CrossRef]

[24] Peter, O.J., Abidemi, A., Fatmawati, F., Ojo, M.M. and Oguntolu, F.A. Optimizing tuberculosis control: a comprehensive simulation of integrated interventions using a mathematical model. *Mathematical Modelling and Numerical Simulation with Applications*, 4(3), 238-255, (2024). [CrossRef]

[25] Mamo, T.T. and Mekonnen, T.T. An age structure mathematical model analysis on the dynamics of chronic and hyper toxic forms of hepatitis B virus by model with vaccination intervention control strategy in Ethiopia. *International Journal of Sciences Basic and Applied Research (IJSBAR)*, 50(1), 106-136, (2020).

[26] Latifah, M.L. and Helma. Mathematical model of vertical transmission of hepatitis B with the effect of treatment and vaccination. *Rangkiang Mathematics Journal*, 2(2), 20-29, (2023). [CrossRef]

[27] Delamater, P.L., Street, E.J., Leslie, T.F., Yang, Y.T. and Jacobsen, K.H. Complexity of the basic reproduction number (R_0). *Emerging Infectious Diseases*, 25(1), 1-4, (2019). [CrossRef]

[28] Inglesby, T.V. Public health measures and the reproduction number of SARS-CoV-2. *Jama*, 323(21), 2186-2187, (2020). [CrossRef]

[29] Castillo-Chavez, C., Blower, S., Van den Driessche, P., Kirschner, D. and Yakubu, A.A. *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: Models, Methods, and Theory*

(Vol. 126). Springer: New York, (2002).

[30] Castillo-Chavez, C., Feng, Z. and Huang W. On the computation of R_0 and its role on global stability. *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction* (pp. 229-250). New York: Springer, (2002).

[31] Khan, A., Zarin, R., Ahmed, I., Yusuf, A. and Humphries, U.W. Numerical and theoretical analysis of Rabies model under the harmonic mean type incidence rate. *Results in Physics,* 29, 104652, (2021). [CrossRef]

[32] Castillo-Chavez, C. and Feng, Z. Global stability of an age-structure model for TB and its applications to optimal vaccination strategies. *Mathematical Biosciences,* 151(2), 135-154, (1998). [CrossRef]

[33] Dai, C., Fan, A. and Wang, K. Transmission dynamics and the control of hepatitis B in China: a population dynamics view. *Journal of Applied Analysis and Computation*, 6(1), 76-93, (2016). [CrossRef]

[34] McCluskey, C.C. Global stability of an SIR epidemic model with delay and general nonlinear incidence. *Mathematical Biosciences and Engineering*, 7(4), 837-850, (2010). [CrossRef]

[35] Koutou, O. and Sangaré, B. Mathematical analysis of the impact of the media coverage in mitigating the outbreak of COVID-19. *Mathematics and Computers in Simulation*, 205, 600-618, (2023). [CrossRef]

[36] Duan, X., Yuan, S. and Li, X. Global stability of an SVIR model with age of vaccination. *Applied Mathematics and Computation*, 226, 528-540, (2014). [CrossRef]

[37] Lasalle, J.P. *The Stability of Dynamical Systems.* SIAM: Philadelphia, (1976).

[38] Din, A., Li, Y. and Shah, M.A. The complex dynamics of hepatitis B infected individuals with optimal control. *Journal of Systems Science and Complexity*, 34(4), 1301-1323, (2021). [CrossRef]

[39] Bounkaicha, C., Allali, K., Tabit, Y. and Danane, J. Global dynamic of spatio-temporal fractional order SEIR model. *Mathematical Modeling and Computing*, 10(2), 299-310 (2023). [CrossRef]

[40] Lakshmikantham, V., Leela, S. and Martynyuk, A.A. *Stability Analysis of Nonlinear Systems.* Marcel Dekker: New York, (1989).

[41] Asamoah, J.K.K., Jin, Z. and Sun, G.Q. Non-seasonal and seasonal relapse model for Q fever disease with comprehensive cost-effectiveness analysis. *Results in Physics,* 22, 103889, (2021). [CrossRef]

[42] Norton, J. An introduction to sensitivity assessment of simulation models. *Environmental Modelling & Software,* 69, 166-174, (2015). [CrossRef]

[43] Mirjalili, S., Mirjalili, S.M. and Lewis, A. Grey wolf optimizer. *Advances in Engineering Software*, 69, 46-61, (2014). [CrossRef]

[44] INSD, General population and housing census, Burkina Faso, 2019. Fifth General Census of Population and Housing of Burkina Faso, Synthesis of final results, (2022). https://insd.bf/

[45] Edmunds, W.J., Medley, G.F. and Nokes, D.J. The transmission dynamics and control of hepatitis B virus in The Gambia. *Statistics in Medicine,* 15(20), 2215-2233, (1996). [CrossRef]

[46] Lu, F.T. and Ni, Y.H. Elimination of mother-to-infant transmission of hepatitis B virus: 35 years of experience. *Pediatric Gastroenterology, Hepatology & Nutrition,* 23(4), 311-318, (2020). [CrossRef]

Mathematical Modelling and Numerical Simulation with Applications (MMNSA)
(https://dergipark.org.tr/en/pub/mmnsa)

How to cite this article: Kiemtoré, A., Sawadogo, W.O., Ouédraogo, P.O.F., Aqel, F., Alaa, H., Somda, K.S. and Sermé, A.K. (2024). Mathematical modelling of the impact of vaccination, treatment and media awareness on the hepatitis B epidemic in Burkina Faso. *Mathematical Modelling and Numerical Simulation with Applications*, 4(5), 139-164. https://doi.org/10.53391/mmnsa.1528691