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Mathematical modelling and Numerical Simulation of Hepatitis B Viral Infection: the case of Burkina Faso

Adama Kiemtoré[1], Wendabbo Olivier Sawadogo[2,*], Fatima Aqel[3], Hamza Alaa[4], Kounpielime Sosthène Somda[5]

[1] *LANIBIO Laboratory, University Joseph Ki-Zerbo, Ouagadougou, Burkina Faso*
[2] *LARED Laboratory, University of Ouahigouya, Burkina Faso*
[3] *Computer, Networks, Mobility and Modeling laboratory (IR2M), Faculty of Sciences and Technics, Hassan First University, Settat 26000, Morocco*
[4] *LAMAI Laboratory, Faculty of Sciences and Technology, University of Cadi Ayyad, Abdelkrim, Khattabi, Marrakech 40000, Morocco*
[5] *Department of Biochemistry and Microbiology, Pietro Annigonni Biomolecular Research Center, Ouagadougou, Burkina Faso*

Abstract. Hepatitis B is a viral infection that can cause inflammation of the liver and lead to severe liver damage and even death. The study of hepatitis in Burkina Faso is crucial for several reasons. Indeed, understanding the epidemiology of hepatitis in Burkina Faso can help develop effective prevention and control strategies. Its study can also contribute to a better understanding of the global burden of the disease and the development of effective interventions in other parts of the world. To this aim, a new differential susceptibility and infectivity mathematical model of Hepatitis B transmission was developed in order to simulate the potential spread of the Hepatitis B virus in the population of Burkina Faso. Once the mathematical model is presented, the existence and uniqueness of non-negative solutions are proved. The model has a globally asymptotically stable disease-free equilibrium when the basic reproduction number $\mathcal{R}_0 < 1$ and a globally asymptotically stable endemic equilibrium when $\mathcal{R}_0 > 1$. The global asymptotic stability of the disease-free equilibrium has been studied using the Castillo Chavez method. The Lyapunov function and the LaSalle invariance principle are used to prove the global asymptotic stability of the endemic equilibrium. To simulate the proposed model, a Matlab numerical code has been developed. Numerical simulations are performed using data of Burkina Faso. The obtained numerical results confirm analytical results as well as the evolution of hepatitis B in Burkina Faso.

2020 Mathematics Subject Classifications: 35A01, 92B05, 34C11, 47H10, 00A71.

Key Words and Phrases: Hepatitis B, susceptibility, infectivity, stability, simulation

*Corresponding author.
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Email addresses: kiemtoreadama@gmail.com (Adama Kiemtoré), wenddabo81@gmail.com (Wendabbo Olivier Sawadogo),fatima.aqel@uhp.ac.ma (Fatima Aqel), hamza.alaa@ced.uca.ma (Hamza Alaa), sosthenesomda@gmail.com (Kounpielime Sosthène Somda)

1. Introduction

Hepatitis B virus (HBV) is a global health threat, and its elimination by 2030 is a World Health Organization priority. Hepatitis B is not easily curable. Most people who are infected will not have any symptoms of infection [11], [12], [10]. They are called asymptomatic carriers and play a very important role in the transmission of this disease. These are people who are affected by the virus but do not show any clinical signs. [33] Serological testing is therefore essential. Thus, many people infected with HBV may not know it and may transmit it to other individuals [13], [9], [33], [41]. According to the World Health Organization (WHO), it has been estimated that 2.5 billion people are infected or have been infected with HBV, representing 30% million of the global population. Among them, there are 257 million people who suffer from chronic hepatitis B (global report 2017), of which 60 million are in Africa with 136,000 deaths in 2015 [33]. The HBV infection is the second known human carcinogenic after tobacco. The hepatitis B virus (HBV) primarily targets the liver, and infection with this virus can cause damage to the liver through either acute or chronic infection.

The major damage from HBV is due to the long-term consequences of the chronic infection, mainly cirrhosis and hepatocellular carcinoma. According to the World Health Organization (WHO), about 25% of people with chronic hepatitis B infection develop cirrhosis or liver cancer, which can lead to a premature death if left untreated [19, 34]. Indeed, the risk of becoming chronic, and of complications such as liver cirrhosis and/or hepatocellular carcinoma (liver cancer), makes it a serious pathology since it causes more than 5000 deaths per year in Burkina Faso [32]. This latter is classified as one of the high-endemicity countries (seroprevalence > 8 %) [34]. Furthermore, in 2011's Demographic Health Survey, HBV seroprevalence was estimated to be 9.1% [31]. Also, a study conducted in Ouagadougou ((the capital city of Burkina Faso)) revealed a prevalence of 14.5 % in the general population [39]. Thus, nearly 2 million people in Burkina Faso are infected with HBV. Nevertheless, the actual number of infected people in Burkina Faso may be higher, as many people with chronic HBV infection may not know that they are infected due to the absence of symptoms or awareness of the disease. The distribution of the prevalence of HBV infection in the regions of Burkina faso is shown in Figure 1.

The main mode of hepatitis B virus (HBV) transmission in highly endemic regions such as Burkina Faso is through mother-to-child transmission during childbirth, or through close contact with infected family members in early childhood. This mode is known as vertical transmission of HBV. In the endemic regions, HBV is often transmitted from infected mothers to their babies during delivery, more particularly when the mother is tested positive for hepatitis B surface antigen (HBsAg) and hepatitis B e antigen (HBeAg) [33]. Transmission of HBV during perinatal period or the first years of childhood has a very high probability of being transmitted to a chronic disease. A pregnant woman with HBV can infect her child at delivery or during the first months of the infant's life, either through breastfeeding or through extremely close contact (perinatal transmission). The

risk of transmission in the mother's womb is related to the maternal viremia. The risk of infection with HBV may also be higher for individuals who engage in high-risk behaviours, such as injecting drugs, having unprotected sex with infected partners, or getting tattoos or piercings with unsterilised equipments. In addition, transmission between individuals living in the same household could be linked to the sharing of toiletries that cause small wounds, as well as razors, toothbrushes and nail clippers. Promiscuity and unsanitary conditions may be risk factors for the transmission of HBV, although they are not the only ones.

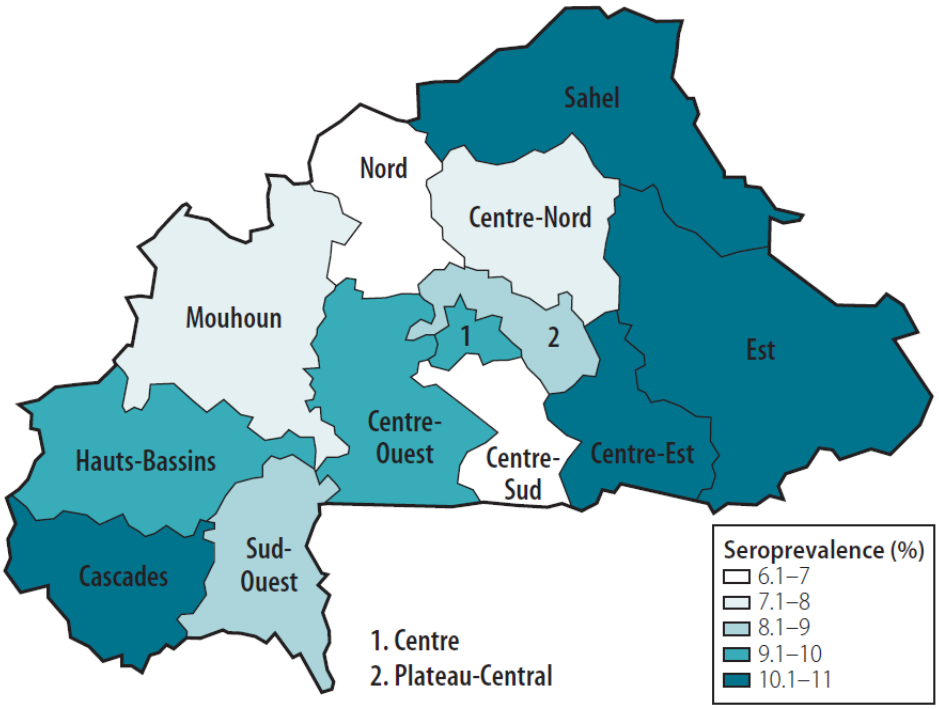

Figure 1: Burkina Faso distribution of HBV prevalence [31]

The hepatitis B virus is an enveloped virus belonging to the Hepadnaviridae family and the Orthohepadnavirus genus. It is a very contagious virus. The reservoir of HBV is strictly human, meaning that the virus only infects humans and has no other natural host. It can survive in the outdoor environment for at least 7 days and remain infectious on surfaces, such as needles or other medical equipments, for even longer [7]. Hepatitis B is considered as a DNA virus (deoxyribonuclease acid) with 3 antigenic systems, HBsAg (hepatitis B surface antigen) for the outer capsule, HBcAg (hepatitis B core antigen) and HBeAg (hepatitis B "e" antigen) for the internal nucleocapsid. HBeAg is associated with high viral replication, but due to certain mutations, HBeAg is not detected in the serum although viral replication is presented. In clinical practices in Burkina Faso, HBeAg negative patients represent about 90%. Thus, the best marker of viral multiplication is the

detection of viral DNA (deoxyribonuclease acid) in the serum [33]. The high transmission of hepatitis B virus is explained by:

- the length of the incubation period (6 weeks to 4 months) [28],
- the very high infectious blood titer (0.0001 mL of plasma can transmit HBV) [16],
- the large number of asymptomatic carriers [16],
- HBV is 50 to 100 times more infectious than HIV [16],
- HBV has the ability to retain its infectivity for more than 7 days at room temperature [16].

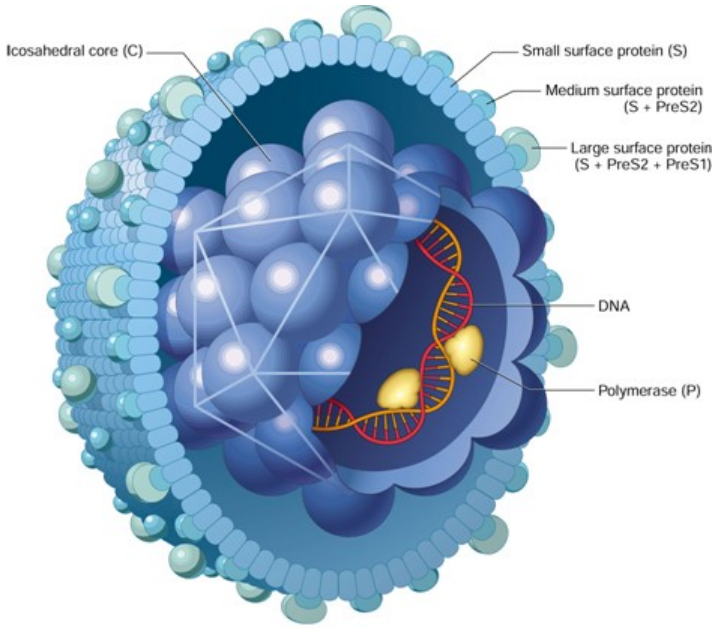

Figure 2: structure of an HBV virus particle (James A. Prekins, medical and scientific illustrations, 2002) [29]

In this context, Therefore, which contribution could mathematics make to improve the situation in Burkina Faso? The answer to this question is to propose a mathematical model to better understand the dynamics of HBV transmission and predict the infection's evolution.

The remainder of this paper is organized as follows: section two is dedicated to the presentation of the proposed transmission network model and to the calculus of the reproduction rate. Section three is devoted to the proof of global existence and uniqueness of solutions for the proposed system, as well as to the calculus of the reproduction rate. The global stability analysis of the model problem for the disease free equilibrium point is presented in the fourth section. In the fifth section, the global stability analysis for

the endemic equilibrium point is proved. In the sixth section, a numerical algorithm is developed to numerically simulate the proposed model for different involved parameters. The obtained numerical results are presented in the sixth section.

2. The proposed model of HBV transmission in Burkina Faso

A SEIR (Susceptible S, Exposed E, Infected I, Recovered R) model was formulated which takes into account the epidemiology of HBV infection. Due to the fact that vertical transmission is the most frequent mode of transmission in Burkina Faso, a model with vertical transmission was developed. The total population births is denoted by Λ. The introduction of vertical transmission decreases the number of births by an amount of $\lambda_I I + \lambda_C C$ which no longer become susceptible, as babies from these births are acutely and chronically infected by vertical transmission. Thus, both $\lambda_I I$ and $\lambda_C C$ appear in acute E_I and chronic E_C compartment of acutely infected exposed [16]. HBV is transmitted vertically by infected individuals (in the acute phase) and chronic carriers of the virus. The description of the variables is given in Table 1 and that of the parameters in Table 2.

2.1. Assumptions

Throughout this paper, it is assumed that :

(H1) Susceptibles individuals were subdivided into three age classes $S_i, i = 1, 2, 3$.

(H2) The population in each compartment is likely to fall into two groups, with a proportion $\alpha_i, i = 1, 2, 3$ of this population developing an acute infection i.e. the acute exposed E_I who will evolve to an acute infected state I and a proportion $(1 - \alpha_i), i = 1, 2, 3$ of this population that will become chronically infected (the chronic exposed E_C that will evolve to a chronic infected state C).

(H3) The high prevalence ($> 8\%$) in Burkina Faso is mainly due to the vertical transmission, hence the choice of the vertical transmission model [16, 31].

(H4) The general recruitment of the population is $\Lambda - \lambda_I I - \lambda_C C$.

(H5) A Part of the births from the acute and chronic HBV infections participate with an amount $\lambda_I + \lambda_C$, which will move to the exposed compartments of both acute and chronic infections.

(H6) Vertical transmission occurs in both acute and chronically infected individuals.

(H7) The adequate contact coefficient for a susceptible person to be infected with HBV, depends on the number of infected individuals [2, 16], i.e. the E_I, E_C, I, C. One has the following

(i) $\beta_1 = \beta_{1,1} E_I + \beta_{1,2} E_C + \beta_{1,3} I + \beta_{1,4} C$ the adequate coefficient of contact so that a susceptible S_1 becomes contaminated by the HBV.

(ii) $\beta_2 = \beta_{2,1}E_I + \beta_{2,2}E_C + \beta_{2,3}I + \beta_{2,4}C$ the appropriate contact coefficient for a susceptible S_2 to become contaminated with HBV.

(iii) $\beta_3 = \beta_{3,1}E_I + \beta_{3,2}E_C + \beta_{3,3}I + \beta_{3,4}C$ the appropriate contact coefficient for a susceptible S_3 to become contaminated with HBV.

(H8) Acute infection includes fulminant hepatitis, which is very rare [28].

(H9) All individuals who do not die from HVB, i.e. individuals in compartments $S_1, S_2, S_3, E_I, E_C, R$ have the same mortality rate μ.

(H10) Λ, λ_I, λ_C, μ, μ_1, μ_2, μ_3, μ_4, δ_1, δ_2, γ_i, γ_c are assumed to be strictly positive.

(H11) The prognosis for the evolution of HBV infection to chronicity in Burkina Faso is generally high, as it is in many other sub-Saharan African countries. The prognosis depends on the age at which the individual was infected. Thus, susceptible infants aged 0-1 year become chronic with a probability of 0.9; children aged 1-5 years with a probability of 0.3; and those aged over 5 years with a probability of 0.05 [17].

(H12) The total population N is not constant.

2.2. The interactions

In the following sub-section, we start by presenting the different parameters, and the different variables involved in our mathematical model. Next, detailed explanations of the different compartments that compose the proposed model as well as their interactions, are given. Table 1 contains the different variables with their meanings, while Table 2 contains the different parameters with their significance.

Table 1: Variables used in the model

Variables	description
$S_1(t)$	babies aged 0-1 year susceptible to disease
$S_2(t)$	infants 1 to 5 years old susceptible to disease
$S_3(t)$	children over 5 years of age, adolescents and adults at risk of disease
$E_I(t)$	Exposed infected individuals who will progress to an acute infection state
$E_C(t)$	Exposed chronic individuals who will progress to a chronically infected state
$I(t)$	infected individuals acute
$C(t)$	chronic carriers of the virus
$R(t)$	recovered persons

Table 2: Parameters used in the model

Parameters	description
Λ	births of total population
λ_I	proportion of births from the infected that will become infected acute
λ_C	proportion of births from the chronic that will become chronic
p_1	Proportion of infants aged $0-1$year that grow healthy to enter the S_2 compartment without being contaminated
p_2	Proportion of infants aged $1-5$ years who grow up healthy to enter the compartment S_3 without being contaminated
$\beta_{1,i}; \quad i = 1,..,4$	Adequate contact coefficient for people likely S_1 to be contaminated.
$\beta_{2,i}; \quad i = 1,..,4$	Adequate contact coefficient for individuals likely S_2 to be contaminated.
$\beta_{3,i}; \quad i = 1,..,4$	Adequate contact coefficient for individuals likely S_3 to be contaminated.
α_1	Probability of S_1 that will be contaminated, and become exposed infected E_I
$1-\alpha_1$	Probability of S_1 becoming contaminated, and exposed chronic E_C
α_2	Probability of S_2 that will be contaminated, and become exposed infected E_I
$1-\alpha_2$	Probability of S_2 that will be contaminated and become exposed chronic E_C
α_3	Probability of S_3 becoming exposed to infection E_I
$1-\alpha_3$	Probability of S_3 that will be contaminated and become chronically exposed E_C
δ_1	Proportion of exposed infected who become infected (acute) I.
δ_2	Proportion of exposed infected that become chronic C.
γ_I	Proportion of acute patients that are cured recovered from the infection process.
γ_C	Proportion of chronic carriers cured and recovered from the infection process.
μ	Natural mortality rate, i.e., mortality not caused by HBV.
μ_I	Death rate caused by acute HBV infection
μ_C	Mortality rate caused by chronic HBV infection

The model is composed of eight compartments. In Burkina Faso, the age at which an individual is infected influences his or her fate in terms of the disease's chronicity. It is therefore necessary to divide susceptible individuals into three age classes, i.e. three compartments of susceptible individuals. Dividing the susceptible population into 3 compartments provides a good approximation of the reality of hepatitis B epidemiology in Burkina Faso.

- The compartment S_1 represents the class of infants aged 0 to 1 year. In this compartment, the total population births is $\Lambda - \lambda_I I - \lambda_C C$. It emerges the infants who grow up with a proportion p_1 to enter the compartment S_2, the natural mortality μS_1, and also the babies who are infected by vertical transmission with a coefficient $\beta_{1,i}$, $i = 1, ..., 4$. The latter become exposed infected E_I with probability α_1, and chronic exposed E_C with probability $(1-\alpha_1)$.

- The compartment S_2 represents children aged 1 to 5 years. In this compartment,

infants from compartment S_1 with a proportion p_1 enter, and babies who grow up with a proportion of p_2 move to compartment S_3, natural mortality μS_2 and those infected with a coefficient $\beta_{1,i}$ for $i = 1, ..., 4$. The latter ones become exposed infected E_I with probability $(1 - \alpha_2)$, and chronic exposed E_C with probability $(1 - \alpha_2)$.

- The S_3 compartment is composed of children over 5 years old, adolescents and susceptible adults. In this compartment, the input stream is $p_2 S_2$, which represents children in compartment S_2 who grow up with a proportion p_2. The output stream is the natural mortality of susceptible S_3, and infected individuals who become infected with the coefficient $\beta_{3,i}$, $i = 1, 2, 3$. The latter ones become exposed infected E_I with probability α_3, and chronically exposed E_C with probability $(1 - \alpha_3)$.

There are two compartments of exposed people:

- The E_I compartment represents exposed infected individuals, i.e. exposed that will evolve towards an infectious state. The entry of this compartment is characterized by the susceptible that have been infected according to the coefficients $\alpha_i \beta_{i,j}$, $i = 1, 2, 3$, $j = 1, 2, 3$, and acute contaminated people $\lambda_I I$ who are infected by vertical transmission. The output is composed of natural mortality E_I, as well as the evolution towards an infectious state with a proportion δ_1.

- The E_C compartment represents the chronically exposed, i.e. exposed that will evolve towards a chronic state. The input of this compartment is characterized by susceptible individuals who have been infected according to the coefficients $(1 - \alpha_i)\beta_{i,j}$ for $(i = 1, 2, 3$ and $j = 1, 2, 3)$. Also, the input of the E_C compartment is characterized by individuals who are chronically infected through vertical transmission $\lambda_C C$. The exit is constituted on one hand by the natural mortality E_C, and on the other hand by the evolution towards an infectious state with a proportion δ_2.

Three further compartments remain:

- The acute infected compartment I, characterized by an input $\delta_1 E_I$, and an output composed of mortality $(\mu+\mu_I)I$ (natural mortality and mortality due to the disease), and recovery $\gamma_I I$.

- The compartment represents the chronically infected C. The entry of this compartment is E_C, and the output is composed of mortality $(\mu + \mu_C)C$ (natural mortality and mortality due to the disease), and recovery $\gamma_C C$.

- The compartment R (Recovered) represents individuals who are cured of the disease. There enters the cured ($\gamma_I I$ and $\gamma_C C$) and emerges the mortality μR.

The described interactions are illustrated in Figure 3.

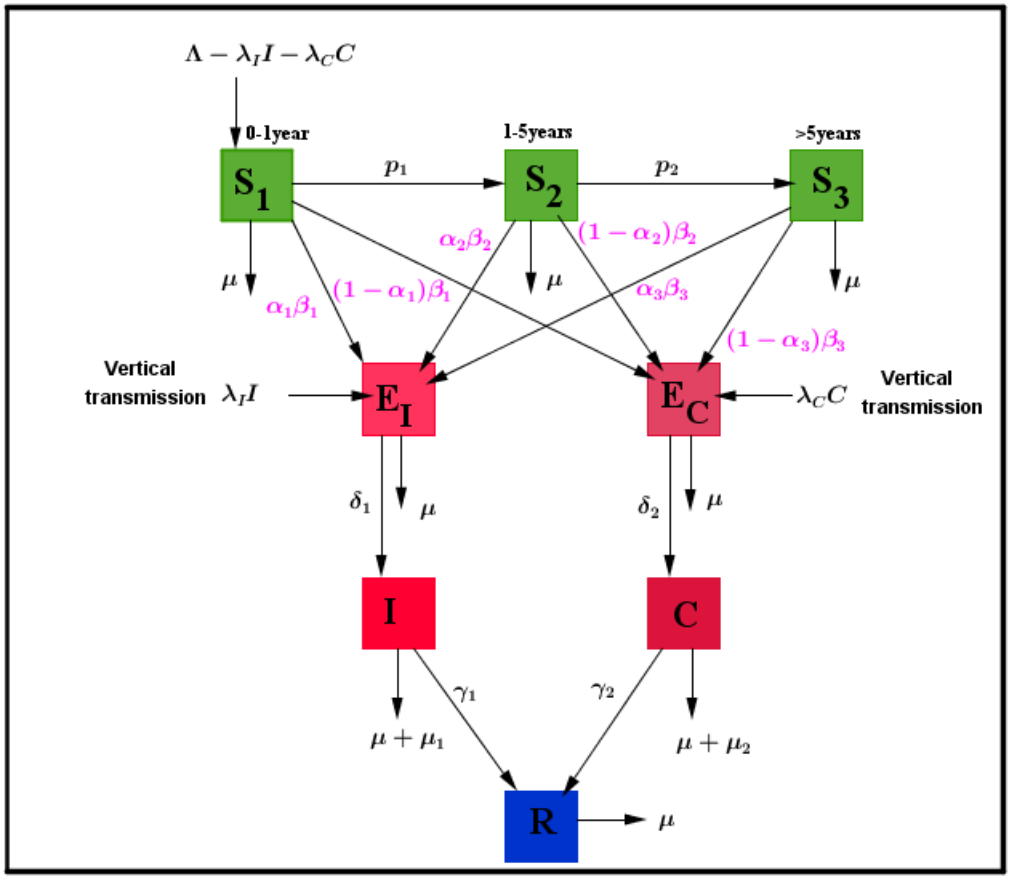

Figure 3: HBV model with vertical transmission

3. Mathematical Analysis of the model

3.1. Mathematical model

By doing a mass balance through the different considered compartments, one obtains the following system:

$$
\begin{cases}
\dfrac{dS_1}{dt} = \Lambda - \lambda_I I - \lambda_C C - \mu S_1 - p_1 S_1 - \beta_{1,1} E_I S_1 - \beta_{1,2} E_C S_1 - \beta_{1,3} I S_1 - \beta_{1,4} C S_1 \\
\dfrac{dS_2}{dt} = p_1 S_1 - \mu S_2 - \beta_{2,1} E_I S_2 - \beta_{2,2} E_C S_2 - \beta_{2,3} I S_2 - \beta_{2,4} C S_2 - p_2 S_2 \\
\dfrac{dS_3}{dt} = p_2 S_2 - \mu S_3 - \beta_{3,1} E_I S_3 - \beta_{3,2} E_I S_3 - \beta_{3,3} I S_3 - \beta_{3,4} C S_3 \\
\dfrac{dE_I}{dt} = \alpha_1(\beta_{1,1} E_I S_1 + \beta_{1,2} E_C S_1 + \beta_{1,3} I S_1 + \beta_{1,4} C S_1) + \alpha_2(\beta_{2,1} E_I S_2 + \beta_{2,2} E_C S_2 + \beta_{2,3} I S_2 + \beta_{2,4} C S_2) \\
\qquad +\alpha_3(\beta_{3,1} E_I S_3 + \beta_{3,2} E_C S_3 + \beta_{3,3} I S_3 + \beta_{3,4} C S_3) - \mu E_I - \delta_1 E_I + \lambda_I I \\
\dfrac{dE_C}{dt} = (1-\alpha_1)(\beta_{1,1} E_I S_1 + \beta_{1,2} E_C S_1 + \beta_{1,3} I S_1 + \beta_{1,4} C S_1) + (1-\alpha_2)(\beta_{2,1} E_I S_2 + \beta_{2,2} E_C S_2 + \beta_{2,3} I S_2 \\
\qquad +\beta_{2,4} C S_2) + (1-\alpha_3)(\beta_{3,1} E_I S_3 + \beta_{3,2} E_C S_3 + \beta_{3,3} I S_3 + \beta_{3,4} C S_3) + \lambda_C C - \mu E_C - \delta_2 E_C \\
\dfrac{dI}{dt} = \delta_1 E_I - \mu I - \mu_I I - \gamma_I I \\
\dfrac{dC}{dt} = \delta_2 E_C - \mu C - \mu_C C - \gamma_C C \\
\dfrac{dR}{dt} = \gamma_I I + \gamma_C C - \mu R
\end{cases}
\tag{1}
$$

The total number $N(t)$ of the population studied is not constant and:

$$N(t) = S_1(t) + S_2(t) + S_3(t) + E_I(t) + E_C(t) + I(t) + C(t) + R(t) \tag{2}$$

Since people and viruses have different dimensions, they therefore have to carry out the following normalization:

$$s_1 = \frac{S_1}{N_0},\ s_2 = \frac{S_2}{N_0},\ s_3 = \frac{S_3}{N_0},\ e_i = \frac{E_I}{N_0},\ e_c = \frac{E_C}{N_0},\ i = \frac{I}{N_0},\ c = \frac{C}{N_0},\ r = \frac{R}{N_0},\ \lambda = \frac{\Lambda}{N_0},$$

$$b_{i,j} = \beta_{i,j} N_0,\ i = 1, ..., 3;\ j = 1, ..., 4$$

Where $N_0 = N(0)$ is the total number of the initial population. Finally the normalized model becomes:

$$\begin{cases} \dfrac{ds_1}{dt} = \lambda - i\lambda_i - c\lambda_c - (\mu + p_1)s_1 - b_{1,1}e_i s_1 - b_{1,2}e_c s_1 - b_{1,3} i s_1 - b_{1,4} c s_1 \\ \dfrac{ds_2}{dt} = p_1 s_1 - (\mu + p_2)s_2 - b_{2,1}e_i s_2 - b_{2,2}e_c s_2 - b_{2,3} i s_2 - b_{2,4} c s_2 \\ \dfrac{ds_3}{dt} = p_2 s_2 - \mu s_3 - b_{3,1}e_i s_3 - b_{3,2}e_c s_3 - b_{3,3} i s_3 - b_{3,4} c s_3 \\ \dfrac{de_i}{dt} = \alpha_1 s_1(b_{1,1}e_i + b_{1,2}e_c + b_{1,3} i + b_{1,4} c) + \alpha_2 s_2(b_{2,1}e_i + b_{2,2}e_c + b_{2,3} i + b_{2,4} c) \\ \qquad +\alpha_3 s_3(b_{3,1}e_i + b_{3,2}e_c + b_{3,3} i + b_{3,4} c) - \mu e_i - \delta_1 e_i + i\lambda_i \\ \dfrac{de_c}{dt} = (1 - \alpha_1)s_1(b_{1,1}e_i + b_{1,2}e_c + b_{1,3} i + b_{1,4} c) + (1 - \alpha_2)s_2(b_{2,1}e_i + b_{2,2}e_c + b_{2,3} i + b_{2,4} c) \\ \qquad +(1 - \alpha_3)s_3(b_{3,1}e_i + b_{3,2}e_c + b_{3,3} i + b_{3,4} c) + c\lambda_c - \mu e_c - \delta_2 e_c \\ \dfrac{di}{dt} = \delta_1 e_i - \mu i - \gamma_i i - \mu_i i \\ \dfrac{dc}{dt} = \delta_2 e_c - \mu c - \gamma_c c - \mu_c c \\ \dfrac{dr}{dt} = \gamma_i i + \gamma_c c - \mu r \end{cases} \tag{3}$$

In the following, the positivity and existence of solutions for the normalized system (3) is given.

3.2. Positivity, boundedness and global existence of solutions

Theorem 1. *Let the initial value* $(s_{1,0}, s_{2,0}, s_{3,0}, e_{i,0}, e_{c,0}, i_0, c_0, r_0) \in \mathbb{R}^8_+$, *such that*

$$s_{1,0} + s_{2,0} + s_{3,0} + e_{i,0} + e_{c,0} + i_0 + c_0 + r_0 = 1,$$

and

$$0 \leq \lambda, \lambda_i, \lambda_c, \mu, p_1, p_2, \mu_i, \mu_c, \delta_1, \delta_2, \gamma_i, \gamma_c \leq 1$$
$$0 \leq b_{1,i}, b_{2,i}, b_{3,i} \leq 1 \quad \textit{for all } 1 \leq i \leq 4,$$

then there exists a unique, nonnegative, bounded global solution to system (3). *Moreover for all* $t \geq 0$

i)

$$0 \leq n(t) \leq \frac{\lambda}{\mu} + 1 \tag{4}$$

where

$$n(t) = s_1(t) + s_2(t) + s_3(t) + e_i(t) + e_c(t) + i(t) + c(t) + r(t)$$

ii) Moreover, if $s_{1,0} \leq \dfrac{\lambda}{\mu + p_1}$, $s_{2,0} \leq \dfrac{p_1\lambda}{(\mu + p_1)(\mu + p_2)}$ *and* $s_{3,0} \leq \dfrac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)}$ *then*

$$s_1(t) \leq \frac{\lambda}{\mu + p_1}, \quad s_2(t) \leq \frac{p_1\lambda}{(\mu + p_1)(\mu + p_2)}, \quad s_3(t) \leq \frac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)}.$$

Proof.

i) For the local existence, all the functions of system (3) are locally Lipschitz continuous. Thus, there exists a unique local solution on $t \in [0, T_{max})$, where T_{max} is the explosion time. The analysis of this kind of system is based on elementary methods of ordinary differential equations. The existence of unique solutions is guaranteed by various fixed point theorems on a maximal interval $[0, T_{max})$. By proving that the components of the solution vector $(s_1(t),\ s_2(t),\ s_3(t),\ e_i(t),\ e_c(t),\ i(t),\ c(t),\ r(t))$ are uniformly bounded on any bounded interval $[0, T_{max})$, one ensures that $T_{max} = \infty$. We remark that the components of the vector

$$F(s_1, s_2, s_3, e_i, e_c, i, c, r) = \begin{pmatrix} F_1(s_1, s_2, s_3, e_i, e_c, i, c, r) \\ F_2(s_1, s_2, s_3, e_i, e_c, i, c, r) \\ F_3(s_1, s_2, s_3, e_i, e_c, i, c, r) \\ F_4(s_1, s_2, s_3, e_i, e_c, i, c, r) \\ F_5(s_1, s_2, s_3, e_i, e_c, i, c, r) \\ F_6(s_1, s_2, s_3, e_i, e_c, i, c, r) \\ F_7(s_1, s_2, s_3, e_i, e_c, i, c, r) \\ F_8(s_1, s_2, s_3, e_i, e_c, i, c, r) \end{pmatrix} \tag{5}$$

Where

$$F_1(s_1, s_2, s_3, e_i, e_c, i, c, r) = \lambda - i\lambda_i - c\lambda_c - (\mu + p_1)s_1 - b_{1,1}e_i s_1 - b_{1,2}e_c s_1 - b_{1,3}i s_1 - b_{1,4}c s_1$$

$$F_2(s_1, s_2, s_3, e_i, e_c, i, c, r) = p_1 s_1 - (\mu + p_2)s_2 - b_{2,1}e_i s_2 - b_{2,2}e_c s_2 - b_{2,3}i s_2 - b_{2,4}c s_2$$

$$F_3(s_1, s_2, s_3, e_i, e_c, i, c, r) = p_2 s_2 - \mu s_3 - b_{3,1}e_i s_3 - b_{3,2}e_c s_3 - b_{3,3}i s_3 - b_{3,4}c s_3$$

$$\begin{aligned} F_4(s_1, s_2, s_3, e_i, e_c, i, c, r) = {} & \alpha_1 s_1(b_{1,1}e_i + b_{1,2}e_c + b_{1,3}i + b_{1,4}c) + \alpha_2 s_2(b_{2,1}e_i + b_{2,2}e_c + b_{2,3}i + b_{2,4}c) + \\ & + \alpha_3 s_3(b_{3,1}e_i + b_{3,2}e_c + b_{3,3}i + b_{3,4}c) + \lambda_i i - \mu e_i - \delta_1 e_i \end{aligned}$$

$$F_5(s_1,s_2,s_3,e_i,e_c,i,c,r) = (1-\alpha_1)s_1(b_{1,1}e_i + b_{1,2}e_c + b_{1,3}i + b_{1,4}c) + (1-\alpha_2)s_2(b_{2,1}e_i + b_{2,2}e_c + b_{2,3}i + b_{2,4}c) + +(1-\alpha_3)s_3(b_{3,1}e_i + b_{3,2}e_c + b_{3,3}i + b_{3,4}c) + + \lambda_c c - \mu e_c - \delta_2 e_c$$

$$F_6(s_1,s_2,s_3,e_i,e_c,i,c,r) = \delta_1 e_i - \mu i - \gamma_i i - \mu_i i$$

$$F_7(s_1,s_2,s_3,e_i,e_c,i,c,r) = \delta_2 e_c - \mu c - \gamma_c c - \mu_c c$$

$$F_8(s_1,s_2,s_3,e_i,e_c,i,c,r) = \gamma_i i + \gamma_c c - \mu r$$

are quasi-positive. Consequently, since the initial conditions are nonnegative, this implies that the solution components are nonnegative for all $t \in [0, T_{max})$.
Now, let the function n be defined as

$$n(t) = s_1(t) + s_2(t) + s_3(t) + e_i(t) + e_c(t) + i(t) + c(t) + r(t)$$

By taking the sum of the first eight equations in (S), we observe

$$\begin{cases} \dfrac{dn}{dt} \leq \lambda - \mu S(t) \\ n(0) = 1. \end{cases} \tag{6}$$

Integrating equation (6) over $(0,t)$ for all $0 < t < T$, one can get the following

$$n(t)e^{\mu t} - 1 \leq \frac{\lambda}{\mu}(e^{\mu t} - 1),$$

which implies that

$$n(t) \leq e^{-\mu t} + \frac{\lambda}{\mu}(1 - e^{-\mu t}).$$

Therefore

$$n(t) \leq (1 - \frac{\lambda}{\mu})e^{-\mu t} + \frac{\lambda}{\mu}.$$

Here, two different cases are distinguished. If $\dfrac{\lambda}{\mu} < 1$, then the following inequality is satisfied

$$n(t) \leq 1 - \frac{\lambda}{\mu} + \frac{\lambda}{\mu} \leq 1,$$

otherwise, if $\dfrac{\lambda}{\mu} \geq 1$ then

$$n(t) \leq \frac{\lambda}{\mu}.$$

Finally, one can get the following

$$n(t) \leq \frac{\lambda}{\mu} + 1. \tag{7}$$

Hence, $T_{max} = \infty$ and the existence of unique, non-negative and bounded global solution are proved.

ii) We remark that s_1 satisfies the following

$$\begin{cases} \dfrac{ds_1}{dt} \leq \lambda - (\mu + p_1)s_1(t) \\ s_1(0) = s_{1,0}. \end{cases} \tag{8}$$

By integrating (8) over $(0, t)$ for all $0 < t < T$, we obtain

$$s_1(t)e^{(\mu+p_1)t} \leq \frac{\lambda}{(\mu + p_1)}(e^{(\mu+p_1)t} - 1) + s_{1,0}.$$

Since $s_{1,0} \leq \dfrac{\lambda}{(\mu + p_1)}$, we obtain $s_1(t) \leq \dfrac{\lambda}{(\mu + p_1)}$.

The same reasoning is applied for s_2 and s_3 which concludes the proof of this theorem.

3.3. Reproduction rate and its interpretations

Infectious diseases are generally modeled mathematically by compartmental models. Individuals can move from one compartment to another, and the population is represented by compartments with labels. The first models date back to the beginning of the 20^{th} century by [36] in 1916, [37, 38] in 1917, [22] in 1927 and [21] in 1956.

Ordinary differential equations (deterministic, stochastic or fractional) are used to analyze these compartmental models mathematically. These mathematical models can be used to predict the spread of a disease, the total number of people infected or the duration of an epidemic, as well as to estimate various epidemiological parameters such as the reproduction rate [1], [3].

This section is devoted to the calculation of the reproduction number $\mathcal{R}_0$ of our proposed model. For this we will use the generation matrix method which was developed by [8] and then adopted by [40] for finite dimensional systems.

Theorem 2. *Consider the system* (3) *with the given parameters*

$$0 \leq \lambda, \lambda_i, \lambda_c, \mu, p_1, p_2, \mu_i, \mu_c, \delta_1, \delta_2, \gamma_i, \gamma_c \leq 1$$

$$0 \leq b_{1,i}, b_{2,i}, b_{3,i} \leq 1 \quad \textit{for all } 1 \leq i \leq 4,$$

Then,

(i) the disease free equilibrium point (DFE) is

$$\mathcal{X}_0 = \left(\frac{\lambda}{\mu + p_1},\ \frac{p_1\lambda}{(\mu+p_1)(\mu+p_2)},\ \frac{p_1p_2\lambda}{\mu(\mu+p_1)(\mu+p_2)}, 0,\ 0,\ 0,\ 0,\ 0 \right), \tag{9}$$

(ii) the reproduction rate is

$$\mathcal{R}_0 = \frac{\eta_{1,1} + \eta_{2,2} + \left((\eta_{1,1} - \eta_{2,2})^2 + 4\eta_{1,2}\eta_{2,1} \right)^{\frac{1}{2}}}{2} \tag{10}$$

Where $\eta_{1,1},\ \eta_{2,2},\ \eta_{1,2},\ \eta_{2,1}$ *are given by* (17)

Proof. Here, we consider the proposed mathematical model (3) with eight homogeneous compartments. This model can be written as

$$\frac{d}{dt}(s_1, s_2, s_3, e_i, e_c, i, c, r) = F(s_1, s_2, s_3, e_i, e_c, i, c, r).$$

Where F is defined by (5). The point $\mathcal{X}_0$ defined by (9) satisfies $F(\mathcal{X}_0) = 0$.

Infected and chronic exposed are infected but not infectious, so they do not participate in the transmission of the virus. Thus $b_{k,1} = b_{k,2} = 0; k = 1, 2, 3$. To compute the reproduction rate $\mathcal{R}_0$, one must consider only the infected and infectious compartments that satisfy the following fourth-order system :

$$\begin{aligned}
&\frac{d}{dt}\begin{pmatrix} e_i \\ e_c \\ i \\ c \end{pmatrix} \\
&= \begin{pmatrix}
\alpha_1 s_1(b_{1,3}i + b_{1,4}c) + \alpha_2 s_2(b_{2,3}i + b_{2,4}c) + \alpha_3 s_3(b_{3,3}i + b_{3,4}c) - \mu e_i - \delta_1 e_i + i\lambda_i \\
(1-\alpha_1)s_1(b_{1,3}i + b_{1,4}c) + (1-\alpha_2)s_2(b_{2,3}i + b_{2,4}c) + (1-\alpha_3)s_3(b_{3,3}i + b_{3,4}c) + c\lambda_c - \mu e_c - \delta_2 e_c \\
\delta_1 e_i - \mu i - \gamma_i i - \mu_i i \\
\delta_2 e_c - \mu c - \gamma_c c - \mu_c c
\end{pmatrix}.
\end{aligned}$$

The rate of occurrence of new infection in the four compartments (e_i, e_c, i, c) is represented by the vector $\mathcal{U}$ as follows

$$\mathcal{U} = \begin{pmatrix}
\alpha_1 s_1(b_{1,3}i + b_{1,4}c) + \alpha_2 s_2(b_{2,3}i + b_{2,4}c) + \alpha_3 s_3(b_{3,3}i + b_{3,4}c) + \lambda_i i \\
(1-\alpha_1)s_1(b_{1,3}i + b_{1,4}c) + (1-\alpha_2)s_2(b_{2,3}i + b_{2,4}c) + (1-\alpha_3)s_3(b_{3,3}i + b_{3,4}c) + \lambda_c c \\
0 \\
0
\end{pmatrix} \tag{11}$$

and the transfer rate of individuals into and out of the infected compartments is given by the vector $\mathcal{T}$

$$\mathcal{T} = \begin{pmatrix} \mu e_i + \delta_1 e_i \\ \mu e_c + \delta_2 e_c \\ -\delta_1 e_i + \mu i + \gamma_i i + \mu_i i \\ -\delta_2 e_c + \mu c + \gamma_c c + \mu_c c \end{pmatrix} \tag{12}$$

Thus, all the epidemiological events leading to new infections are incorporated into the model via the matrix Θ, while all the other events are included in the matrix Γ. Progression to either death or immunity ensures that Γ is invertible. By substituting the variables (s_1, s_2, s_3) with the free equilibrium point $\mathcal{X}_0$, we obtain the two matrices Θ and Γ which are expressed as follows :

$$\Theta = \begin{bmatrix} 0 & 0 & \Theta_{1,3} & \Theta_{1,4} \\ 0 & 0 & \Theta_{2,3} & \Theta_{2,4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{13}$$

Where

$$\Theta_{1,3} = \left[\alpha_1 b_{1,3} + \alpha_2 b_{2,3}\frac{p_1}{\mu + p_2} + \alpha_3 b_{3,3}\frac{p_1 p_2}{\mu(\mu + p_2)}\right]\frac{\lambda}{\mu + p_1} + \lambda_i$$

$$\Theta_{1,3} = \left[\alpha_1 b_{1,4} + \alpha_2 b_{2,4}\frac{p_1}{\mu + p_2} + \alpha_3 b_{3,4}\frac{p_1 p_2}{\mu(\mu + p_2)}\right]\frac{\lambda}{\mu + p_1}$$

$$\Theta_{2,3} = \left[(1-\alpha_1)\beta_{1,3} + (1-\alpha_2) b_{2,3}\frac{p_1}{\mu + p_2} + (1-\alpha_3) b_{3,3}\frac{p_1 p_2}{\mu(\mu + p_2)}\right]\frac{\lambda}{\mu + p_1}$$

$$\Theta_{2,4} = \left[(1-\alpha_1) b_{1,4} + (1-\alpha_2) b_{2,4}\frac{p_1}{\mu + p_2} + (1-\alpha_3) b_{3,4}\frac{p_1 p_2}{\mu(\mu + p_2)}\right]\frac{\lambda}{\mu + p_1} + \lambda_c$$

$$\Gamma = \begin{bmatrix} \mu + \delta_1 & 0 & 0 & 0 \\ 0 & \mu + \delta_2 & 0 & 0 \\ -\delta_1 & 0 & \mu + \gamma_i + \mu_i & 0 \\ 0 & -\delta_2 & 0 & \mu + \gamma_c + \mu_c \end{bmatrix} \tag{14}$$

By calculating Γ^{-1}, we obtain :

$$\Gamma^{-1} = \begin{bmatrix} \frac{1}{\mu+\delta_1} & 0 & 0 & 0 \\ 0 & \frac{1}{\mu+\delta_2} & 0 & 0 \\ \frac{\delta_1}{(\mu+\gamma_i+\mu_i)(\mu+\delta_1)} & 0 & \frac{1}{\mu+\gamma_i+\mu_i} & 0 \\ 0 & \frac{\delta_2}{(\mu+\gamma_c+\mu_c)(\mu+\delta_2)} & 0 & \frac{1}{\mu+\gamma_c+\mu_c} \end{bmatrix} \tag{15}$$

We calculate the matrix of the new generation, we obtain :

$$\Theta\Gamma^{-1} = \begin{bmatrix} \eta_{1,1} & \eta_{1,2} & \eta_{1,3} & \eta_{1,4} \\ \eta_{2,1} & \eta_{2,2} & \eta_{2,3} & \eta_{2,4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \tag{16}$$

Where

$$\begin{aligned}
\eta_{1,1} &= \left[\left(\alpha_1\beta_{1,3} + \alpha_2 b_{2,3}\frac{p_1}{\mu+p_2} + \alpha_3 b_{3,3}\frac{p_1p_2}{\mu(\mu+p_2)}\right)\frac{\lambda}{\mu+p_1} + \lambda_i\right]\frac{\delta_1}{(\mu+\gamma_i+\mu_i)(\mu+\delta_1)} \\
\eta_{1,2} &= \left[\alpha_1 b_{1,4} + \alpha_2 b_{2,4}\frac{p_1}{\mu+p_2} + \alpha_3 b_{3,4}\frac{p_1p_2}{\mu(\mu+p_2)}\right]\frac{\lambda}{(\mu+p_1)}\frac{\delta_2}{(\mu+\gamma_c+\mu_c)(\mu+\delta_2)} \\
\eta_{1,3} &= \left[\left(\alpha_1 b_{1,3} + \alpha_2 b_{2,3}\frac{p_1}{\mu+p_2} + \alpha_3 b_{3,3}\frac{p_1p_2}{\mu(\mu+p_2)}\right)\frac{\lambda}{\mu+p_1} + \lambda_i\right]\frac{1}{\mu+\gamma_i+\mu_i} \\
\eta_{1,4} &= \left[\alpha_1 b_{1,4} + \alpha_2 b_{2,4}\frac{p_1}{\mu+p_2} + \alpha_3 b_{3,4}\frac{p_1p_2}{\mu(\mu+p_2)}\right]\frac{\lambda}{(\mu+p_1)(\mu+\gamma_c+\mu_c)} \\
\eta_{2,1} &= \left[(1-\alpha_1)b_{1,3} + (1-\alpha_2)b_{2,3}\frac{p_1}{\mu+p_2} + (1-\alpha_3)b_{3,3}\frac{p_1p_2}{\mu(\mu+p_2)}\right]\frac{\lambda}{(\mu+p_1)}\frac{\delta_1}{(\mu+\gamma_i+\mu_i)(\mu+\delta_1)} \\
\eta_{2,2} &= \left[\left((1-\alpha_1)b_{1,4} + (1-\alpha_2)b_{2,4}\frac{p_1}{\mu+p_2} + (1-\alpha_3)b_{3,4}\frac{p_1p_2}{\mu(\mu+p_2)}\right)\frac{\lambda}{\mu+p_1} + \lambda_c\right]\frac{\delta_2}{(\mu+\gamma_c+\mu_c)(\mu+\delta_2)} \\
\eta_{2,3} &= \left[(1-\alpha_1)b_{1,3} + (1-\alpha_2)b_{2,3}\frac{p_1}{\mu+p_2} + (1-\alpha_3)b_{3,3}\frac{p_1p_2}{\mu(\mu+p_2)}\right]\frac{\lambda}{(\mu+p_1)(\mu+\gamma_i+\mu_i)} \\
\eta_{2,4} &= \left[\left((1-\alpha_1)b_{1,4} + (1-\alpha_2)b_{2,4}\frac{p_1}{\mu+p_2} + (1-\alpha_3)b_{3,4}\frac{p_1p_2}{\mu(\mu+p_1)}\right)\frac{\lambda}{(\mu+p_1)} + \lambda_c\right]\frac{1}{\mu+\gamma_c+\mu_c}
\end{aligned} \tag{17}$$

By the next generation matrix approach, $\mathcal{R}_0$ for the model. By calculating the spectral radius of the next generation matrix $\Theta\Gamma^{-1}$, we get :

$$\begin{aligned}\mathcal{R}_0 &= \rho\left(\Theta\Gamma^{-1}\right)\\ &= \max\{|\omega_i|, i=1,2,3\}\\ &= \max\left\{\left|\frac{\eta_{1,1}+\eta_{2,2}+\left(\left(\eta_{1,1}-\eta_{2,2}\right)^2+4\eta_{1,2}\eta_{2,1}\right)^{\frac{1}{2}}}{2}\right|, \left|\frac{\eta_{1,1}+\eta_{2,2}-\left(\left(\eta_{1,1}-\eta_{2,2}\right)^2+4\eta_{1,2}\eta_{2,1}\right)^{\frac{1}{2}}}{2}\right|, 0\right\}\end{aligned} \tag{18}$$

Then the reproduction rate $\mathcal{R}_0$ of the normalized system is given as :

$$\mathcal{R}_0 = \frac{\eta_{1,1}+\eta_{2,2}+\left(\left(\eta_{1,1}-\eta_{2,2}\right)^2+4\eta_{1,2}\eta_{2,1}\right)^{\frac{1}{2}}}{2} \tag{19}$$

Where $\eta_{1,1}$, $\eta_{2,2}$, $\eta_{1,2}$, $\eta_{2,1}$ are given by (17).

Remark 1. *The reproduction number $\mathcal{R}_0$ has a crucial role in the control of Hepatitis B viral infection. When this number is less than* 1*, an infected individual infects less than one other individual on average, therefore the disease is under control. Conversely, when this number exceeds* 1*, the disease spreads through the population and becomes epidemic [40].*

4. Global stability analysis of the model problem for the disease free equilibrium point

In the section, the global stability analysis of the model for the disease free equilibrium is shown. In the following, we aim to provide a brief investigation of the Castillo Chavez technique [5, 6] to prove the stability of system (3) in the global sense at the disease free equilibrium point. Therefore, by applying the Castillo Chavez technique [6], the given problem (3) is converted into the following sub-models:

$$\begin{cases}\dfrac{dX_1}{dt} = F(X_1, X_2),\\[2ex] \dfrac{dX_2}{dt} = G(X_1, X_2),\\[2ex] G(X_1, 0) = 0.\end{cases} \tag{20}$$

Where X_1 and X_2 designate the population of uninfected individuals, and infected individuals, respectively.
The following conditions (A1) and (A2) must be satisfied to guarantee the local asymptotic stability.

(A1) If $\dfrac{dX_1}{dt} = F(X_1, 0)$ then $\mathcal{X}_0$ is globally asymptotically stable.

(A2) $G(X_1, X_2) = AX_2 - \hat{G}(X_1, X_2)$.

Where $\hat{G}(X_1, X_2) \geq 0$ for $(X_1, X_2) \in \Omega$, the matrix A is a M-matrix whose off diagonal elements are nonnegative.

In the proposed system (3), $X_1 = (s_1, s_2, s_3, r) \in \mathbf{R}^4$ and $X_1 = (e_i, e_c, i, c) \in \mathbf{R}^4$. According to the results obtained in Section 3, the free disease equilibrium point (DFE) was denoted by $\mathcal{X}_0$, it is defined by the following sense

$$\mathcal{X}_0 = \left(\frac{\lambda}{\mu + p_1},\ \frac{p_1\lambda}{(\mu + p_1)(\mu + p_2)},\ \frac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)}, 0,\ 0,\ 0,\ 0,\ 0 \right)$$

which can be written as $\mathcal{X}_0 = (s^0, 0)$.

In order to ensure the globally asymptotically stability of DFE point, the results given above [6] were applied.

Theorem 3. *If* $\mathcal{R}_0 < 1$ *and* $s_{1,0} \leq \dfrac{\lambda}{\mu + p_1}$, $s_{2,0} \leq \dfrac{p_1\lambda}{(\mu + p_1)(\mu + p_2)}$, $s_{3,0} \leq \dfrac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)}$, *then the DFE point* $\mathcal{X}_0$ *of the model* (3) *is globally asymptotically stable.*

Proof. In System 20, since infected and chronically exposed individuals are infected but not infectious. Therefore, they do not participate in the transmission of the virus. Hence $b_{k,1} = 0$ and $b_{k,2} = 0$ for $k = 1, 2, 3$. Here, one can set the following

$$F(X_1, X_2) = \begin{pmatrix} \lambda - i\lambda_1 - c\lambda_2 - (\mu + p_1)s_1 - b_{1,3}is_1 - b_{1,4}cs_1 \\ p_1 s_1 - (\mu + p_2)s_2 - b_{2,3}is_2 - b_{2,4}cs_2 \\ p_2 s_2 - \mu s_3 - b_{3,3}is_3 - b_{3,4}cs_3 \\ \gamma_1 i + \gamma_2 c - \mu r \end{pmatrix}$$

and

$$G(X_1, X_2) = \begin{pmatrix} G_1(X_1, X_2) \\ G_2(X_1, X_2) \\ G_3(X_1, X_2) \\ G_4(X_1, X_2) \end{pmatrix}$$

Where each component is defined by the following sense

$$\begin{aligned}
G_1(X_1, X_2) &= \alpha_1 s_1(b_{1,3}i + b_{1,4}c) + \alpha_2 s_2(b_{2,3}i + b_{2,4}c) + \alpha_3 s_3(b_{3,3}i + b_{3,4}c) - \mu e_i - \delta_1 e_i + i\lambda_i \\
G_2(X_1, X_2) &= (1 - \alpha_1)s_1(b_{1,3}i + b_{1,4}c) + (1 - \alpha_2)s_2(b_{2,3}i + b_{2,4}c) + (1 - \alpha_3)s_3(b_{3,3}i + b_{3,4}c) \\
&\quad + c\lambda_c - \mu e_c - \delta_2 e_c \\
G_3(X_1, X_2) &= \delta_1 e_i - \mu i - \gamma_i i - \mu_i i \\
G_4(X_1, X_2) &= \delta_2 e_c - \mu c - \gamma_c c - \mu_c c.
\end{aligned}$$

At the DFE point, it is clear that $G(X_1, 0) = 0$.
Now, we need to prove that (A_1) is satified. To this aim, we calculate the eignvalues of the Jacobian matrix associated to F at the DFE point.

$$D_{X_1}F(\mathcal{X}_0) = \begin{pmatrix} -(\mu+p_1) & 0 & 0 & 0 \\ p_1 & -(\mu+p_2) & 0 & 0 \\ 0 & p_2 & -\mu & 0 \\ 0 & 0 & 0 & -\mu \end{pmatrix} \tag{21}$$

Since the eigenvalues $\zeta_1 = -(\mu+p_1)$, $\zeta_2 = -(\mu+p_2)$, $\zeta_3 = -\mu$, $\zeta_4 = -\mu$ of the jacobian matrix are negatives, then the DFE point $\mathcal{X}_0$ is globally asymptotically stable.
To derive the condition (A_2), we first calculate the matrix $A = D_{X_2}G(\mathcal{X}_0)$ at the DFE point

$$A = \begin{pmatrix} -(\mu+\delta_1) & 0 & \lambda_i+\Theta_1 & \Theta_2 \\ 0 & -(\mu+\delta_2) & \Theta_3 & \lambda_c+\Theta_4 \\ \delta_1 & 0 & -(\mu+\gamma_i+\mu_i) & 0 \\ 0 & \delta_2 & 0 & -(\mu+\gamma_c+\mu_c) \end{pmatrix} \tag{22}$$

Where

$$\begin{aligned}
\Theta_1 &= \alpha_1 b_{1,3}\frac{\lambda}{\mu+p_1} + \alpha_2 b_{2,3}\frac{p_1\lambda}{(\mu+p_1)(\mu+p_2)} + \alpha_3 b_{3,3}\frac{p_1p_2\lambda}{\mu(\mu+p_1)(\mu+p_2)} \\
\Theta_2 &= \alpha_1 b_{1,4}\frac{\lambda}{\mu+p_1} + \alpha_2 b_{2,4}\frac{p_1\lambda}{(\mu+p_1)(\mu+p_2)} + \alpha_3 b_{3,4}\frac{p_1p_2\lambda}{\mu(\mu+p_1)(\mu+p_2)} \\
\Theta_3 &= (1-\alpha_1) b_{1,3}\frac{\lambda}{\mu+p_1} + (1-\alpha_2) b_{2,3}\frac{p_1\lambda}{(\mu+p_1)(\mu+p_2)} + (1-\alpha_3) b_{3,3}\frac{p_1p_2\lambda}{\mu(\mu+p_1)(\mu+p_2)} \\
\Theta_4 &= (1-\alpha_1) b_{1,4}\frac{\lambda}{\mu+p_1} + (1-\alpha_2) b_{2,4}\frac{p_1\lambda}{(\mu+p_1)(\mu+p_2)} + (1-\alpha_3) b_{3,4}\frac{p_1p_2\lambda}{\mu(\mu+p_1)(\mu+p_2)}
\end{aligned} \tag{23}$$

It is clear that the matrix A given above is an M-matrix.
Now, one can calculate the following function $\hat{G}(X_1, X_2)$. We have

$$\hat{G}(X_1, X_2) = \begin{pmatrix} \hat{G}_1(X_1, X_2) \\ \hat{G}_2(X_1, X_2) \\ 0 \\ 0 \end{pmatrix} \tag{24}$$

$$\hat{G}_1(X_1, X_2) = -\alpha_1 s_1(b_{1,3}i + b_{1,4}c) - \alpha_2 s_2(b_{2,3}i + b_{2,4}c) - \alpha_3 s_3(b_{3,3}i + b_{3,4}c) + i\Theta_1 + c\Theta_2$$

$$\hat{G}_2(X_1, X_2) = -(1-\alpha_1)s_1(b_{1,3}i + b_{1,4}c) - (1-\alpha_2)s_2(b_{2,3}i + b_{2,4}c) - (1-\alpha_3)s_3(b_{3,3}i + b_{3,4}c) +$$

$$+ i\Theta_3 + c\Theta_4.$$

By replacing Θ_1, Θ_2, Θ_3 and Θ_4 by their expressions given in (23), one obtains the following result

$$\begin{aligned}\hat{G}_1(X_1, X_2) = {} & \alpha_1 b_{1,3} i\left(\frac{\lambda}{\mu + p_1} - s_1\right) + \alpha_2 b_{2,3} i\left(\frac{p_1\lambda}{(\mu + p_1)(\mu + p_2)} - s_2\right) + \alpha_3 b_{3,3} i\left(\frac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)} - s_3\right) + \\ & + \alpha_1 b_{1,4} c\left(\frac{\lambda}{\mu + p_1} - s_1\right) + \alpha_2 b_{2,4} c\left(\frac{p_1\lambda}{(\mu + p_1)(\mu + p_2)} - s_2\right) + \alpha_3 b_{3,4} c\left(\frac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)} - s_3\right)\end{aligned}$$

$$\begin{aligned}\hat{G}_2(X_1, X_2) = {} & (1-\alpha_1) b_{1,3} i\left(\frac{\lambda}{\mu + p_1} - s_1\right) + (1-\alpha_2) b_{2,3} i\left(\frac{p_1\lambda}{(\mu + p_1)(\mu + p_2)} - s_2\right) + \\ & + (1-\alpha_3) b_{3,3} i\left(\frac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)} - s_3\right) + (1-\alpha_1) b_{1,4} c\left(\frac{\lambda}{\mu + p_1} - s_1\right) + \\ & + (1-\alpha_2) b_{2,4} c\left(\frac{p_1\lambda}{(\mu + p_1)(\mu + p_2)} - s_2\right) + (1-\alpha_3) b_{3,4} c\left(\frac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)} - s_3\right)\end{aligned}$$

Thanks To Theorem 1, ii), one gets the following result

$$\frac{\lambda}{\mu + p_1} - s_1 \geq 0,\ \frac{p_1\lambda}{(\mu + p_1)(\mu + p_2)} - s_2 \geq 0,\ \frac{p_1 p_2 \lambda}{\mu(\mu + p_1)(\mu + p_2)} - s_3 \geq 0.$$

Since i, c, $b_{1,3}$, $b_{1,3}$, $b_{2,3}$, $b_{3,3}$, $b_{1,4}$, $b_{2,4}$, $b_{3,3}$ are nonnegatives, and $0 \leq \alpha_k \leq 1$ for $k = 1, 2, 3$, the following result is obtained

$$\hat{G}_1(X_1, X_2) \geq 0,\ \hat{G}_2(X_1, X_2) \geq 0.$$

Consequently, the hypothesis (A_1) and (A_2) are satisfied. Moreover, one uses Castillo Chavez [6] technique to conclude that if $\mathcal{R}_0 < 1$ then the DFE point is globally asymptotically stable.

5. Endemic Equilibrium Point $\mathcal{E}^*$

In this section, the global stability analysis of the proposed model problem for the endemic equilibrium point is studied. To prove this result, Lyapunov functions are used. The concept of global asymptotic stability refers to the property that if a system starts near the endemic point, it will eventually converge to that point.

5.1. Exixtence of $\mathcal{E}^*$

Theorem 4. *System (1) admits a unique positive emdemic equilibrium* $\mathcal{E}^* = (S_1^*,\ S_2^*,\ S_3^*,\ E_I^*,\ E_C^*,\ I^*,\ C^*,\ R^*)$ *whenever* $\mathcal{R}_0 > 1$

Proof. By seting the right hand side of system (1) equal to zero, and keeping each state variable non-zero ($S_1 \neq 0,\ S_2 \neq 0,\ S_3 \neq 0,\ E_I \neq 0,\ E_C \neq 0,\ I \neq 0,\ C \neq 0,\ R \neq 0$), then one obtains

$$S_1^* = \frac{\Lambda - \Lambda_I I^* - \Lambda_C C^*}{\mu + p_1 + \beta_{1,1}E_I^* + \beta_{1,2}E_C^* + \beta_{1,3}I^* + \beta_{1,4}C^*},$$

$$S_2^* = \frac{p_1 S_1^*}{\mu + p_2 + \beta_{2,1}E_I^* + \beta_{2,2}E_C^* + \beta_{2,3}I^* + \beta_{2,4}C^*},$$

$$S_3^* = \frac{p_2 S_2^*}{\mu\beta_{3,1}E_I^* + \beta_{3,2}E_C^* + \beta_{3,3}I^* + \beta_{3,4}C^*},$$

$$E_I^* = \frac{\Lambda_I I^* + \Lambda_C C^* \alpha_1 \left(\beta_{1,2}E_C^* S_1^* - \beta_{1,3}I^* S_1^* - \beta_{1,4}C^* S_1^*\right)}{\mu + \delta_1 - \alpha_1\beta_{1,1}S_1^* - \alpha_2\beta_{2,1}S_2^* - \alpha_3\beta_{3,1}S_3^*} +$$
$$\frac{\alpha_2(\beta_{2,2}E_C^* S_2^* - \beta_{2,3}I^* S_2^* - \beta_{2,4}C^* S_2^*) + \alpha_3(\beta_{3,2}E_C^* S_3^* - \beta_{3,3}I^* S_3^* - \beta_{3,4}C^* S_3^* + \beta_{2,5}T^* S_3^*)}{\mu + \delta_1 - \alpha_1\beta_{1,1}S_1^* - \alpha_2\beta_{2,1}S_2^* - \alpha_3\beta_{3,1}S_3^*},$$

$$E_C^* = \frac{\Lambda_I I^* + \lambda_C C^* + (1-\alpha_1)(\beta_{1,1}E_I^* S_1^* - \beta_{1,3}I^* S_1^* - \beta_{1,4}C^* S_1^*)}{\mu + \delta_2 - \alpha_1\beta_{1,1}S_1^* - \alpha_2\beta_{2,1}S_2^* - \alpha_3\beta_{3,1}S_3^*} +$$
$$\frac{(1-\alpha_2)(\beta_{2,1}E_I^* S_2^* - \beta_{2,3}I^* S_2^* - \beta_{2,4}C^* S_2^*) + (1-\alpha_3)(\beta_{3,1}E_I^* S_3^* - \beta_{3,3}I^* S_3^* - \beta_{3,4}C^* S_3^*)}{\mu + \delta_2 - \alpha_1\beta_{1,1}S_1^* - \alpha_2\beta_{2,1}S_2^* - \alpha_3\beta_{3,1}S_3^*},$$

$$I^* = \frac{\delta_1 E_I^*}{\mu + \gamma_I + \mu_I}, \qquad C^* = \frac{\delta_2 E_C^*}{\mu + \gamma_C + \mu_C}, \qquad R^* = \frac{\gamma_I I^* + \gamma_C C^*}{\mu}$$

5.2. Global stability of $\mathcal{E}^*$

In this subsubsection, by constructing suitable Lyapunov function, we will prove the global asymptotic stability of the endemic equilibrium point $\mathcal{E}^*$.

Theorem 5. *If* $\mathcal{R}_0 > 1$*, the global endemic equilibrium point* $\mathcal{E}^*$ *of the system (1) is globally asymptotically stable..*

Proof. When $\mathcal{R}_0 > 1$, one can define the following Lyapunov function as in [4, 23, 25, 26, 30] :

$$\begin{aligned} V = (S_1 - S_1^*) + (S_2 - S_2^*) + (S_3 - S_3^*) + (E_I - E_I^*) + (E_C - E_C^*) + (I - I^*) + \\ + (C - C^*) + (R - R^*) - (S_1^* + S_2^* + S_3^* + E_I^* + E_C^* + I^* + C^* + +R^*) \times \\ ln\left(\frac{S_1 + S_2 + S_3 + E_I + E_C + I + C + R}{S_1^* + S_2^* + S_3^* + E_I^* + E_C^* + I^* + C^* + R^*}\right) \end{aligned} \tag{25}$$

As $N = S_1 + S_2 + S_3 + E_I + E_C + I + C + R$, one can set

$$N^* = S_1^* + S_2^* + S_3^* + E_I^* + E_C^* + I^* + C^* + R^*.$$

Then, the Lyapunov function can also be rewriten as follows:

$$\begin{aligned} V &= N - N^* - N^* ln\frac{N}{N^*} \\ V &= N^*\left(\frac{N}{N^*} - 1 - ln\frac{N}{N^*}\right) \end{aligned} \tag{26}$$

We shall use the family of Volterra-type Lyapunov function defined by $g(x) = x - 1 - ln(x)$, $x \in \mathbf{R}^+$ which admits a global mimnimun at $x = 1$, and satisfies $g(1) = 0$. Since $S_1(t) > 0$, $S_2(t) > 0$, $S_3(t) > 0$, $E_I(t) > 0$, $E_C(t) > 0$, $I(t) > 0$, $C(t) > 0$, $R(t) > 0$, then one can obtain the following

$$V = N - N^* - N^* ln\frac{N}{N^*} \geq 0 \tag{27}$$

Therefore, the Lyapunov function V derivative is given by the following sense

$$\frac{dV}{dt} = \left(1 - \frac{N^*}{N}\right)\frac{dN}{dt} \tag{28}$$

Note that according to system (1)

$$\frac{dN}{dt} = \Lambda - \mu_I I - \mu_C C - \mu\, N \tag{29}$$

As at the endemic equilibrium point $\dfrac{dN}{dt} = 0$, then one obtains

$$\Lambda = \mu_I I^* + \mu_C C^* + \mu\, N^* \tag{30}$$

From (28), (29), (30), and by assuming that $N - N^* \geq 0$, $I - I^* \geq 0$, $C - C^* \geq 0$, one has

$$\begin{aligned} \frac{dV}{dt} &= \left(1 - \frac{N^*}{N}\right)(\mu_I I^* + \mu_C C^* + \mu\, N^* - \mu_I I - \mu_C C - \mu\, N) \\ \frac{dV}{dt} &= -\left(\frac{N - N^*}{N}\right)[\mu_a\,(I - I^*) + \mu_C\,(C - C^*) + \mu\,(N - N^*)] \\ \frac{dV}{dt} &\leq 0 \end{aligned} \tag{31}$$

From (31) and by using the fact that $\frac{dV}{dt} = 0$ if and only if $S_1 = S_1^*,\ S_2 = S_2^*,\ S_3 = S_3^*,$ $E_I = E_I^*,\ E_C = E_C^*,\ I = I^*,\ C = C^*,\ R = R^*$, then $\frac{dV}{dt}$ converges when $t \longrightarrow \infty$.
Thanks to LaSalle's invariance principle theorem [26], the endemic equilibrium point $\mathcal{E}^*$ is said to be globally asymptotically stable when $\mathcal{R}_0 > 1$ [4, 14, 18, 27]

6. Numerical results and discussion

For the numerical simulation of the proposed system, the Rung Kutta 4 method was used. In table 3 the values of the different parameters used in the model problem (1) are based on data from Burkina Faso and the literature. With these parameters, the basic reproduction number $\mathcal{R}_0 = 3.6625$. We set $N_0 = 20818036$: total number of Burkina Faso's population in 2020 [20]. Initial conditions were estimated using prevalence in 2020 and biological parameters. This gives

$$S_1(0) = 1191200,\ S_2(0) = 2384100,\ S_3(0) = 11938000,\ E_I(0) = 1551400,$$

$$E_C(0) = 172604,\ I(0) = 1665482,\ C(0) = 416354,\ R(0) = 1498896.$$

Table 3: **Parameters used in the model**

Parameters	Values	References
N_0	20818036	[20]
λ	0.0444	[20]
λ_i	0.091	Assumed
λ_c	0.0051	Assumed
p_1	0.9	Assumed
p_2	0.9	Assumed
$b_{1,j}, j = 1, 2$	0	[16]
$b_{2,j}, j = 1, 2$	0	[16]
$b_{3,j}, j = 1, 2$	0	[16]
$b_{1,j}, j = 3, 4$	0.159	[[24],[15]]
$b_{2,j}, i = 3, 4$	0.144	[[24],[15]]
$b_{3,j}, j = 3, 4$	0.5	Assumed
α_1	0.1	[[19],[17]]
α_2	0.7	[[19],[17]]
α_3	0.95	[[19],[17]]
δ_1	0.6	[[24],[15]]
δ_2	0.6	[[24],[15]]
γ_i	0.8	[[24],[15]]
γ_c	0.023	[[24],[15]]
μ	0.009	[20]
μ_i	0.00461	[[24],[15]]
μ_c	0.01	[[24],[15]]

We carried out simulations to see the dynamics of the hepatitis B virus in the population of Burkina Faso over a period of 50 years.
Figure 4 represents the evolution of the three susceptible compartments, figure 5 represents the evolution of infected and chronic exposed compartments, while figure 6 is the one of infected and chronic individuals. The last figure 7 is that of recovered individuals.

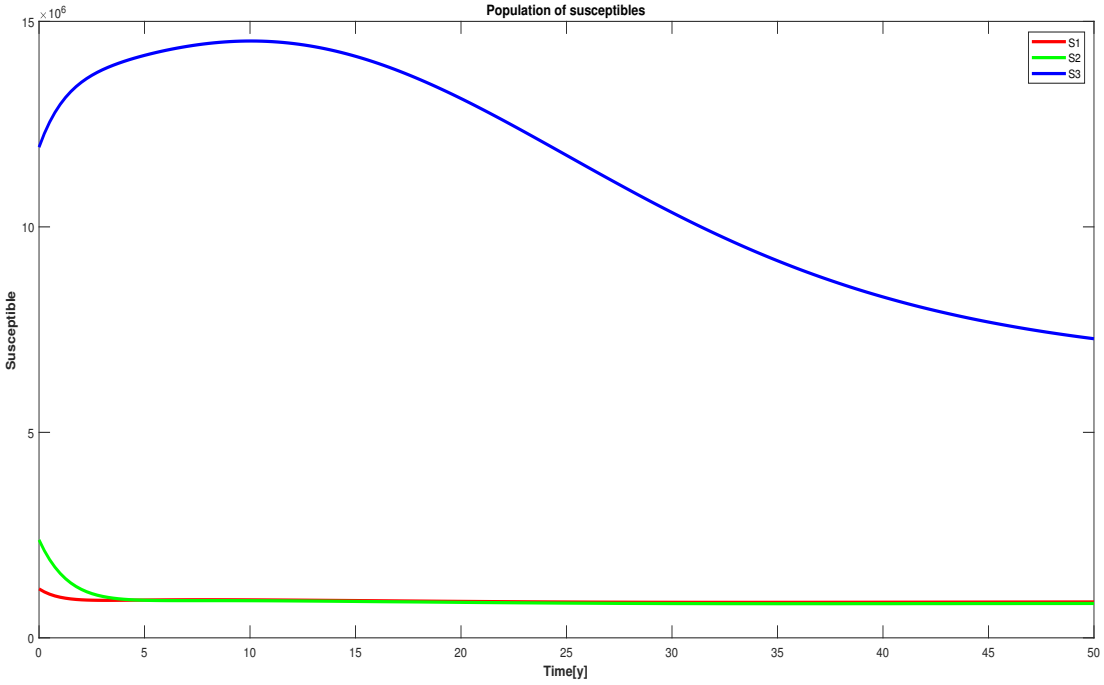

Figure 4: Evolution of susceptible individuals

In Figure 4, the number of all susceptible individuals is decreasing, confirming the high prevalence of HBV infection in Burkina Faso.

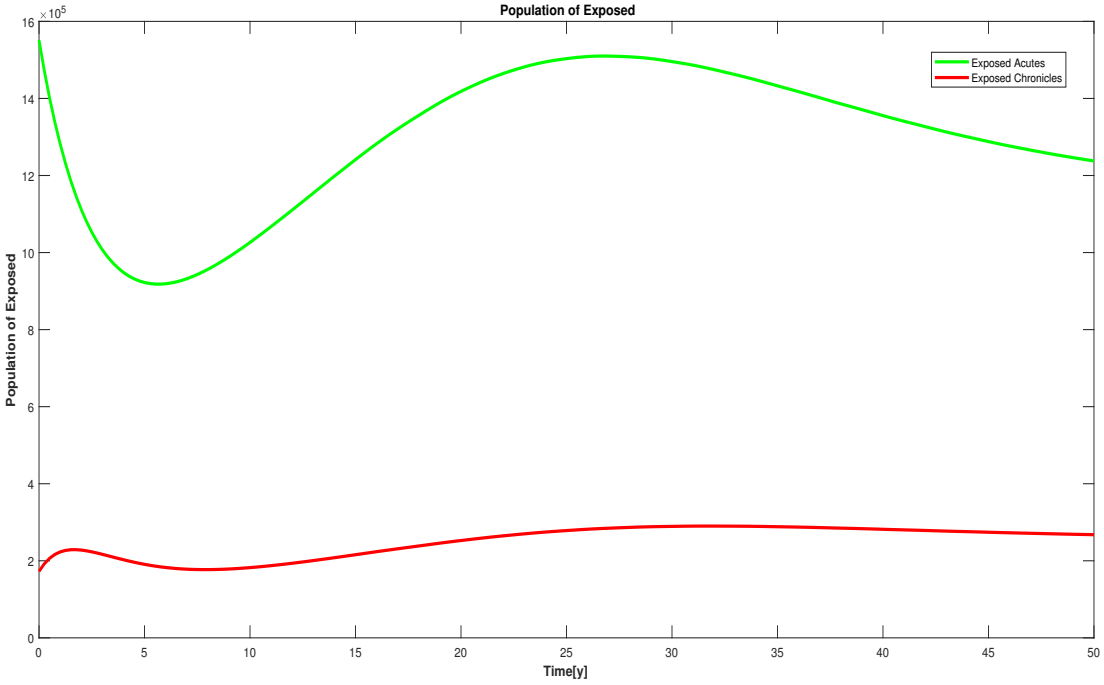

Figure 5: Evolution of acutes exposed together with chronic exposed individuals

According to the results presented in this figure, the number of cases in the acute and chronic infected exposed compartments (Figure 5) increases as they come from susceptible individuals, before decreasing to become acute and chronically infected.

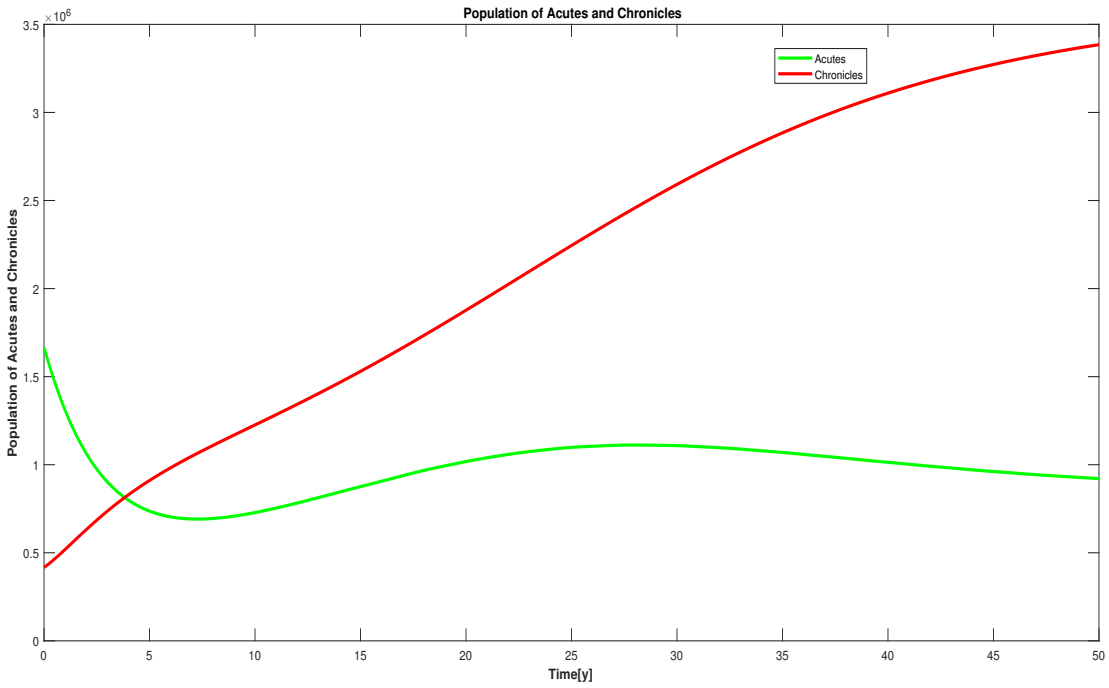

Figure 6: Evolution of acutes and chronics individuals

As shown in this figure, the number of people in the acutes and chronics compartments (6) increases, since we are in a zone of high endemicity and this confirms the high prevalence of heaptitis B in Burkina Faso [31]. The number of chronic carriers of the virus is rising sharply, while the number of acutely infected individuals is falling, as they either recover or become chronic.

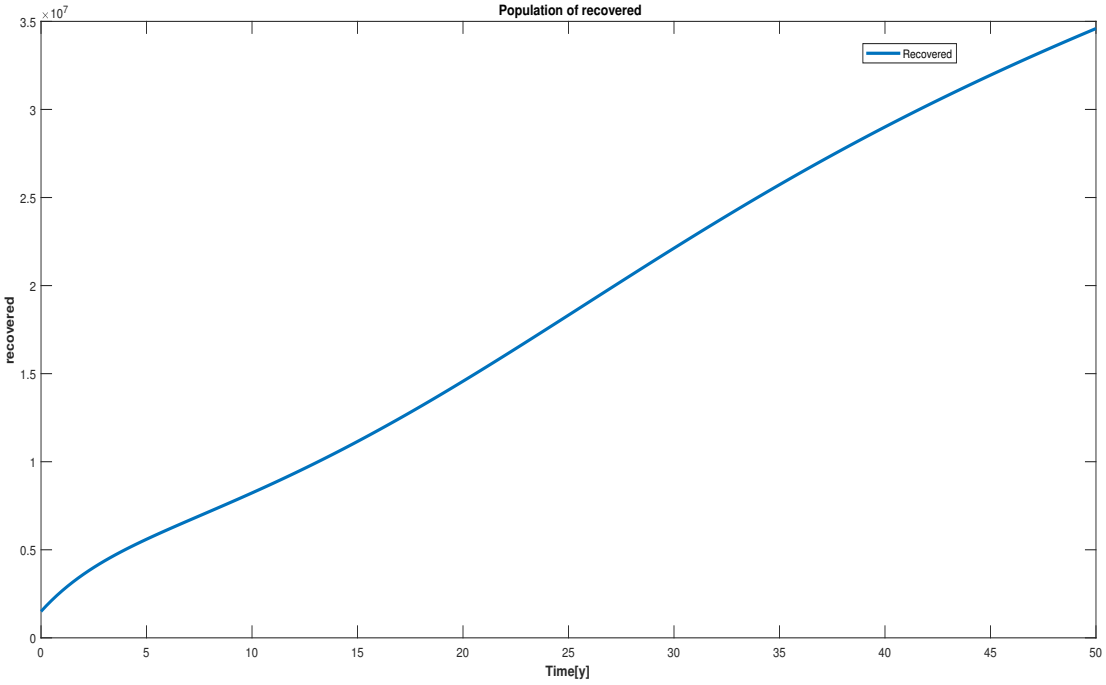

Figure 7: Evolution of recovered individuals

Here, the number of cured or recovered patients increases significantly (7), as $90-95\%$ of acutely infected patients recover, and 30% of those $(5-10\%)$ who become chronically infected are healthy carriers [35], and therefore recovered from the hepatitis B virus transmission process.

7. Conclusion

In this paper, a new differential mathematical model of susceptibility and infectivity of hepatitis B transmission was developed to simulate the potential spread of hepatitis B virus in the population of Burkina Faso. The global existence and uniqueness of the model's solutions have been proven. Mathematical analysis of the model shows that disease progression is governed by the basic reproduction number $\mathcal{R}_0$, which is a key concept in epidemiology. When $\mathcal{R}_0 < 1$, the disease disappears from the population, resulting in a disease-free equilibrium that has been proven to be globally asymptotically stable. On the other hand, when $\mathcal{R}_0 > 1$, the disease persists, leading to an endemic state that is also globally asymptotically stable.

The numerical simulation of the model used to predict the spread of infection in the population showed that :

- $\mathcal{R}_0 = 3.6625 > 1$, which proves that the disease is persisting in Burkina Faso;
- the number of susceptible people falls sharply as they become infected due to the persistence of the disease;
- the number of chronic hepatitis B virus carriers increases gradually over the fifty-year simulation period.

These numerical results confirm the high endemicity of hepatitis B viral infection in Burkina Faso, which is why this pathology remains a public health problem for the country.

In perspective, we intend to improve this model by taking into account vaccination, treatment and awareness, in order to assess the impact of the strategies implemented in Burkina Faso to combat the spread of the virus.

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A. Appendix

```
% hepatis.m
function dtdy = hepatis(~,u)
global N l l1 l2 r mu mu1 mu2   a1 a2 a3 p1 p2
global b11  b12  b13  b14  b21 b22 b23 b24  b31  b32
   b33  b34
global    d1 d2   g2 g3
dtdy = zeros(8,1);
dS1 = l -l1*u(6)-l2*u(7) -(mu+p1)*u(1) -b11*u(1)*u(4)
    -b12*u(5)*u(1)-b13*u(6)*u(1)-b14*u(7)*u(1);
dS2 = p1*u(1) -(mu+p2)*u(2) -b21*u(4)*u(2)- b22*u(5)*
   u(2)-b23*u(6)*u(2) -b24*u(7)*u(2);
dS3 = p2*u(2) -b31*u(4)*u(3) -b32*u(3)*u(5)  -b33*u
   (3)*u(6)-b34*u(3)*u(7) -(mu)*u(3);
```

```
dEI = a1*b11*u(1)*u(4) +a1*b12*u(1)*u(5) +a1*b13*u(1)
   *u(6)+a1*b14*u(1)*u(7) ...
+ a2*b21*u(2)*u(4)+a2*b22*u(2)*u(5)+a2*b23*u(2)*u(6)+
   a2*b24*u(2)*u(7)  ...
+ a3*b31*u(3)*u(4)+a3*b32*u(3)*u(5)+a3*b33*u(3)*u(6)+
   a3*b34*u(3)*u(7)-(mu+d1)*u(4) + r*(l1*u(6)+l2*u(7)
   );
dEC = (1-a1)*b11*u(1)*u(4) +(1-a1)*b12*u(1)*u(5) +(1-
   a1)*b13*u(1)*u(6)+(1-a1)*b14*u(1)*u(7) ...
+ (1-a2)*b21*u(2)*u(4)+(1-a2)*b22*u(2)*u(5)+(1-a2)*
   b23*u(2)*u(6)+(1-a2)*b24*u(2)*u(7) ...
+ (1-a3)*b31*u(3)*u(4)+(1-a3)*b32*u(3)*u(5)+(1-a3)*
   b33*u(3)*u(6)+(1-a3)*b34*u(3)*u(7) +(1-r)*(l1*u(6)
    + l2*u(7))- (mu+d2)*u(5);
dI =d1*u(4) - (mu+g2+mu1)*u(6);
dC = d2*u(5) -(mu+g3+mu2)*u(7);
dR =g2*u(6) +g3*u(7)- (mu)*u(8);
dtdy=[dS1;dS2;dS3;dEI;dEC;dI;dC;dR];

% main.m
global N l l1 l2 r mu mu1 mu2  mu3 a1 a2 a3 p1 p2
global b11  b12  b13  b14  b21 b22 b23 b24  b31  b32
   b33  b34
global  d1 d2  g2 g3

N=20818036;
l=0.0444;
l1= 0.091;
l2=0.0051;
r=0.1;
[mu, mu1, mu2, mu3]= deal(0.009, 0.00461, 0.01,
   0.005);
[a1,a2,a3]= deal(0.1, 0.7, 0.95);
[p1, p2]= deal(0.9 , 0.9);
[b11 , b12 , b13 , b14]=deal(0.0, 0.0 , 0.159, 0.159)
   ;
[b21 , b22 , b23 , b24]=deal(0.0, 0.0 , 0.144, 0.144)
   ;
[b31 , b32 , b33 , b34]= deal(0.0, 0.0 , 0.5, 0.5);
[d1, d2]=deal(0.6, 0.6);
[g2, g3] =deal(0.8, 0.023);
y0=[1191200,2384100,11938000,1551400,172604,
1665482,416354,1498896]/N;
```

```
M=200;
tspan=linspace(0,50,M);
[t,y1]= ode45(@hepatis,tspan,y0);
figure(1)
plot(t,N*y1(:,1),'r',t,N*y1(:,2),'g',t,N*y1(:,3),'b',
   'Linewidth', 2.5);legend('S1','S2','S3');title('\
   bf Population of susceptibles '); xlabel('\bf{Time
   [y]}');ylabel('\bf{Susceptible}');
figure(2)
plot(t,N*y1(:,4),'g',t,N*y1(:,5),'r','Linewidth',
   2.5);legend('Exposed Acutes','Exposed Chronicles')
   ;title('\bf Population of Exposed');xlabel('\bf{
   Time[y]}');ylabel('\bf{Population of Exposed}');
figure(3)
plot(t,N*y1(:,6),'g',t,N*y1(:,7),'r','Linewidth',
   2.5);legend('Acutes','Chronicles');title('\bf
   Population of Acutes and Chronicles');xlabel('\bf{
   Time[y]}');ylabel('\bf{Population of Acutes and
   Chronicles}');
figure(4)
plot(t,N*y1(:,8),'Linewidth', 2.5);legend('Recovered'
   );title('\bf Population of recovered');xlabel('\bf
   {Time[y]}');ylabel('\bf{recovered}');
```