ARAB JOURNAL OF BASIC AND APPLIED SCIENCES
2021, VOL. 28, NO. 1, 87–106
https://doi.org/10.1080/25765299.2021.1896423

University of Bahrain
Taylor & Francis
Taylor & Francis Group

ORIGINAL ARTICLE

A fractional order model for the transmission dynamics of hepatitis B virus with two-age structure in the presence of vaccination

Getachew Teshome Tilahun[a], Woldegebriel Assefa Woldegerima[b,c] and Nesredin Mohammed[a]

[a]Department of Mathematics, CNCS, Haramaya University, Haramaya, Ethiopia; [b]Department of Mathematics and Applied Mathematics, University of Pretoria, Pretoria, South Africa; [c]Department of Mathematics, CNCS, Mekelle University, Mekelle, Ethiopia

ABSTRACT

In this study, we proposed a fractional order model of hepatitis B virus transmission dynamics with two-age structure under vaccination. A qualitative analysis of the model is performed. Basic reproduction number of the model is determined. Local stability conditions of disease-free equilibrium point are proven by using fractional Ruth-Hurwitz conditions. Global stability conditions of both disease-free and endemic equilibrium points are shown by constructing appropriate Lyapunov functions. Sensitivity analysis was done by using normalized forward sensitivity index approach. Numerical simulation is performed to investigate the effect of memory on hepatitis B disease dynamics by varying order of derivatives and to simulate the effects of vaccinating newborns immediately after birth, vaccinating children and adult vaccination. Then, we compared their effects on hepatitis B disease dynamics in the sense of control and elimination. It is observed that the number of infective individuals decreases faster and even falls to zero over a long run for the model with memory than memory-less model. Comparing results between vaccination of different ages show that increasing newborn vaccination immediately after birth has the highest effect on hepatitis B disease control.

ARTICLE HISTORY
Received 3 October 2020
Revised 23 January 2021
Accepted 20 February 2021

KEYWORDS
Hepatitis B virus; memory; fractional order; age structure; mathematical model; vaccination; basic reproduction number

1. Introduction

Hepatitis is an inflammation of liver and mostly caused by viruses (Moorman et al., 2015; Rehermann & Nascimbeni, 2005). Hepatitis viral infection is a serious public health problem worldwide. Hepatitis B (HB) disease is liver infection caused by hepatitis virus called hepatitis B virus (HBV). It can be acute and resolve without treatment. However, some forms can cause chronic hepatitis, meaning it lasts more than six months. Having chronic hepatitis B increases your risk of developing liver failure, liver cancer or cirrhosis, (Rehermann & Nascimbeni, 2005; World Health Organization, 2020). Hepatitis B virus is one of the most serious and prevalent health problems, affecting more than 2 billion people worldwide. Although highly effective vaccines against hepatitis B virus have been available since 1982, there are still more than 350 million chronic carriers, 75% of whom reside in the Asia Pacific region (Liaw & Chu, 2009). It is estimated that 1.4 million deaths per year are caused by viral hepatitis, which is higher than death caused by HIV and compared with death caused by tuberculosis. Of those deaths approximately 47% are caused by HBV (Rehermann & Nascimbeni, 2005; World Health Organization and others, 2017). It is estimated that 240 million people are chronically infected with hepatitis B. The largest number of people with chronic HBV lives in the African region (over 75 million) next to the Western Pacific region (over 95 million) (Umare, Seyoum, Gobena, & Haile Mariyam, 2016).

HBV infection can be transmitted through vertical transmission (from mother to child at birth), horizontal transmission (exposure to infected blood) and sexual transmission (unprotected sex), and by using needles with infected persons or inapparent percutaneous or permucosal exposure to infected blood or other body fluids (Liaw & Chu, 2009; World Health Organization and others, 2012). Although symptoms not appear on most people when newly infected, some people have acute illness with symptoms, including yellowing of the skin and eyes (jaundice), dark urine, extreme fatigue, nausea, vomiting and abdominal pain. HB infection becomes chronic depends on the age at which a person infected. 80%−90% of infants infected during the first year of life, 30%−50% of children infected before the age of 6 years and less than 5% of healthy persons who are

CONTACT Getachew Teshome Tilahun gmgech@gmail.com Department of Mathematics, CNCS, Haramaya University, Haramaya, Ethiopia.

infected as adults will develop chronic infections (Dan, Moses-Eisenstein, & Valdiserri, 2015).

Vaccination of susceptible individuals especially newborns is the most important way to reduce the incidence of HBV infection (Lai, Ratziu, Yuen, & Poynard, 2003; Liang, Zu, & Zhuang, 2018). Despite the success availability of vaccine, problem of HBV infection still remains. Therefore, it is important to predict the future trends in HBV prevalence and provide full information for public health. One of the possible methods to predict the prevalence of infectious disease is to use mathematical model (Liang et al., 2018). As it is directly mentioned in Liaw and Chu (2009) since the introduction of the hepatitis B vaccine and other preventive measures, the worldwide prevalence of hepatitis B infection has fallen. However, chronic infection remains a challenging global health problem. Thus, an improved understanding of hepatitis B virology, immunology, and the natural course of chronic infection is important.

Mathematical model can be helpful in both performing qualitative and quantitative evaluation, forecasting for both injecting drug use and epidemiological information on the incidence of and prevalence of infection, and evaluate the impact of interventions aimed at secondary prevention or harm reduction (Thomson, 2005). There are several types of mathematical models have been used to describe the spread of disease including hepatitis B. These are the deterministic models, stochastic models, fractional order models, etc.

A system of ordinary differential equations is the most used mathematical expression in modelling the transmission dynamics of hepatitis B (Liang et al., 2018). When ordinary differential equation is used the state of the systems at each time does not depend on the previous history of the systems. It is memory-less. However, the evolution and control of epidemic processes in human population can not be considered without memory effect (Pimenov et al., 2012). If people know the history of particular disease in their region, they use different preventive measures, such as isolation of infected individuals and vaccination, when possible (Saeedian, Khalighi, Azimi-Tafreshi, Jafari, & Ausloos, 2017). Since memory indicates the dependence of the system not only on the current state of the system but also on previous history of the system, it enables the prediction of the future on the basis of experience (Kumar, Ghosh, Samet, & Goufo, 2020; Pimenov et al., 2012; Tarasov, 2018). Fractional calculus is a powerfull tool to describe the effect of memory. Fractional derivatives and non-integer orders can be used to describe systems with memory. Due to its memory properties, fractional differential equations have been used in different areas such as physics, engineering, biological systems and financial systems (Pimenov et al., 2012; Saeedian et al., 2017; Tarasov, 2018).

In the modelling of hepatitis B diseases, one of the most important characteristics is considering age (Liang et al., 2018). In the last 10 years, many researchers have been using mathematical model to study the dynamics of HBV transmission. For example, the authors (Adu, Aidoo, Darko, & Osei-Frimpong, 2014; Akbari, Kamyad, & Heydari, 2016; Aniji, Kavitha, & Balamuralitharan, 2019; Kamyad et al., 2014; 2014; Khan & Zaman, 2016; Kimbir, Aboiyar, Abu, & Onah, 2014; Nana-Kyere, Ackora-Prah, Okyere, Marmah, & Afram, 2017; Nayeem & Podder, 2014; Seyoum Desta & Koya, 2019; Shaban & Manoza, 2016; Shi, Lu, & Wang, 2019; Wiah, Makinde, & Adetunde, 2015; Zhang, Guo, & Smith, 2018; Zou, Zhang, & Ruan, 2010) propose mathematical model of HBV transmission dynamics. However, most these works did not consider both the effect of memory using fractional order derivatives and age structure model simultaneously. It is widely accepted that the fractional order models have played substantial role in the enhancement of results associated with the existing mathematical model based on classical system of ordinary differential equations. A large number of researchers have modelled many real processes with the aid of fractional calculus. Due to the applicability of differential equations with fractional order in science and engineering, more applied researchers have been attracted to it (Carvalho, Pinto, & Baleanu, 2018; Ghanbari, Kumar, & Kumar, 2020; Kazem, Abbasbandy, & Kumar, 2013; Khan, Hammouch, & Baleanu, 2019; Khodabakhshi, Vaezpour, & Baleanu, 2017; Kumar, 2014; Kumar, Ghosh, et al., 2020; Kumar, Kumar, & Baleanu, 2016; Qureshi & Atangana, 2019; Rashidi, Hosseini, Pop, Kumar, & Freidoonimehr, 2014), and the references there in, just to mention a few. Generally speaking, fractional calculus is a generalization of classical differentiation and integration to arbitrary (non-integer) order.

Zhang, Wang, and Zhang, (2015) consider age structure model with out memory effect. Wiah et al. (2015) and Shi et al. (2019) consider memory effect but they did not consider age structure. To our knowledge, no works have been done to model the transmission dynamics of hepatitis B virus infection by considering both age structure and effect of memory. Thus, our main contribution is to formulate a mathematical model that considers both memory effect (fractional order derivatives) and structure at the same time. Web has also performed stability analysis, sensitivity analysis and numerical simulations and such model is used to investigate the effect of varying parameters related to the memory and age structure. Motivated by this, in this paper we

proposed a compartmental model of HBV transmission dynamics with two age structures by including memory effect. Fractional calculus involves various types of fractional order derivatives such as Riemann-Liouville (RL), Caputo (C), Hamdard (H), Caputo Febrizo (CF), Atangana-Baleanu (AB) and Atangana-Baleanu-Caputo (ABC). The three most commonly used fractional order differential operators, especially in modelling, are the Caputo, the Caputo-Fabrizio and the Atangana-Baleanu-Caputo operators (Qureshi & Atangana, 2019). However, in this paper, we use the fractional order derivative in the sense of the Capito. More details and definition of these fractional order derivatives can be obtained (Podlubny, 1998; Qureshi & Atangana, 2019) and the references therein.

The rest of the paper is arranged as follows: in Section 2, we formulate age-structured compartmental model represented by system of ordinary differential equations and we extend to fractional order model represented by system of fractional differential equations. In Section 3, qualitative analysis of fractional order model is analysed. In Section 4, we perform sensitivity analysis of parameters. In Section 5, we present numerical simulation. Summary and conclusions of our study are presented in Section 6.

2. Model description

We divided the total population into eight classes where S_c represent susceptible children (children not infected with hepatitis B virus (HBV) and not vaccinated), R_c children vaccinated immediately after birth, I_c children infected with HBV infection at birth, S_a susceptible adult (uninfected adult), E_a infected adult with no symptoms (in latent incubation period), I_a acutely infected adult with symptoms, C_a chronically infected adults and R_a recovered adults. New born to infected mother become infected with $(1-k)$ probability and those children infected at birth does not go through the acute phase. Newborn vaccinated immediately after birth join R_c with (ω) being effectiveness of vaccine. Susceptible children S_c join recovered children R_c with (v) rate when they got vaccine. Susceptible adult become infected with HB infection through horizontal transmission (any means of blood to blood contact with infected classes) with (β) rate of transmission and join exposed adult E_a. Exposed adult E_a join acutely infected adult I_a with (σ) rate. Individuals in acutely infected adult I_a naturally recovered and join recovered children R_a with (γ) rate and also join chronically infected adults C_a with (θ) rate.

The model is based on the following main assumptions:

- The death rate is assumed to be constant for all classes, and the total death is balanced by total birth (the population constant).
- We assume that latently infected (E_a), acutely infected (I_a) and chronically infected (C_a) by horizontal transmission.
- Children born from exposed, infected and chronically infected mothers can be infected at birth and newborn infected at birth does not go through the acute phase.
- The susceptible who vaccinated become recovered and got permanent immunity.
- Individuals in acutely infected adults naturally recovered and not infected again.

Children infected with HB infection at birth are asymptomatic and do not go through the acute phase of HB infection and they progress to chronic phase of HB infection after 20–30 years (Goyal & Murray, 2014). For simplicity, we assume that children infected at birth progress to chronic phase after approximately 14 years. We consider two classes, children (aged$\leq$ 14 years) and adult (aged $>$14 years). The model parameters are defined in Table 1.

We present all information about the model description in the flow diagram given in Figure 1 below.

From the above flow diagram, the model without memory can be represented by the following system of nonlinear ordinary differential equations

$$\begin{cases} \frac{dS_a}{dt} = mS_c - \beta S_a(E_a + I_a + C_a) - (p+d)S_a, \\ \frac{dS_c}{dt} = b(1-\omega)(S_a + R_a + k(E_a + I_a + C_a)) - (v+m+d)S_c, \\ \frac{dR_c}{dt} = b\omega(S_a + R_a + E_a + I_a + C_a) + vS_c - (m+d)R_c, \\ \frac{dI_c}{dt} = b(1-\omega)(1-k)(E_a + I_a + C_a) - (m+d)I_c, \\ \frac{dE_a}{dt} = \beta S_a(E_a + I_a + C_a) - (\sigma+d)E_a, \\ \frac{dI_a}{dt} = \sigma E_a - (\gamma+\theta+d)I_a, \\ \frac{dC_a}{dt} = \theta I_a + mI_c - (d+\delta)C_a, \\ \frac{dR_a}{dt} = mR_c + pS_a + \gamma I_a - dR_a, \end{cases} \tag{2.1}$$

where the total population at any time $t \geq 0$ is given by, $N(t) = S_a(t) + E_a(t) + I_a(t) + C_a(t) + R_a(t) + S_c(t) + R_c(t) + I_c(t)$ and given initial conditions are $S_a(0) = S_{a0}, S_c(0) = S_{c0}, R_c(0) = R_{c0}, I_c(0) = I_{c0}, E_a(0) = E_{a0}, I_a(0) = I_{a0}, C_a(0) = C_{a0}$, and $R_a(0) = R_{a0}$.

To include the effect of memory we rewrite systems of ordinary differential equations (2.1) using time-dependent integrals as given below,

$$\begin{cases} \frac{dS_a(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(mS_c(x) - \beta S_a(x)(E_a(x) + I_a(x) + C_a(x)) - (p+d)S_a(x))dx, \\ \frac{dS_c(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(b(1-\omega)(S_a(x) + R_a(x) + k(E_a(x) + I_a(x) + C_a(x))) - (v+m+d)S_c(x))dx, \\ \frac{dR_c(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(b\omega(S_a(x) + R_a(x) + E_a(x) + I_a(x) + C_a(x)) + vS_c(x) - (m+d)R_c(x))dx, \\ \frac{dI_c(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(b(1-\omega)(1-k)(E_a(x) + I_a(x) + C_a(x)) - (m+d)I_c(x))dx, \\ \frac{dE_a(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(\beta S_a(x)(E_a(x) + I_a(x) + C_a(x)) - (\sigma+d)E_a(x))dx, \\ \frac{dI_a(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(\sigma E_a(x) - (\gamma+\theta+d)I_a(x))dx, \\ \frac{dC_a(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(\theta I_a(x) + mI_c(x) - (d+\delta)C_a(x))dx, \\ \frac{dR_a(t)}{dt} = \int_{t_0}^{t} \delta_1(t-x)(mR_c(x) + pS_a(x) + \gamma I_a(x) - dR_a(x))dx, \end{cases} \tag{2.2}$$

where $\delta_1(t-x)$ used as time-dependent kernel defined by the following power law correlation function.

$$\delta_1(t-x) = \frac{1}{\Gamma(\alpha-1)}(t-x)^{\alpha-2}, \tag{2.3}$$

where $0 < \alpha \leq 1$ and $\Gamma(x)$ represents Gamma function.

If we substitute (2.3) into (2.2), then (2.2) becomes

$$\begin{cases} \frac{dS_a(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(mS_c(x) - \beta S_a(x)(E_a(x) + I_a(x) + C_a(x)) - (p+d)S_a(x))dx, \\ \frac{dS_c(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(b(1-\omega)(S_a(x) + R_a(x) + k(E_a(x) + I_a(x) + C_a(x))) - (v+m+d)S_c(x))dx, \\ \frac{dR_c(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(b\omega(S_a(x) + R_a(x) + E_a(x) + I_a(x) + C_a(x)) + vS_c(x) - (m+d)R_c(x))dx, \\ \frac{dI_c(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(b(1-\omega)(1-k)(E_a(x) + I_a(x) + C_a(x)) - (m+d)I_c(x))dx, \\ \frac{dE_a(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(\beta S_a(x)(E_a(x) + I_a(x) + C_a(x)) - (\sigma+d)E_a(x))dx, \\ \frac{dI_a(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(\sigma E_a(x) - (\gamma+\theta+d)I_a(x))dx, \\ \frac{dC_a(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(\theta I_a(x) + mI_c(x) - (d+\delta)C_a(x))dx, \\ \frac{dR_a(t)}{dt} = \frac{1}{\Gamma(\alpha-1)}\int_{t_0}^{t} (t-x)^{\alpha-2}(mR_c(x) + pS_a(x) + \gamma I_a(x) - dR_a(x))dx. \end{cases} \tag{2.4}$$

The right-hand side of system of Equation (2.4) is fractional integral of order $(\alpha-1)$, $0 \leq \alpha \leq 1$ on the interval $[t_0, t]$, which is represented by ${}_{t_0}D_t^{-(\alpha-1)}$.

Since fractional derivatives are left inverse of fractional integrals, we apply Caputo fractional derivatives of order $(\alpha-1)$ on both sides of Equation (2.4). Then, the following system of fractional differential equations in Caputo is sense obtained.

$$\begin{cases} D^{\alpha}S_a = mS_c - \beta S_a(E_a + I_a + C_a) - (p+d)S_a, \\ D^{\alpha}S_c = b(1-\omega)(S_a + R_a + k(E_a + I_a + C_a)) - (v+m+d)S_c, \\ D^{\alpha}R_c = b\omega(S_a + R_a + E_a + I_a + C_a) + vS_c - (m+d)R_c, \\ D^{\alpha}I_c = b(1-\omega)(1-k)(E_a + I_a + C_a) - (m+d)I_c, \\ D^{\alpha}E_a = \beta S_a(E_a + I_a + C_a) - (\sigma+d)E_a, \\ D^{\alpha}I_a = \sigma E_a - (\gamma+\theta+d)I_a, \\ D^{\alpha}C_a = \theta I_a + mI_c - (d+\delta)C_a, \\ D^{\alpha}R_a = mR_c + pS_a + \gamma I_a - dR_a. \end{cases} \tag{2.5}$$

Since the total population $N(t) = S_a(t) + E_a(t) + I_a(t) + C_a(t) + R_a(t) + S_c(t) + R_c(t) + I_c(t)$, we normalize by dividing both sides by (N(t)) as $1 = S_a + E_a + I_a + C_a + R_a + S_c + R_c + I_c$, where $S_a, E_a, I_a, C_a, R_a, S_c, R_c, I_c$ are fraction of each classes in the total population.

Since, $R_a = 1-(S_a + E_a + I_a + C_a + S_c + R_c + I_c)$, we omit the last equation of (2.5) and replace for R_a in the second and third equations of (2.5), then we obtain

$$\begin{cases} D^{\alpha}S_a = mS_c - \beta S_a(E_a + I_a + C_a) - (p+d)S_a, \\ D^{\alpha}S_c = b(1-\omega) - b(1-\omega)((1-k)(E_a + I_a + C_a) + S_c + R_c + I_c) - (v+m+d)S_c, \\ D^{\alpha}R_c = b\omega - b\omega(S_c + R_c + I_c) + vS_c - (m+d)R_c, \\ D^{\alpha}I_c = b(1-\omega)(1-k)(E_a + I_a + C_a) - (m+d)I_c, \\ D^{\alpha}E_a = \beta S_a(E_a + I_a + C_a) - (\sigma+d)E_a, \\ D^{\alpha}I_a = \sigma E_a - (\gamma + \theta + d)I_a, \\ D^{\alpha}C_a = \theta I_a + mI_c - (d+\delta)C_a, \end{cases} \tag{2.6}$$

where $D = \frac{d}{dt}$ and D^{α} represents Caputo fractional derivatives of order $0 < \alpha \leq 1$. $S_a(0) = S_{a0} \geq 0, S_c(0) = S_{c0} \geq 0, R_c(0) = R_{c0} \geq 0, I_c(0) = I_{c0} \geq 0, E_a(0) = E_{a0} \geq 0, I_a(0) = I_{a0} \geq 0,$ and $C_a(0) = C_{a0} \geq 0$ are given initial conditions.

3. Model Analysis

3.1. Basic mathematical properties of the model

Here, we show the solution to model Equation (2.6) is non-negative. Since we are dealing with the number of population which cannot be negative number, we show there is non-negative solution to our model.

Theorem 3.1 (Non-negativity of the solution). *Solution to our fractional order model system (2.6) is non-negative, where, $S_{a0} \geq 0, S_{c0} \geq 0, R_{c0} \geq o, I_{c0} \geq 0, E_{a0} \geq 0, I_{a0} \geq 0,$ and $C_{a0} \geq 0$.*

Proof. We prove non-negativity of the solution to system of Equation (2.6) as follows

Let us take the first equation of (2.6)

$$\begin{aligned} &D^{\alpha}S_a = mS_c - \beta S_a(E_a + I_a + C_a) - (p+d)S_a \\ &\Rightarrow D^{\alpha}S_a \geq -\beta S_a(E_a + I_a + C_a) - (p+d)S_a \\ &\Rightarrow D^{\alpha}S_a \geq -(\beta h_1 + p + d)S_a, \end{aligned} \tag{3.1}$$

where $h_1 = \max\{E_a + I_a + C_a\}$.

By taking the Laplace transform of (3.1), we get

$$\begin{aligned} &L\{D^{\alpha}S_a\} \geq L\{-(\beta h_1 + p + d)S_a\} \\ &\Rightarrow s^{\alpha}L\{S_a\} - s^{\alpha-1}S_{a0} \geq -(\beta h_1 + p + d)L\{S_a\} \\ &\Rightarrow (s^{\alpha} + (\beta h_1 + p + d))L\{S_a\} \geq s^{\alpha-1}S_{a0} \\ &\Rightarrow L\{S_a\} \geq \frac{s^{\alpha-1}S_{a0}}{s^{\alpha} + \beta h_1 + p + d}. \end{aligned} \tag{3.2}$$

By applying the inverse Laplace transform to (3.2), we obtain

$$S_a \geq L^{-1}\left\{\frac{s^{\alpha-1}S_{a0}}{s^{\alpha} + \beta h_1 + p + d}\right\}. \Rightarrow S_a \geq S_{a0}E_{\alpha,1}(-(\beta h_1 + p + d)t^{\alpha}).$$

Since $S_{a0} \geq 0$ and $0 \leq E_{\alpha,1}(-(\beta h_1 + p + d)t^{\alpha}) \leq 1$, $S_a \geq 0$, then $S_a(t) \geq 0, \quad \forall t \geq 0$.

Let us take the second equation of (2.6): $D^{\alpha}S_c = b(1-\omega) - b(1-\omega)((1-k)(E_a + I_a + C_a) + S_c + R_c + I_c) - (v+m+d)S_c$. When $\omega = 1$, we have

$$D^{\alpha}S_c \geq -(v+m+d)S_c. \tag{3.3}$$

Taking Laplace transform of (3.3), we get

$$L\{S_c\} \geq \frac{s^{\alpha-1}S_{c0}}{s^{\alpha} + v + m + d}. \tag{3.4}$$

Applying the inverse Laplace transform to (3.4), we obtain

$$S_c \geq S_{c0}E_{\alpha,1}(-(v+m+d)t^{\alpha}).$$

Since, $S_{c0} \geq 0$, and $0 \leq E_{\alpha,1}(-(v+m+d)t^{\alpha}) \leq 1$, we have $S_c \geq 0$

Let us take third equations of (2.6)

$$D^{\alpha}R_c = b\omega - b\omega(S_c + R_c + I_c) + vS_c - (m+d)R_c \tag{3.5}$$

$$\Rightarrow D^{\alpha}R_c \geq b\omega - b\omega(S_c + R_c + I_c) - (m+d)R_c. \tag{3.6}$$

When $\omega = 0$, we have

$$D^{\alpha}R_c \geq -(m+d)R_c. \tag{3.7}$$

Taking Laplace transform of (3.7), we get

$$L\{R_c\} \geq \frac{s^{\alpha-1}R_{c0}}{(s^{\alpha} + m + d)}. \tag{3.8}$$

By taking inverse Laplace transform of (3.8), we obtain

$$R_c \geq R_{c0}E_{\alpha,1}(-(m+d)t^{\alpha}) \geq 0.$$

Let us take fourth equation of (2.6)

$$\begin{aligned} &D^{\alpha}I_c = b(1-\omega)(1-k)(E_a + I_a + C_a) - (m+d)I_c \\ &\Rightarrow D^{\alpha}I_c \geq -(m+d)I_c. \end{aligned} \tag{3.9}$$

By taking Laplace transform of (3.9), we obtain

$$L\{I_c\} \geq \frac{s^{\alpha-1}I_{c0}}{s^{\alpha}+m+d},$$

by applying inverse Laplace transform we get

$$I_c \geq I_{c0}E_{\alpha,1}(-(m+d)t^{\alpha}) \geq 0.$$

Let us take the fifth equation of (2.6)

$$\begin{aligned} &D^{\alpha}E_a = \beta S_a(E_a + I_a + C_a) - (\sigma+d)E_a \\ &\Rightarrow D^{\alpha}E_a \geq -(\sigma+d)E_a. \end{aligned} \tag{3.10}$$

Solving (3.10) by method of Laplace transform, we obtain

$$E_a \geq E_{a0}E_{\alpha,1}(-(\sigma+d)t^{\alpha}) \geq 0.$$

Let us take sixth equation of (2.6)

$$D^{\alpha}I_a = \sigma E_a - (\gamma + \theta + d)I_a,$$

$$\Rightarrow D^{\alpha}I_a \geq -(\gamma+\theta+d)I_a. \tag{3.11}$$

When solving (3.11) by method of Laplace transform we obtain

$$I_a \geq I_{a0}E_{\alpha,1}(-(\gamma+\theta+d)t^{\alpha}) \geq 0.$$

Let us take seventh equation of (2.6)

$$D^{\alpha}C_a = \theta I_a + mI_c - (d+\delta)C_a.$$

So,

$$\Rightarrow D^{\alpha}C_a \geq -(d+\delta)C_a. \tag{3.12}$$

Solving (3.12) by method of inverse of Laplace transform, we obtain

$$C_a \geq C_{a0}E_{\alpha,1}(-(\delta+d)t^{\alpha}) \geq 0.$$

Therefore, solution to system of fractional differential equation (2.6) is non-negative provided that initial data are non-negative.

Theorem 3.2 (Positively Invariant Region). *The region* $\Omega = \{(S_a, S_c, R_c, I_c, E_a, I_a, C_a) \in R_+^7 | 0 \leq S_c + R_c + I_c + S_a + E_a + I_a + C_a \leq 1\}$ *is positively invariant region given that the initial data is non-negative.*

Proof. Suppose $0 \leq S_{c0} + R_{c0} + I_{c0} + S_{a0} + E_{a0} + I_{a0} + C_{a0} \leq 1$. Then adding all equations of (2.6), we get

$$\begin{aligned} &D^{\alpha}(S_c + R_c + I_c + S_a + E_a + I_a + C_a) \\ &= b - d(S_c + R_c + I_c + S_a + E_a + I_a + C_a) \\ &\quad - b(S_c + R_c + I_c) - mR_c - pS_a - \gamma I_a - \delta C_a, \\ &\Rightarrow D^{\alpha}(S_c + R_c + I_c + S_a + E_a + I_a + C_a) \\ &\leq b - d(S_c + R_c + I_c + S_a + E_a + I_a + C_a) \end{aligned}$$

Let $Z = (S_c + R_c + I_c + S_a + E_a + I_a + C_a)$., then

$$\begin{aligned} &D^{\alpha}Z \leq b - dZ \Rightarrow L\{D^{\alpha}Z\} \leq L\{b - dZ\}, \\ &\Rightarrow s^{\alpha}L\{Z\} - s^{\alpha-1}Z_0 \leq bs^{-1} - dL\{Z\} \\ &\Rightarrow (s^{\alpha}+d)L\{Z\} \leq bs^{-1} + s^{\alpha-1}Z_0, \\ &\Rightarrow LZ\} \leq b\frac{s^{-1}}{(s^{\alpha}+d)} + \frac{s^{\alpha-1}}{(s^{\alpha}+d)}Z_0 \\ &\Rightarrow LZ\} \leq b\frac{s^{-1}}{(s^{\alpha}+d)} + \frac{s^{\alpha-1}}{(s^{\alpha}+d)}, \end{aligned}$$

where L is Laplace transform operator. By applying inverse Laplace transform we obtain $Z \leq bt^{\alpha}E_{\alpha,\alpha+1}(-dt^{\alpha}) + E_{\alpha,1}(-dt^{\alpha})$.

Using the relation $E_{\alpha,\beta}(z) = zE_{\alpha,\alpha+\beta}(z) + \frac{1}{\Gamma(\beta)}$ we have $t^{\alpha}E_{\alpha,\alpha+1}(-dt^{\alpha}) = \frac{1}{d}(1 - E_{\alpha,1}(-dt^{\alpha}))$. Then $Z \leq \frac{b}{d}(1 - E_{\alpha,1}(-dt^{\alpha})) + E_{\alpha,1}(-dt^{\alpha})$.

Since $0 \leq E_{\alpha,1}(-dt^{\alpha}) \leq 1$, we have $0 \leq Z \leq 1$ which implies that, $0 \leq (S_c + R_c + I_c + S_a + E_a + I_a + C_a) \leq 1$.

Therefore, region $\Omega = \{(S_a, S_c, R_c, I_c, E_a, I_a, C_a) \in R_+^7 | 0 \leq S_c + R_c + I_c + S_a + E_a + I_a + C_a \leq 1\}$ is positively invariant region.

3.2. Disease-free equilibrium(DFE) point and basic reproduction number (R_0)

We begin by determining the disease-free equilibrium (DFE) point when there is no infection in the population. So DFE point of model system (2.6) is obtained by making all equations of (2.6) equal to zero. That is

$$\begin{cases} D^{\alpha}S_a = mS_c - \beta S_a(E_a + I_a + C_a) - (p+d)S_a = 0, \\ D^{\alpha}S_c = b(1-\omega) - b(1-\omega)((1-k)(E_a + I_a + C_a) + S_c + R_c + I_c) - (v+m+d)S_c = 0, \\ D^{\alpha}R_c = b\omega - b\omega(S_c + R_c + I_c) + vS_c - (m+d)R_c = 0, \\ D^{\alpha}I_c = b(1-\omega)(1-k)(E_a + I_a + C_a) - (m+d)I_c = 0, \\ D^{\alpha}E_a = \beta S_a(E_a + I_a + C_a) - (\sigma+d)E_a = 0, \\ D^{\alpha}I_a = \sigma E_a - (\gamma+\theta+d)I_a = 0, \\ D^{\alpha}C_a = \theta I_a + mI_c - (d+\delta)C_a = 0. \end{cases} \tag{3.13}$$

Since, at DFE point there is no infection we have $I_c = 0, E_a = 0, I_a = 0$, and $C_a = 0$, when we substitute $I_c = 0, E_a = 0, I_a = 0$ and $C_a = 0$ into system of Equation (3.13), we obtain

$$\begin{cases} mS_c - (p+d)S_a = 0, \\ b(1-\omega) - b(1-\omega)(S_c + R_c) - (v+m+d)S_c = 0, \\ b\omega - b\omega(S_c + R_c) + vS_c - (m+d)R_c = 0. \end{cases} \tag{3.14}$$

Therefore, DFE point is obtained by solving for system of Equation (3.14). From first equation of (3.14), we have

$$S_a = \frac{m}{p+d}S_c. \tag{3.15}$$

From the second equation of (3.14), we have

$$R_c = 1 - S_c - \frac{v+m+d}{b(1-\omega)}S_c. \tag{3.16}$$

From the third equation of (3.14), we have

$$R_c = \frac{b\omega}{(b\omega+m+d)} + \frac{v - b\omega}{(b\omega+m+d)}S_c. \tag{3.17}$$

Equate (3.16) and (3.17) and solve for S_c, we obtain

$$S_c^0 = \frac{b(1-\omega)(m+d)}{(v+m+d)(b+m+d)}. \tag{3.18}$$

Substituting (3.18) into (3.15), we get

$$S_a^0 = \frac{mb(1-\omega)(m+d)}{(p+d)(v+m+d)(b+m+d)}.$$

Then substituting (3.18) into (3.16), we get

$$R_c^0 = \frac{bv + b\omega(m+d)}{(v+m+d)(b+m+d)}.$$

Therefore, DFE point is given by,

$$E^0 = \left(\frac{mb(1-\omega)(m+d)}{(p+d)(v+m+d)(b+m+d)}, \frac{b(1-\omega)(m+d)}{(v+m+d)(b+m+d)}, \frac{bv + b\omega(m+d)}{(v+m+d)(b+m+d)}, 0, 0, 0, 0\right).$$

Next, we find threshold parameter known as basic reproduction number (R_0). Basic reproduction number is defined as the average number of secondary infective produced by the presence of one-infective individuals in totally susceptible populations. To compute R_0, we use method of next-generation matrix method and R_0 is spectral radius of next-generation matrix (Van den Driessche & Watmough, 2002).

Let us consider from (2.6) only an equation of infective classes only as follows

$$\begin{aligned}
D^\alpha I_c &= b(1-\omega)(1-k)(E_a + I_a + C_a) - (m+d)I_c,\\
D^\alpha E_a &= \beta S_a(E_a + I_a + C_a) - (\sigma + d)E_a,\\
D^\alpha I_a &= \sigma E_a - (\gamma + \theta + d)I_a,\\
D^\alpha C_a &= \theta I_a + mI_c - (d+\delta)C_a.
\end{aligned}$$

Let f be the rate of introduction of new infective and v be transmission after new infection and transfer of individual out of compartment, then

$$f = \begin{bmatrix} b(1-\omega)(1-k)(E_a + I_a + C_a) \\ \beta S_a(E_a + I_a + C_a) \end{bmatrix}$$

$$v = \begin{bmatrix} (m+d)I_c \\ (\sigma+d)E_a \\ (\gamma+\theta+d)I_a - \sigma E_a \\ (d+\delta)C_a - \theta I_a - mI_c \end{bmatrix}.$$

Let F and V be the Jacobian matrix of f and v evaluated at DFE point, then

$$F = \begin{pmatrix} 0 & b(1-\omega)(1-k) & b(1-\omega)(1-k) & b(1-\omega)(1-k) \\ 0 & \beta S_a^0 & \beta S_a^0 & \beta S_a^0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V = \begin{pmatrix} (m+d) & 0 & 0 & 0 \\ 0 & (\sigma+d) & 0 & 0 \\ 0 & -\sigma & (\gamma+\theta+d) & 0 \\ -m & 0 & -\theta & (\delta+d) \end{pmatrix}.$$

The inverse of V is given by

$$V^{-1} = \begin{pmatrix} \frac{1}{(m+d)} & 0 & 0 & 0 \\ 0 & \frac{1}{(\sigma+d)} & 0 & 0 \\ 0 & \frac{\sigma}{(\sigma+d)(\gamma+\theta+d)} & \frac{1}{(\gamma+\theta+d)} & 0 \\ \frac{m}{(m+d)(\delta+d)} & \frac{\sigma\theta}{(\delta+d)(\sigma+d)(\gamma+\theta+d)} & \frac{\theta}{(\delta+d)(\gamma+\theta+d)} & \frac{1}{(\delta+d)} \end{pmatrix}.$$

Then

$$FV^{-1} = \begin{pmatrix} a_1b_5 & a_1(b_2+b_3+b_6) & a_1(b_4+b_7) & a_1b_8 \\ a_2b_5 & a_2(b_2+b_3+b_6) & a_2(b_4+b_7) & a_2b_8 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

where $a_1 = b(1-\omega)(1-k)$, $a_2 = \beta S_a^0, b_2 = \frac{1}{(\sigma+d)}$, $b_3 = \frac{\sigma}{(\sigma+d)(\gamma+\theta+d)}$, $b_6 = \frac{\sigma\theta}{(\delta+d)(\sigma+d)(\gamma+\theta+d)}$, $b_7 = \frac{\theta}{(\delta+d)(\gamma+\theta+d)}$, $b_8 = \frac{1}{(\delta+d)}, b_5 = \frac{m}{(m+d)(\delta+d)}$.

The characteristic polynomial of FV^{-1} is given by $p(\lambda) = (\lambda)^3(\lambda - a_1b_5 - a_2(b_2+b_3+b_6))$. The eigenvalues of FV^{-1} are roots of characteristic polynomials $P(\lambda)$.

Roots of characteristic polynomial are $\lambda_1 = \lambda_2 = \lambda_3 = 0$ and $\lambda_4 = a_1b_5 + a_2(b_2+b_3+b_6)$.

When we substitute for a_1, b_5, a_2, b_2, b_3 and b_6, we get λ_4

$$\lambda_4 = \frac{mb(1-\omega)(1-k)}{(m+d)(\delta+d)} + \frac{\beta mb(1-\omega)(m+d)((\delta+d)(\sigma+\gamma+\theta+d)+\sigma\theta)}{(p+d)(b+m+d)(v+m+d)(\sigma+d)(\gamma+\theta+d)(\delta+d)} > 0.$$

Since the largest eigenvalue is λ_4, then the basic reproduction number is given as

$$R_0 = \frac{mb(1-\omega)(1-k)}{(m+d)(\delta+d)} + \frac{\beta mb(1-\omega)(m+d)((\delta+d)(\sigma+\gamma+\theta+d)+\sigma\theta)}{(p+d)(b+m+d)(v+m+d)(\sigma+d)(\gamma+\theta+d)(\delta+d)}.$$

3.3. Local and global stability of disease-free equilibrium point

Theorem 3.3. (Local stability of DFE) *The disease free equilibrium (DFE) point of our model system* (2.6) *is locally asymptotically stable if and only if* $R_0 < 1$.

Proof. To show that the DFE point of our model system (2.6) is locally asymptotically stable, we need to show that all the eigenvalues of Jacobian matrix J of system of Equation (2.6) evaluated at DFE point satisfies the condition $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$.

Let us find the Jacobian matrix J of model system of Equation (2.6) at DFE point. Jacobian matrix J of (2.6) evaluated at DFE point is given by

$$J_{E^0} = \begin{pmatrix} -(p+d) & 0 & 0 & 0 & -\beta S_a^0 & -\beta S_a^0 & -\beta S_a^0 \\ 0 & a_1 & -b(1-\omega) & -b(1-\omega) & a_2 & a_2 & a_2 \\ 0 & -b\omega+v & a_3 & -b\omega & 0 & 0 & 0 \\ 0 & 0 & 0 & -(m+d) & b(1-\omega) & b(1-\omega) & b(1-\omega) \\ 0 & 0 & 0 & 0 & \beta S_a^0 - (\sigma+d) & \beta S_a^0 & \beta S_a^0 \\ 0 & 0 & 0 & 0 & \sigma & -(\gamma+\theta+d) & 0 \\ 0 & 0 & 0 & m & 0 & \theta & -(\delta+d) \end{pmatrix},$$

where $-b(1-\omega)-(v+m+d) = a_1, -b(1-\omega)(1-k) = a_2, -b\omega-(m+d) = a_3$. The characteristic polynomial $P(\lambda)$ of J_{E^0} is given by

$$P(\lambda) = -(p+d+\lambda)(v+m+d+\lambda)(b+m+d+\lambda)(\lambda^4 + A\lambda^3 + D\lambda^2 + H\lambda + Q),$$

where $A = \sigma+\gamma+\theta+\delta+4d-\beta S_a^0$, $D = (\gamma+\theta+d)(\sigma+d) + (\sigma+\gamma+\theta+2d)(\delta+m+2d) + (\delta+d)(m+d) - \beta S_a^0(\sigma+\gamma+\delta+\theta+m+3d) - mb(1-\omega)(1-k)$, $H = (\sigma+\gamma+\theta+2d)(\delta+d)(m+d) + (\gamma+\theta+d)(\sigma+d)(\delta+m+2d) - mb(1-\omega)(1-k)(\sigma+\gamma+\theta+2d) - \beta S_a^0((\delta+d)(m+d) + (\delta+m+2d)(\gamma+\theta+d) + \sigma(m+\delta+\theta+2d))$, $Q = (\gamma+\theta+d)(\sigma+d)(\delta+d)(m+d)(1-R_0)$.

The eigenvalues of J_{E^0} are the roots of $P(\lambda)$. Roots of characteristic polynomial are $\lambda_1 = -(p+d), \lambda_2 = -(v+m+d), \lambda_3 = -(b+m+d)$ and

$$\lambda^4 + A\lambda^3 + D\lambda^2 + H\lambda + Q = 0.$$

Let us set

$$p(\lambda) = \lambda^4 + A\lambda^3 + D\lambda^2 + H\lambda + Q. \tag{3.19}$$

To show all roots of polynomial (3.19) satisfies $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$, we use fractional Ruth-Hurwitz conditions (Ahmed, El-Sayed, & El-Saka, 2006)

The discriminant $D(p)$ of the polynomial $p(\lambda)$ is given by

$$D(p) = \begin{vmatrix} 1 & A & D & H & Q & 0 & 0 \\ 0 & 1 & A & D & H & Q & 0 \\ 0 & 0 & 1 & A & D & H & Q \\ 4 & 3A & 2D & H & 0 & 0 & 0 \\ 0 & 4 & 3A & 2D & H & 0 & 0 \\ 0 & 0 & 4 & 3A & 2D & H & 0 \\ 0 & 0 & 0 & 4 & 3A & 2D & H \end{vmatrix}.$$

$$D(p) = 16D^2Q + 144DH^2Q + 144A^2DQ^2 + 18ADH^3 + A^2D^2H^2 + 18A^3DHQ + 68AD^2HQ + 256Q^3 - 64D^2Q^2 - 4A^2D^3Q - 27A^4Q^2 - 27H^4 - 4D^3H^2 - 4A^3H^3 - 192AHQ^2 - 6A^2H^2Q.$$

All roots of polynomial (3.19) satisfies $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$ if the following conditions holds

- If $D(p) > 0, A \geq 0, D \geq 0, H \geq 0, Q > 0, \alpha < \frac{1}{2}$, then all roots of polynomial (3.19) satisfies $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$, which implies E^0 is locally asymptotically stable.
- If $D(p) > 0, A < 0, D < 0, H < 0, \alpha > \frac{1}{2}$, then all roots of polynomial (3.19) are not satisfies $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$, which implies E^0 *is unstable.*
- If $D(p) > 0, A > 0, D > 0, H > 0, ADH - H^2 = A^2Q$, then all roots of polynomial (3.19) satisfies $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$ for all $\alpha \in (0, 1)$.
- If $D(p) < 0, A \geq 0, D \geq 0, H \geq 0, Q > 0, \alpha < \frac{1}{2}$, then all roots of polynomial (3.19) satisfies $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$
- If $D(p) < 0, A < 0, D < 0, H < 0, \alpha > \frac{1}{2}$, then all roots of polynomial (3.19) are not satisfies $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$.
- If $D(p) < 0, A > 0, D > 0, H > 0, ADH - H^2 = A^2Q$, then all roots of polynomial (3.19) satisfy $|arg(\lambda_i)| > \alpha\frac{\pi}{2}$, for all $\alpha \in (0, 1)$

The necessary condition that all roots of (3.19) satisfy $|(\lambda_i)| > \alpha\frac{\pi}{2}$ is $Q > 0$, and $Q > 0$, if and only if $R_0 < 1$.

Theorem 3.4. (Global Stability of DFE). *The disease free equilibrium point is globally asymptotically stable if* $R_0 < 1$.

Proof. To proof this theorem we construct a Lyapunov function given below

$$V = \frac{m}{m+d}I_c + \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\sigma+d)(\gamma+\theta+d)}\right]E_a + \left(\frac{\theta+\delta+d}{\gamma+\theta+d}\right)I_a + C_a.$$

Then, we find Caputo fractional derivatives of V, to obtain

$$D^\alpha V = \frac{m}{m+d}D^\alpha I_c + \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\sigma+d)(\gamma+\theta+d)}\right]D^\alpha E_a + \left(\frac{\theta+\delta+d}{\gamma+\theta+d}\right)D^\alpha I_a + D^\alpha C_a. \tag{3.20}$$

Then substitute for $D^\alpha I_c, D^\alpha E_a, D^\alpha I_a$, and $D^\alpha C_a$ from system (2.6) into (3.20), we get

$D^\alpha V = \frac{m}{m+d}\ (b(1-\omega)(1-k)(E\ \ a + I_a + C_a) - (m+d)\ \ I_c) + \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\sigma+d)(\gamma+\theta+d)}\right]\ (\beta S_a(E_a + I_a + C_a) - (\sigma+d)E_a) + \left(\frac{\theta+\delta+d}{\gamma+\theta+d}\right)(\sigma E_a - (\gamma+\theta+d)I_a) + (\theta I_a + mI_c - (d+\delta)C_a).$

This implies

$$D^\alpha V = \frac{mb(1-\omega)(1-k)}{m+d}(E_a + I_a + C_a) - mI_c + \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\sigma+d)(\gamma+\theta+d)}\right]\beta S_a(E_a + I_a + C_a) - \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\gamma+\theta+d)}\right]E_a + \frac{(\theta+\delta+d)\sigma}{\gamma+\theta+d})E_a - (\theta+\delta+d)I_a + \theta I_a + mI_c - (d+\delta)C_a.$$

By collecting like term we obtain

$$D^\alpha V = \frac{mb(1-\omega)(1-k)}{m+d}(E_a + I_a + C_a) + \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\sigma+d)(\gamma+\theta+d)}\right]\beta S_a(E_a + I_a + C_a) - (\delta+d)E_a - (\delta+d)(I_a + C_a),$$

which implies

$$D^{\alpha}V = \left[\frac{mb(1-\omega)(1-k)}{m+d} + \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\sigma+d)(\gamma+\theta+d)}\right]\beta S_a - (\delta+d)\right](E_a+I_a+C_a).$$

So, we have

$$D^{\alpha}V = (\delta+d)\left[\frac{mb(1-\omega)(1-k)}{(\delta+d)(m+d)} + \left[\frac{(\delta+d)(\sigma+\gamma+\theta+d)+\theta\sigma}{(\delta+d)(\sigma+d)(\gamma+\theta+d)}\right]\beta S_a - 1\right](E_a+I_a+C_a). \tag{3.21}$$

At the DFE point, (3.21) becomes

$D^{\alpha}V = (\delta+d)[R_0-1](E_a+I_a+C_a)$, that is, $D^{\alpha}V = [R_0-1](\delta+d)(E_a+I_a+C_a)$.

So, $D^{\alpha}V \le 0$, *if* $R_0 \le 1$.

Furthermore, $D^{\alpha}V = 0$ if $E_a = I_a = C_a = 0$ or $R_0 = 1$. Therefore, V is Lyapunov function on the feasible region and the largest compact set in the feasible region.

$\{(S_a, S_c, R_c, I_c, E_a, I_a, C_a) \in R_+^7, D^{\alpha}V = 0\}$ *is the singleton* $(S_a^0, S_c^0, R_c^0, 0, 0, 0, 0)$.

Hence, the DFE point of our model is globally asymptotically stable if and only if $R_0 < 1$.

3.4. Endemic equilibrium (EE) point

Endemic equilibrium point occurs when the disease exist in the population. EE point of our model is obtained by making right hand side of all equations of system (2.6) equal to zero. EE point of our model is obtained by solving the following system of equations

$$\begin{cases} mS_c - \beta S_a(E_a+I_a+C_a) - (p+d)S_a = 0, \\ b(1-\omega) - b(1-\omega)((1-k)(E_a+I_a+C_a) + S_c + R_c + I_c) - (v+m+d)S_c = 0, \\ b\omega - b\omega(S_c+R_c+I_c) + vS_c - (m+d)R_c = 0, \\ b(1-\omega)(1-k)(E_a+I_a+C_a) - (m+d)I_c = 0, \\ \beta S_a(E_a+I_a+C_a) - (\sigma+d)E_a = 0, \\ \sigma E_a - (\gamma+\theta+d)I_a = 0, \\ \theta I_a + mI_c - (d+\delta)C_a = 0. \end{cases} \tag{3.22}$$

From fourth equation of (3.22) we have

$$E_a + I_a + C_a = \frac{(m+d)}{b(1-\omega)(1-k)}I_c. \tag{3.23}$$

From sixth equation of (3.22) we have

$$I_a = \frac{\sigma}{(\gamma+\theta+d)}E_a. \tag{3.24}$$

From seventh equation of (3.22) we have

$$C_a = \frac{\theta}{d+\delta}I_a + \frac{m}{d+\delta}I_c. \tag{3.25}$$

Plugging (3.24) into (3.25)

$$C_a = \frac{\theta\sigma}{(d+\delta)(\gamma+\theta+d)}E_a + \frac{m}{d+\delta}I_c. \tag{3.26}$$

Substituting (3.24) and (3.26) into (3.23) and solving for E_a in terms of I_c, we obtain

$$\Rightarrow E_a = \frac{(\gamma+\theta+d)((m+d)(\delta+d) - mb(1-\omega)(1-k))}{b(1-\omega)(1-k)((\delta+d)(\gamma+\theta+d)+\sigma(\delta+d)+\sigma\theta)}I_c. \tag{3.27}$$

From the fifth equation of (3.22), we have

$$\beta S_a(E_a+I_a+C_a) = (\sigma+d)E_a. \tag{3.28}$$

Put (3.23) and (3.27) into (3.28) and solve for S_a

$$\Rightarrow S_a^{\star} = \frac{(\sigma+d)(\gamma+\theta+d)((m+d)(\delta+d) - mb(1-\omega)(1-k))}{\beta(m+d)((\delta+d)(\gamma+\theta+d)+\sigma(\delta+d)+\sigma\theta)}. \tag{3.29}$$

Substitute (3.23) into the second equation of (3.22) and solve for R_c in terms of I_C and S_c, we get

$$R_c = 1 - \left(1 + \frac{m+d}{b(1-\omega)}\right)I_c - \left(1 + \frac{v+m+d}{b(1-\omega)}\right)S_c. \tag{3.30}$$

From third equation of (3.22), we have

$$\Rightarrow R_c = \frac{b\omega}{b\omega+m+d} + \frac{v-b\omega}{b\omega+m+d}S_c - \frac{b\omega}{b\omega+m+d}I_c. \tag{3.31}$$

By equate (3.30) and (3.31) and solve for S_c in terms of I_c, we get

$$\Rightarrow S_c = \frac{b(1-\omega)(m+d)}{(v+m+d)(b+m+d)} - \frac{m+d}{v+m+d}I_c. \tag{3.32}$$

Substituting (3.28) into the first equation of (3.22) yields

$$mS_c-(\sigma+d)E_a-(p+d)S_a = 0 \tag{3.33}$$

Substituting (3.27), (3.29) and (3.32) into (3.33) gives

$$\frac{mb(1-\omega)(m+d)}{(v+m+d)(b+m+d)} - \frac{m(m+d)}{v+m+d}I_c - \frac{(\sigma+d)(\gamma+\theta+d)((m+d)(\delta+d)-mb(1-\omega)(1-k))}{b(1-\omega)(1-k)((\delta+d)(\gamma+\theta+d)+\sigma(\delta+d)+\sigma\theta)}I_c-(p+d)S_a^\star = 0.$$

$$\Rightarrow \frac{mb(1-\omega)(m+d)}{(v+m+d)(b+m+d)} - \frac{m(m+d)}{v+m+d}I_c - \frac{(\sigma+d)(\gamma+\theta+d)((m+d)(\delta+d)-mb(1-\omega)(1-k))}{b(1-\omega)(1-k)((\delta+d)(\gamma+\theta+d)+\sigma(\delta+d)+\sigma\theta)}I_c = (p+d)S_a^\star.$$

Hence, we have

$$\frac{mb(1-\omega)(m+d)}{(v+m+d)(b+m+d)} - (p+d)S_a^\star = \left(\frac{m(m+d)}{v+m+d} + \frac{(\sigma+d)(\gamma+\theta+d)((m+d)(\delta+d)-mb(1-\omega)(1-k))}{b(1-\omega)(1-k)((\delta+d)(\gamma+\theta+d)+\sigma(\delta+d)+\sigma\theta)}\right)I_c.$$

After some calculation, we obtain

$$I_c^\star = M(R_0-1), \tag{3.34}$$

where $M = \frac{b(1-\omega)(1-k)(v+m+d)(p+d)(\delta+d)(\sigma+d)(\gamma+\theta+d)}{\beta y}$ and $y = mb(1-\omega)\ (1-k)(m+d)((\delta+\ d)(\sigma+\gamma+\theta+d)+\theta\sigma) + (\sigma+d)\ (\gamma+\theta+d)(v+m+d)((m+\ d)(\delta+d)\ -mb(1-\omega)(1-k))$.

Substituting (3.34) into (3.27), we obtain

$$E_a^\star = M_1M(R_0-1), \tag{3.35}$$

where $M_1 = \frac{(\gamma+\theta+d)((m+d)(\delta+d)-mb(1-\omega)(1-k))}{b(1-\omega)(1-k)((\delta+d)(\gamma+\theta+d)+\sigma(\delta+d)+\sigma\theta)}$.

Substituting (3.35) into (3.24), we obtain

$$I_a^\star = (R_0-1)\frac{\sigma M_1 M}{(\gamma+\theta+d)}. \tag{3.36}$$

Substituting (3.34) and (3.36) into (3.26), we get $C_a^\star = (R_0-1)M\left(\frac{\sigma\theta M_1}{(\delta+d)(\gamma+\theta+d)} + \frac{m}{\delta+d}\right)$.
Substituting (3.34) into (3.32) we get $S_c^\star = \frac{m+d}{(v+m+d)(b+m+d)}(b(1-\omega)-M(b+m+d)(R_0-1))$.
Substituting (3.32) into (3.30), then (3.30) becomes

$$R_c = \frac{bv+b\omega(m+d)}{(v+m+d)(b+m+d)} - \frac{v}{v+m+d}I_c. \tag{3.37}$$

Then substituting (3.34) into (3.37), we obtain

$$R_c^\star = \frac{bv+b\omega(m+d)-v(b+m+d)M(R_0-1)}{(b+m+d)(v+m+d)}.$$

Hence, if $R_0>1$, then model (2.6) has unique endemic equilibrium point given by $E^\star = (S_a^\star, S_c^\star, R_c^\star, I_c^\star, E_a^\star, I_a^\star, C_a^\star)$. In this section, we prove global stability of endemic equilibrium *(EE) point* $(E^\star)$.

Theorem 3.5. (Global Stability of Endemic Equilibrium(EE) Point). *Let* $\alpha \in (0,1)$ *and* $R_0>1$,*then unique endemic equilibrium point* $E^\star$ *is globally asymptotically stable.*

Proof. Consider the following function

$$L(t) = L_1(S_a(t)) + L_2(S_c(t)) + L_3(R_c(t)) + L_4(I_c(t)) + L_5(E_a(t)) + L_6(I_a(t)) + L_7(C_a(t)),$$

where $L_1(S_a(t)) = \frac{1}{2}(S_a-S_a^\star)^2$, $L_2(S_c(t)) = \frac{1}{2}(S_c-S_c^\star)^2, L_3(R_c(t)) = \frac{1}{2}(R_c-R_c^\star)^2, L_4(I_c(t)) = \frac{1}{2}(I_c-I_c^\star)^2, L_5(E_a(t)) = \frac{1}{2}(E_a-E_a^\star)^2, L_6(I_a(t)) = \frac{1}{2}(I_a-I_a^\star)^2$ and $L_7(C_a(t)) = \frac{1}{2}(C_a-C_a^\star)^2$.

The function L is well defined, continuous, and positive definite for all $S_a(t)>0, S_c(t)>0, R_c(t)>0, I_c(t)>0, E_a(t)>0, I_a(t)>0, C_a(t)>0$.

Table 1. Table of parameter symbols and their description.

Parameter symbol	Parameter description
β	Transmission rate
σ	Progression rate from exposed adult to acutely infected adult
θ	Progression rate from acutely infected adult to chronically infected adult
b	Birth rate
d	Death rate
γ	Recovery rate from acutely infected adult
δ	Disease-induced death rate in chronically infected adult
ω	Infant vaccination coverage immediately after birth
p	Adult vaccination coverage
m	Maturation to adult age above 14 years of age
v	Vaccination coverage in children
$1-k$	Probability that disease transmit to new born at birth

The Caputo fractional derivative of L yields

$$\begin{aligned} D^{\alpha}L(t) &\le (S_a-S_a^{\star})D^{\alpha}(S_a-S_a^{\star})+(S_c-S_c^{\star})D^{\alpha}(S_c-S_c^{\star})+(R_c-R_c^{\star})D^{\alpha}(R_c-R_c^{\star})+(I_c-I_c^{\star})D^{\alpha}(I_c-I_c^{\star})\\ &\quad+(E_a-E_a^{\star})D^{\alpha}(E_a-E_a^{\star})+(I_a-I_a^{\star})D^{\alpha}(I_a-I_a^{\star})+(C_a-C_a^{\star})D^{\alpha}(C_a-C_a^{\star}).\\ \Rightarrow D^{\alpha}L(t) &\le (S_a-S_a^{\star})D^{\alpha}S_a+(S_c-S_c^{\star})D^{\alpha}S_c+(R_c-R_c^{\star})D^{\alpha}R_c+(I_c-I_c^{\star})D^{\alpha}I_c+(E_a-E_a^{\star})D^{\alpha}E_a\\ &\quad+(I_a-I_a^{\star})D^{\alpha}I_a+(C_a-C_a^{\star})D^{\alpha}C_a. \end{aligned}$$

From model equation (2.6) it follows that

$$\begin{aligned} D^{\alpha}L(t) &\le (S_a-S_a^{\star})(mS_c-\beta S_a(E_a+I_a+C_a)-(p+d)S_a)\\ &\quad+(S_c-S_c^{\star})(b(1-\omega)-b(1-\omega)(1-k)(E_a+I_a+C_a)-b(1-\omega)(s_c+I_c+R_c)-(v+m+d)S_c)\\ &\quad+(R_c-R_c^{\star})(b\omega-b\omega(S_c+R_c+I_c)+vS_c-(m+d)R_c)+(I_c-I_c^{\star})(b(1-\omega)(1-k)(E_a+I_a+C_a)-(m+d)I_c)\\ &\quad+(E_a-E_a^{\star})(\beta S_a(E_a+I_a+C_a)-(\sigma+d)E_a)+(I_a-I_a^{\star})(\sigma E_a-(\gamma+\theta+d)I_a)+(C_a-C_a^{\star})(\theta I_a+mI_c-(\delta+d)C_a). \end{aligned}$$

This implies $D^{\alpha}L(t) \le mS_cS_a\ +\beta S_aS_a^{\star}\ (E_a+I_a\ +C_a)+(p+d)\ S_aS_a^{\star}-\beta S_a^2\ (E_a+I_a+C_a)-(p+d)\ S_a^2-mS_c\ S_a^{\star}+b(1-\omega)S_c+\ \ b(1-\omega)(1-k)(E_a+I_a+\ \ C_a)S_c^{\star}+b(1-\omega)(S_c+R\ \ c+I_c)S_c^{\star}+(v+m+d)S_c\ \ S_c^{\star}-b(1-\omega)(1-k)(E_a+I_a+C_a)\ \ S_c-b(1-\omega)(S_c+R_c\ \ +I_c)S_c-(v+m+d)\ \ S_c^2-b(1-\omega)S_c^{\star}\ \ +b\omega R_c+vS_cR_c+\ \ b\omega(S_c+R_c+\ \ I_c)R_c^{\star}+(m+d)R_c\ \ \ \ R_c^{\star}-b\omega(S_c+\ \ \ \ R_c+I_c)R_c-(m+d)\ \ \ \ R_c^2-b\omega R_c^{\star}-vS_cR_c^{\star}\ \ \ \ +b(1-\omega)(1-k)(E_a+I_a+C_a)I_c\ \ \ \ +(m+d)I_cI_c^{\star}-b(1-\omega)(1-k)\ \ (E_a+I_a+C_a)I_c^{\star}-(m+d)I_c^2+\beta\ \ S_a(E_a+I_a+C_a)E_a+(\sigma+d)\ \ E_aE_a^{\star}-(\sigma+d)E_a^2\ \ -\beta S_a(E_a+Ia+C_a)E_a^{\star}+\sigma E_aI_a+\ \ \ (\gamma+\theta+d)I_aI_a^{\star}\ \ \ -(\gamma+\theta+d)I_a^2-\sigma E_a\ \ \ I_a^{\star}+\theta I_aC_a\ \ \ +mI_cC_a+(\delta+d)C_a\ \ \ C_a^{\star}-(\delta+d)C_a^2-\theta I_aC_a^{\star}-m\ _{Ic}C_a^{\star}.$

By collecting the positive terms together and negative terms together, we obtain $D^{\alpha}L(t) \le Y-X$, where

$$\begin{aligned} Y &= S_a(mS_c+(p+d)S_a^{\star})+\beta S_a(E_a+I_a+C_a)(S_a^{\star}+E_a)+b(1-\omega)(1-k)(E_a+I_a+C_a)(S_c^{\star}+I_c)\\ &\quad+(b(1-\omega)S_c^{\star}+b\omega R_c^{\star})(S_c+R_c+I_c)+S_c(v(S_c^{\star}+R_c)+b(1-\omega))+b\omega R_c+(m+d)(R_cR_c^{\star}+I_cI_c^{\star}+S_cS_c^{\star})\\ &\quad+(\sigma+d)E_aE_a^{\star}+\sigma I_a(E_a+E_a^{\star})+(\theta I_a+mI_c+(\delta+d)C_a^{\star})C_a. \end{aligned}$$

And

$$\begin{aligned} X &= \beta S_a(E_a+I_a+C_a)(S_a+E_a^{\star})+(p+d)S_a^2+mS_cS_a^{\star}+b(1-\omega)(1-k)(E_a+I_a+C_a)(S_c+I_c^{\star})\\ &\quad+(b(1-\omega)S_c+b\omega R_c)(S_c+R_c+I_c)+vS_c^2+b(1-\omega)S_c^{\star}+(b\omega+vS_c)R_c^{\star}+(m+d)(S_c^2+R_c^2+I_c^2)+(\sigma+d)E_a^2\\ &\quad+(\gamma+\theta+d)I_a^2+\sigma E_aI_a^{\star}+(\delta+d)C_a^2+\theta I_aC_a^{\star}+mI_cC_a^{\star}. \end{aligned}$$

Hence, $D^{\alpha}L(t)<0$ if and only if $Y<X$.

Furthermore, $D^{\alpha}L(t)=0$ if and only if $S_a=S_a^{\star}, S_c=S_c^{\star}, R_c=R_c^{\star}, I_c=I_c^{\star}, E_a=E_a^{\star}, I_a=I_a^{\star}, C_a=C_a^{\star}$.

Therefore, L is Lyapunov function on the feasible region and the largest set in the feasible region $\{(S_a,S_c,R_c,I_c,E_a,I_a,C_a)\in R_+^7, D^{\alpha}L=0\}$ *is the singleton* $(S_a^{\star},S_c^{\star},R_c^{\star},I_c^{\star},E_a^{\star},I_a^{\star},C_a^{\star})$.

4. Sensitivity analysis

In this section, we do sensitivity analysis of model parameters. For this, we do sensitivity analysis of basic reproduction number R_0 by computing the sensitivity index of each parameter. Sensitivity index allows to measure the relative change in basic reproduction number when parameter value changes. For this, we use normalized forward sensitivity index of basic reproduction number with respect to a given parameter using similar approach used (Tilahun, Makinde, & Malonza, 2017; Utoyo & Sa'adah, 2018).

Definition 4.1. (Utoyo & Sa'adah, 2018) Normalized forward sensitivity index of basic reproduction number (R_0) with respect to the given parameter k is defined as $Y_k^{R_0} = \frac{\partial R_o}{\partial k}\frac{k}{R_0}$.

Using the above formula, we obtained the sign of the sensitivity index for each parameter in basic reproduction number, as summarized in Table 2.

Next, we present the interpretation of sensitivity indices. From Table 2, those parameters with positive indices include $b, \beta, \theta, m,$ and $1-k$ and those parameters with negative indices include $v, p, \omega, \delta, d, \sigma,$ and γ. Those parameters with positive indices have great role in the expansion of the disease in the population as their value increases because when their value increases, basic reproduction number increases. Those parameters with negative indices have great role in eliminating the disease from the population as their value increases. Therefore, to eliminate the diseases from the population it is important to increase the value of those parameters with negative indices.

Table 2. Parameter symbols and their sensitivity indices.

Parameter symbols	Sensitivity indices
β	+ve
θ	+ve
b	+ve
m	+ve
$1-k$	+ve
σ	–ve
d	–ve
γ	–ve
δ	–ve
ω	–ve
p	–ve
v	–ve

Table 3. Table of parameter symbols and their value.

Parameter symbols	Parameters value	Sources
β	0.3795	Assumed
θ	0.4	(Goyal & Murray, 2014)
b	0.0335	Assumed
m	0.07142	(Goyal & Murray, 2014)
$1-k$	0.9	(Goyal & Murray, 2014)
σ	2.511	Assumed
d	0.0078	Assumed
γ	3.6	(Goyal & Murray, 2014)
δ	0.0013	(Goyal & Murray, 2014)
ω	0.9	(Goyal & Murray, 2014)
p	0.00013	Assumed
v	2.0401	Assumed

5. Numerical simulation

Under this section, we performed numerical simulation of the proposed fractional order model. For simulation parameters value from Table 3 is used. To obtain the numerical results of fractional order model we used Euler fractional method. We simulate effects of memory (order of derivatives)(α) on number of HB-infected individuals. We also simulated the effects of newborn vaccination immediately after birth(ω), vaccination of children(v) and adult vaccination(p) and their effects compared on the number of HB-infected individuals when the order of derivative is ($\alpha = 0.85$).

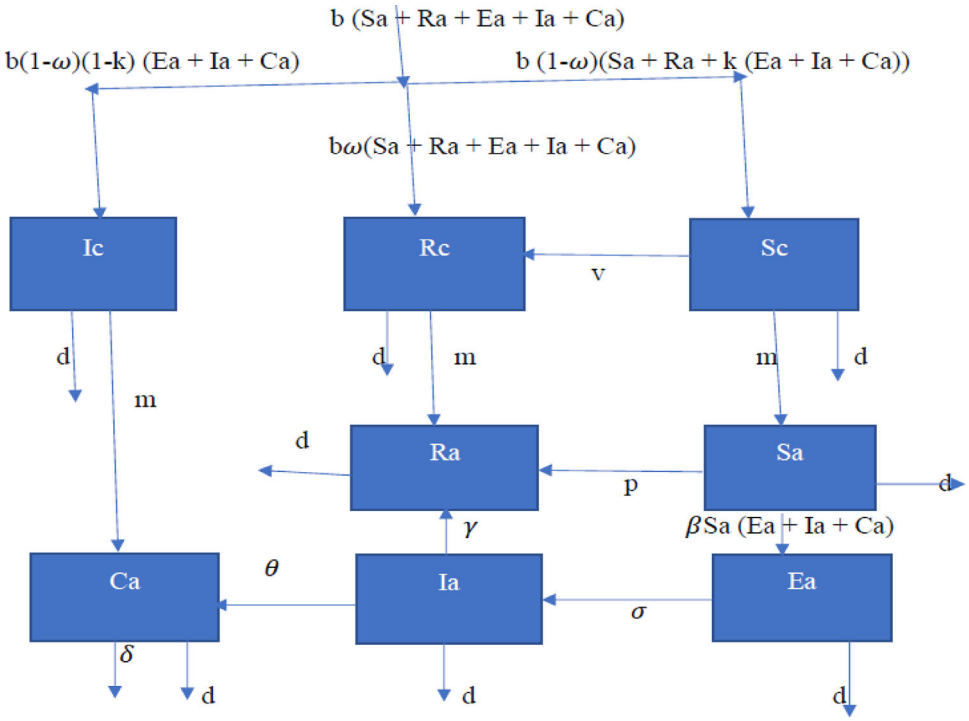
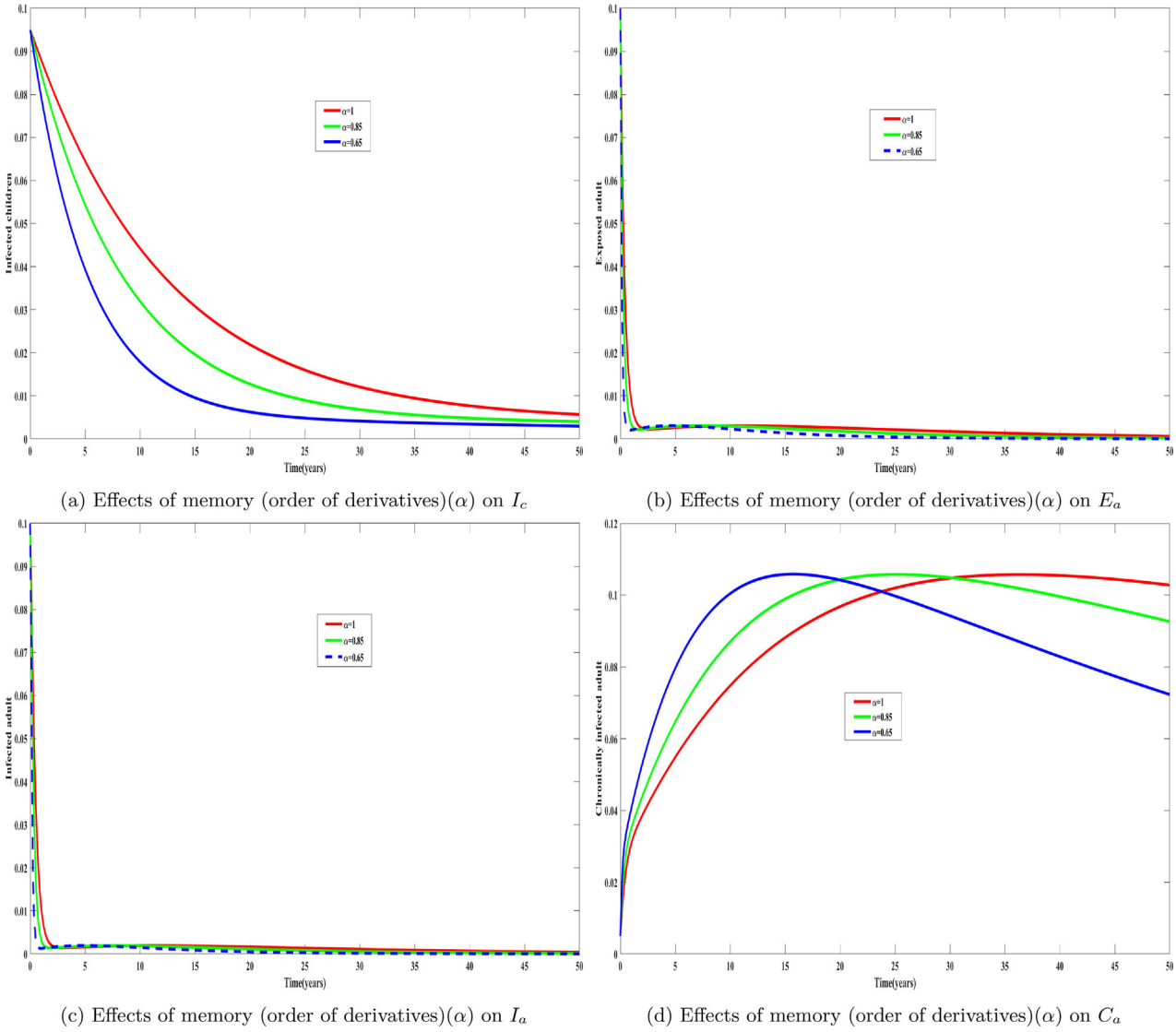


Figure 1. Flow diagram of the model. (a) Effects of memory (order of derivatives) (α) on I_c. (b) Effects of memory (order of derivatives) (α) on E_a. (c) Effects of memory (order of derivatives) (α) on I_a. (d) Effects of memory (order of derivatives) (α) on C_a.



(a) Effects of memory (order of derivatives)(α) on I_c

(b) Effects of memory (order of derivatives)(α) on E_a

(c) Effects of memory (order of derivatives)(α) on I_a

(d) Effects of memory (order of derivatives)(α) on C_a

Figure 2. Effects of memory (order of derivatives) (α). (a) Effects of newborn vaccine immediately after birth(ω) on I_c. (b) Effects of newborn vaccine immediately after birth(ω) on E_a. (c) Effects of newborn vaccine immediately after birth(ω) on I_a. (d) Effects of newborn vaccine immediately after birth(ω) on C_a.

5.1. Effect of memory on HB infection

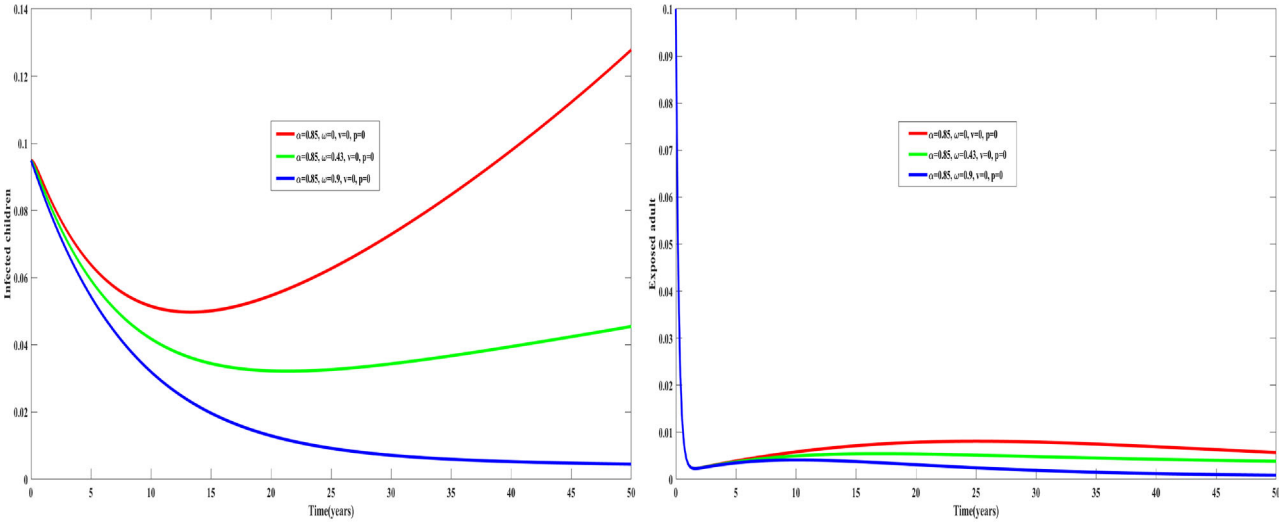
Here, we simulate the effects of memory (order of derivatives)(α) on the number of HB-infected individuals. In simulation, we compare the number of infected individuals for memory-less(integer order) model that is when ($\alpha = 1$) and model with memory that is when ($\alpha = 0.85$ and $\alpha = 0.65$). We display the effects of memory(order of derivatives)(α) on the number of HB-infected individuals on Figure 2a–d.

As we observe in Figure 2a, the number of I_c is larger in system with out memory(memory-less model)($\alpha = 1$) compared with systems with memory ($\alpha = 0.85$ and $\alpha = 0.65$).

Similarly as we observe from Figure 2b,c, the number of E_a and I_a is larger in system with out memory(memory-less model)($\alpha = 1$) compared with systems with memory ($\alpha = 0.85$ and $\alpha = 0.65$). From Figure 2d, the number of C_a increases initially, this is due to movement from I_c to C_a by maturation and progression form I_a to C_a, but through time the number of C_a becomes larger in system without memory(memory-less model)($\alpha = 1$) compared with systems with memory ($\alpha = 0.85$ and $\alpha = 0.65$). In other words, as we observe from Figure 2a–d as memory decreases (order of derivatives(α) increases) the number of infected individuals decreases slowly through time.

5.2. Effect of newborn vaccine immediately after birth(ω)

Here, we simulate the effect of newborn vaccine (ω) on HB infection by comparing the number of HB-infected individuals when no vaccine implemented($\omega = 0$, $v = 0$ and $p = 0$) and when only newborn vaccine immediately after birth (ω) implemented($\omega \neq 0, v = 0$ and $p = 0$).



(a) Effects of newborn vaccine immediately after birth(ω) on I_c

(b) Effects of newborn vaccine immediately after birth(ω) on E_a

(c) Effects of newborn vaccine immediately after birth(ω) on I_a

(d) Effects of newborn vaccine immediately after birth(ω) on C_a

Figure 3. Effects of newborn vaccine immediately after birth(ω). (a) Effects of children vaccination(v) on I_c. (b) Effects of children vaccination(v) on E_a. (c) Effects of children vaccination(v) on I_a. (d) Effects of children vaccination(v) on C_a.

On Figure 3a–d, we simulate the effects of newborn vaccine immediately after birth(ω) on the number of HB-infected individuals order of derivative is ($\alpha = 0.85$).

From Figure 3a, the number of I_c decreases as value of (ω) increases and also from Figure 3b,c, the number of E_a and I_a decreases and even falls to zero over times as value of (ω) increases. From Figure 3d, increasing value of (ω) decreases the number of C_a. Therefore, as we observe from Figure 3a–d, increasing newborn vaccine (ω) decreases the number of infected individuals.

5.3. Effect of children vaccination(v) on HB infection

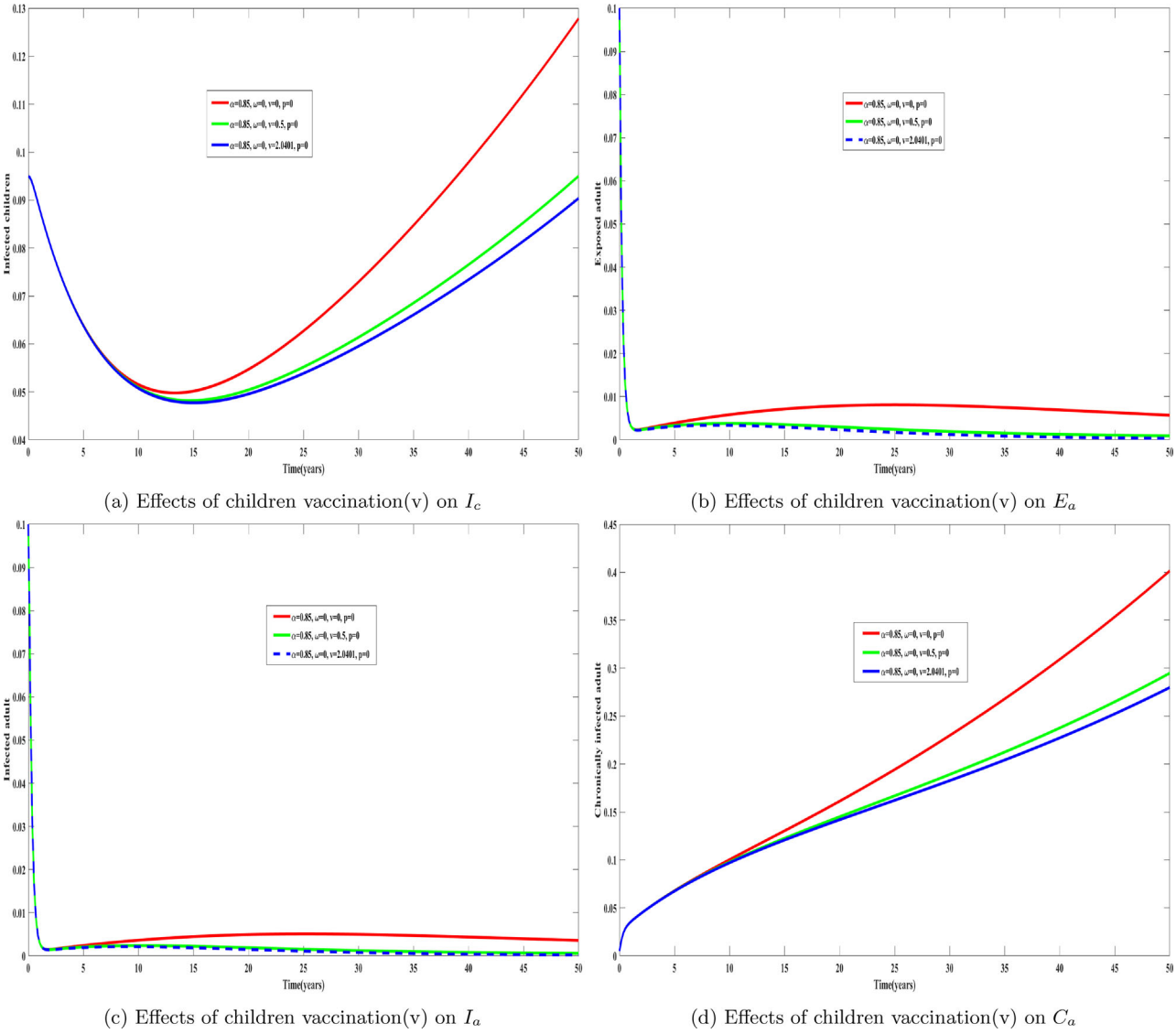
Here, we simulate effect of children vaccination(v) on HB infection by comparing the number of HB-infected individuals when no vaccine implemented($\omega = 0$, $v = 0$ and $p = 0$) and when only children vaccination(v) implemented($\omega = 0$,$v \neq 0$ and $p = 0$).

On Figure 4a–d, we simulate the effects of children vaccination(v) on number of HB-infected individuals when order of derivative is ($\alpha = 0.85$).

From Figure 4a,d, increasing value of children vaccination(v) reduces the number of I_c and C_a. From Figure 4b,c, the number of E_a and I_a decreases and even falls to zero over time as children vaccination(v) increases. In general as we observe from Figure 4a–d, increasing children vaccination(v) reduces HB infection from the population.

5.4. Effect of adult vaccination(p) on HB infection

Here, we simulate the effect of adult vaccination(p) on HB infection by comparing the number of HB-infected individuals when no vaccine was implemented($\omega = 0$, $v = 0$ and $p = 0$) and when only adult vaccination(p) is implemented($\omega = 0$,$v = 0$ and $p \neq 0$). On Figure 5a–d, we simulate the effects of adult vaccination(p) on the number of



(a) Effects of children vaccination(v) on I_c

(b) Effects of children vaccination(v) on E_a

(c) Effects of children vaccination(v) on I_a

(d) Effects of children vaccination(v) on C_a

Figure 4. Effects of children vaccination (v). (a) Effects of adult vaccination (p) on I_c. (b) Effects of adult vaccination (p) on E_a. (c) Effects of adult vaccination (p) on I_a. (d) Effects of adult vaccination (p) on C_a.

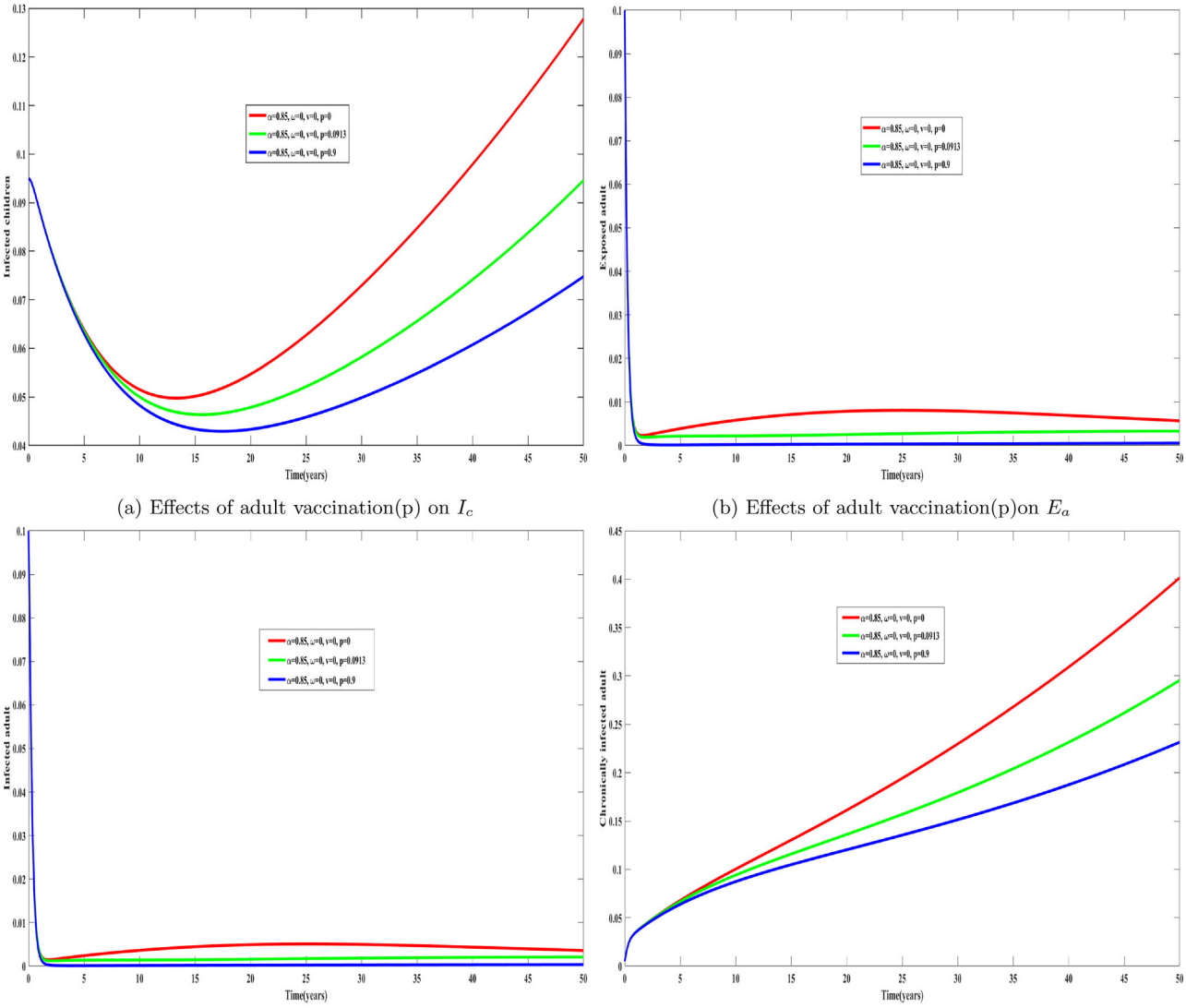
HB-infected individuals when order of derivative is ($\alpha = 0.85$).

From Figure 5a,d, the number of I_c and C_a increases as adult vaccination(p) decreases. From Figure 5b,c, the number of E_a and I_a decreases as adult vaccination(p) increases. Therefore, from Figure 5a–d, we conclude that increasing adult vaccination reduces the number of HB-infected individuals.

5.5. Comparing the effects of newborn vaccine immediately after birth(ω), children vaccination(v) and adult vaccination(p) on HB infection

Here, we compare the effects of newborn vaccine(ω), children vaccination(v), and adult vaccination(p) on HB infection by comparing number of infected individuals when no vaccine implemented ($\omega = 0$, $v = 0$ and $p = 0$), only newborn vaccine immediately after birth (ω) implemented ($\omega \neq 0$, $v = 0$ and $p = 0$), only children vaccination(v) implemented ($\omega = 0, v \neq 0$ and $p = 0$) and only adult vaccination(p) implemented ($\omega = 0$, $v = 0$ and $p \neq 0$) when order of derivative is ($\alpha = 0.85$). On Figure 6a–d, we compare the effects of newborn vaccine (ω), children vaccination(v), and adult vaccination(p) on the number of HB-infected individuals when order of derivative is ($\alpha = 0.85$).

From Figures 6a,d, we observe that increasing newborn vaccine (ω) decreases the number of I_c and C_a than increasing children vaccination(v) and adult vaccination(p). From Figure 6b,c, we observe that increasing children vaccination(v) decreases the number of E_a and I_a than increasing newborn vaccine (ω) and adult vaccination(p), and also increasing newborn vaccine (ω) decreases the number of E_a and I_a than increasing adult vaccination(p). Therefore, as we observe from Figure 6a–d, increasing newborn vaccine (ω) is preferable to eliminate HB infection from the population than increasing children vaccination(v) and adult vaccination(p).



(a) Effects of adult vaccination(p) on I_c

(b) Effects of adult vaccination(p)on E_a

(c) Effects of adult vaccination(p) on I_a

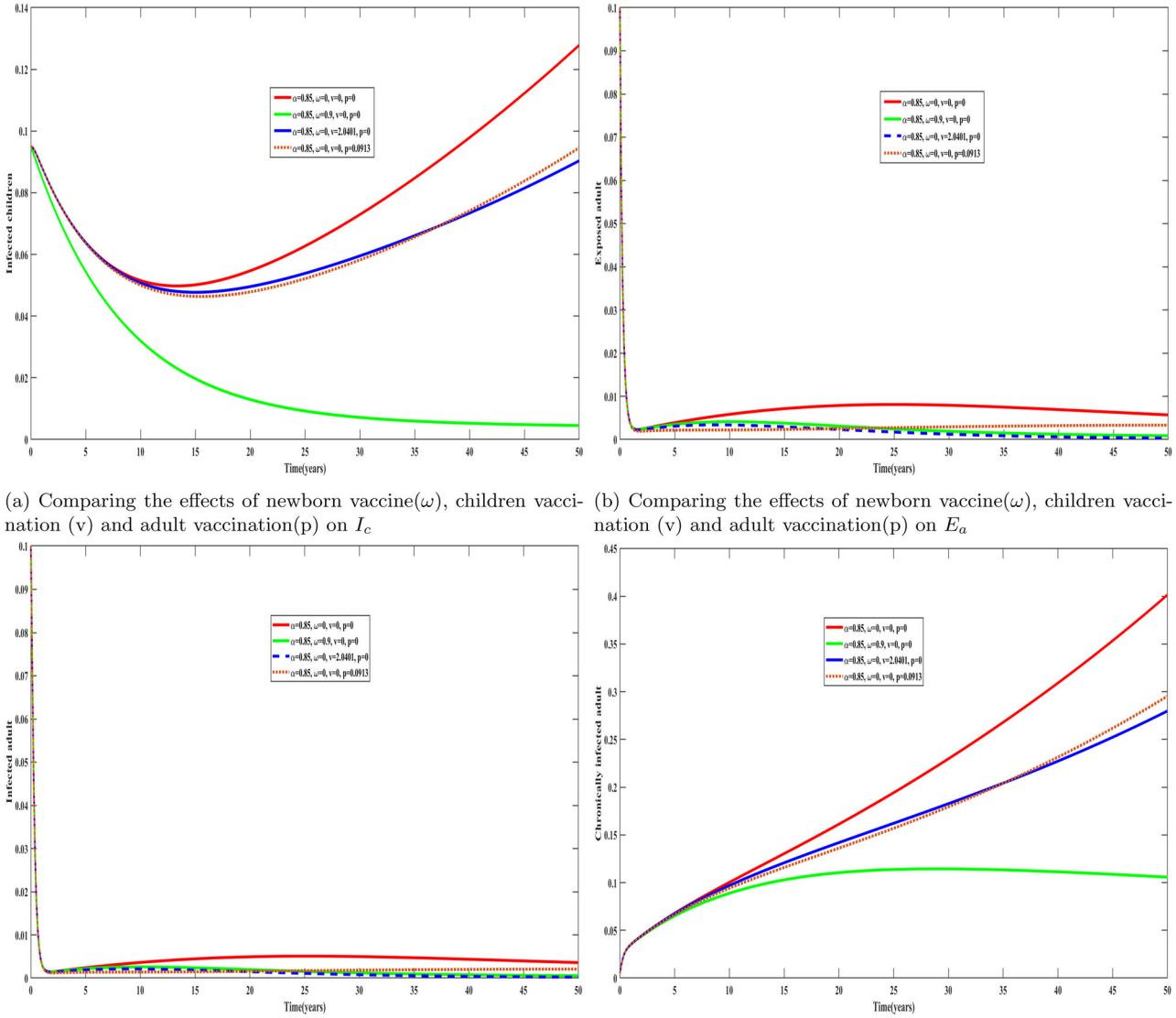
(d) Effects of adult vaccination(p) on C_a

Figure 5. Effect of adult vaccination (p). (a) Comparing the effects of newborn vaccine (ω), children vaccination (v) and adult vaccination (p) on I_c. (b) Comparing the effects of newborn vaccine (ω), children vaccination (v) and adult vaccination (p) on E_a. (c) Comparing the effects of newborn vaccine (ω), children vaccination (v) and adult vaccination (p) on I_a. (d) Comparing the effects of newborn vaccine (ω), children vaccination (v) and adult vaccination (p) on C_a.

6. Summary and conclusions

In this study, we formulated mathematical model of HB disease using fractional order to study the dynamics of HB disease in the population taking into consideration memory effect. In Section 2, we described the model assumptions and formulated the deterministic model represented by systems of ordinary differential equations and the model extended to fractional order. In Section 3, model analysis was done by proving positively invariant region in which the solution to the fractional order model is bounded and non-negative, finding equilibrium points and basic reproduction number. Local and global stability analyses of both disease-free and endemic equilibrium points are presented. In Section 4, local normalized sensitivity analysis is performed. In Section 5, numerical simulation was performed to investigate the effect of memory on HB disease dynamics and also to study the effects of newborn vaccine immediately after birth, children vaccination and adult vaccination and to compare their effects on HB disease dynamics. We used MATLAB to perform numerical simulationa.

Using numerical simulations, we investigated the impact of memory on the number of HB-infected individuals, effect of new born vaccination immediately after birth, vaccination of children and adult vaccination by using different values for the order of fractional derivative. Generally, our result shows that memory has great influence on disease dynamics and the result of the comparison between vaccination shows that increasing newborn vaccination is better to eliminate HB disease. The dynamics of the HVB is complicated and needs further research both biologically and mathematically. Although the modern fractional order models developed in this



(a) Comparing the effects of newborn vaccine(ω), children vaccination (v) and adult vaccination(p) on I_c

(b) Comparing the effects of newborn vaccine(ω), children vaccination (v) and adult vaccination(p) on E_a

(c) Comparing the effects of newborn vaccine(ω), children vaccination (v) and adult vaccination(p) on I_a

(d) Comparing the effects of newborn vaccine(ω), children vaccination (v) and adult vaccination(p) on C_a

Figure 6. Comparing the effects of newborn vaccine (ω), children vaccination (v) and adult vaccination (p).

research study could produce better results in the comparison of existing classical models, we strongly believe that this research analysis can further be enhanced. However, we notice that the models with fractional derivatives are more complicated than the ones with classical derivatives.

Disclosure statement

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

ORCID

Getachew Teshome Tilahun http://orcid.org/0000-0001-5372-4051

References

Adu, I. K., Aidoo, A. Y., Darko, I. O., & Osei-Frimpong, E. (2014). Mathematical model of hepatitis B in the Bosomtwe district of Ashanti region, Ghana. *Applied Mathematical Sciences*, *8*(67), 3343–3358. doi:10.12988/ams.2014.44263

Ahmed, E. M. E., El-Sayed, A. M. A., & El-Saka, H. A. A. (2006). On some Routh-Hurwitz conditions for fractional order differential equations and their applications in Lorenz, Rössler, Chua and Chen systems. *Physics Letters A*, *358*(1), 1–4. doi:10.1016/j.physleta.2006.04.087

Akbari, R., Kamyad, A. V., & Heydari, A. (2016). The analysis of a disease-free equilibrium of Hepatitis B model. *Sahand Communications in Mathematical Analysis*, *3*(2), 1–11. Retrieved from https://iranjournals.nlai.ir/handle/123456789/117565

Aniji, M., Kavitha, N., & Balamuralitharan, S. (2019). Analytical solution of SEICR model for Hepatitis B virus using HPM. *AIP Conference Proceedings*, *2112*, 020024.

Carvalho, A. R., Pinto, C. M., & Baleanu, D. (2018). Hiv/hcv coinfection model: A fractional-order perspective for the effect of the hiv viral load. *Advances in Difference Equations*, *2018*(1), 1–22. doi:10.1186/s13662-017-1456-z

Dan, C., Moses-Eisenstein, M., & Valdiserri, R. O. (2015). CE: Viral hepatitis: New US screening recommendations, assessment tools, and treatments. *AJN, American Journal*

of Nursing, *115*(7), 26–35. doi:10.1097/01.NAJ.0000467272.98199.7e

Ghanbari, B., Kumar, S., & Kumar, R. (2020). A study of behaviour for immune and tumor cells in immunogenetic tumour model with non-singular fractional derivative. *Chaos, Solitons & Fractals*, *133*, 109619. doi:10.1016/j.chaos.2020.109619

Goyal, A., & Murray, J. M. (2014). The impact of vaccination and antiviral therapy on hepatitis B and hepatitis D epidemiology. *PloS One*, *9*(10), e110143. doi:10.1371/journal.pone.0110143

Kamyad, A. V., Akbari, R., Heydari, A. A., & Heydari, A. (2014). Mathematical modeling of transmission dynamics and optimal control of vaccination and treatment for hepatitis B virus. *Computational and Mathematical Methods in Medicine*, *2014*, 475451. doi:10.1155/2014/475451

Kamyad, A. V., Akbari, R., Heydari, A. A., & Heydari, A. (2014). Mathematical modeling of transmission dynamics and optimal control of vaccination and treatment for hepatitis B virus. *Computational and Mathematical Methods in Medicine*, *2014*, 475451. doi:10.1155/2014/475451

Kazem, S., Abbasbandy, S., & Kumar, S. (2013). Fractional-order legendre functions for solving fractional-order differential equations. *Applied Mathematical Modelling*, *37*(7), 5498–5510. doi:10.1016/j.apm.2012.10.026

Khan, T., & Zaman, G. (2016). Classification of different Hepatitis B infected individuals with saturated incidence rate. *SpringerPlus*, *5*(1), 1082. doi:10.1186/s40064-016-2706-3

Khan, M. A., Hammouch, Z., & Baleanu, D. (2019). Modeling the dynamics of hepatitis E via the Caputo–Fabrizio derivative. *Mathematical Modelling of Natural Phenomena*, *14*(3), 311. doi:10.1051/mmnp/2018074

Khodabakhshi, N., Vaezpour, S. M., & Baleanu, D. (2017). On dynamics of fractional-order model of HCV infection. *Journal of Mathematical Analysis*, *8*(1), 16–27. Retrieved from http://hdl.handle.net/20.500.12416/2492

Kimbir, A. R., Aboiyar, T., Abu, O., & Onah, E. S. (2014). Simulation of a mathematical model of hepatitis B virus transmission dynamics in the presence of vaccination and treatment. *Mathematical Theory and Modeling*, *4*(12), 44–59.

Kumar, S. (2014). A new analytical modelling for fractional telegraph equation via Laplace transform. *Applied Mathematical Modelling*, *38*(13), 3154–3163. doi:10.1016/j.apm.2013.11.035

Kumar, S., Ghosh, S., Samet, B., & Goufo, E. F. D. (2020). An analysis for heat equations arises in diffusion process using new Yang-Abdel-Aty-Cattani fractional operator. *Mathematical Methods in the Applied Sciences*, *43*(9), 6062–6080. doi:10.1002/mma.6347

Kumar, S., Kumar, R., Agarwal, R. P., & Samet, B. (2020). A study of fractional Lotka-Volterra population model using Haar wavelet and Adams-Bashforth-Moulton methods. *Mathematical Methods in the Applied Sciences*, *43*(8), 5564–5578. doi:10.1002/mma.6297

Kumar, S., Kumar, A., & Baleanu, D. (2016). Two analytical methods for time-fractional nonlinear coupled Boussinesq–Burger's equations arise in propagation of shallow water waves. *Nonlinear Dynamics*, *85*(2), 699–715. doi:10.1007/s11071-016-2716-2

Lai, C. L., Ratziu, V., Yuen, M.-F., & Poynard, T. (2003). Viral hepatitis B. *The Lancet*, *362*(9401), 2089–2094. doi:10.1016/S0140-6736(03)15108-2

Liang, P., Zu, J., & Zhuang, G. (2018). A literature review of mathematical models of hepatitis B virus transmission applied to immunization strategies from 1994 to 2015. *Journal of Epidemiology*, *28*(5), 221–2017. doi:10.2188/jea.JE20160203

Liaw, Y.-F., & Chu, C.-M. (2009). Hepatitis B virus infection. *The Lancet*, *373*(9663), 582–592. doi:10.1016/S0140-6736(09)60207-5

Moorman, A. C., Xing, J., Ko, S., Rupp, L. B., Xu, F., Gordon, S. C., … Holmberg, S. D. (2015). Late diagnosis of hepatitis C virus infection in the Chronic Hepatitis Cohort Study (CHeCS): Missed opportunities for intervention. *Hepatology (Baltimore, Md.)*, *61*(5), 1479–1484. doi:10.1002/hep.27365

Nana-Kyere, S., Ackora-Prah, J., Okyere, E., Marmah, S., & Afram, T. (2017). Hepatitis B optimal control model with vertical transmission. *Applied Mathematics*, *7*(1), 5–13. doi:10.5923/j.am.20170701.02

Nayeem, J., & Podder, C. N. (2014). A mathematical study on the vaccination impact on the disease dynamics of HBV. *IOSR Journal of Mathematics*, *10*(6), 26–44. doi:10.9790/5728-10612644

Pimenov, A., Kelly, T. C., Korobeinikov, A., O'Callaghan, M. J., Pokrovskii, A. V., & Rachinskii, D. (2012). Memory effects in population dynamics: Spread of infectious disease as a case study. *Mathematical Modelling of Natural Phenomena*, *7*(3), 204–226. doi:10.1051/mmnp/20127313

Podlubny, I. (1998). *Fractional differential equations: An introduction to fractional derivatives, fractional differential equations, to methods of their solution and some of their applications*. San Diego, CA: Academic Press.

Qureshi, S., & Atangana, A. (2019). Mathematical analysis of dengue fever outbreak by novel fractional operators with field data. *Physica A: Statistical Mechanics and Its Applications*, *526*, 121127. doi:10.1016/j.physa.2019.121127

Rashidi, M. M., Hosseini, A., Pop, I., Kumar, S., & Freidoonimehr, N. (2014). Comparative numerical study of single and two-phase models of nanofluid heat transfer in wavy channel. *Applied Mathematics and Mechanics*, *35*(7), 831–848. doi:10.1007/s10483-014-1839-9

Rehermann, B., & Nascimbeni, M. (2005). Immunology of hepatitis B virus and hepatitis C virus infection. *Nature Reviews Immunology*, *5*(3), 215–229. doi:10.1038/nri1573

Saeedian, M., Khalighi, M., Azimi-Tafreshi, N., Jafari, G. R., & Ausloos, M. (2017). Memory effects on epidemic evolution: The susceptible-infected-recovered epidemic model. *Physical Review E*, *95*(2-1), 022409. doi:10.1103/PhysRevE.95.022409

Seyoum Desta, B., & Koya, P. R. (2019). Modified model and stability analysis of the spread of hepatitis B virus disease. *American Journal of Applied Mathematics*, *7*(1), 13–20. doi:10.11648/j.ajam.20190701.13

Shaban, N., & Manoza, D. (2016). Hepatitis B virus disease: A mathematical model for vertical transmission with treatment strategy. *Mathematical Theory and Modeling*, *6*(6), 25–35.

Shi, R., Lu, T., & Wang, C. (2019). Dynamic analysis of a fractional-order model for hepatitis B virus with Holling II functional response. *Complexity*, *2019*, 1–13. doi:10.1155/2019/1097201

Tarasov, V. E. (2018). Generalized memory: Fractional calculus approach. *Fractal and Fractional*, *2*(4), 23. doi:10.3390/fractalfract2040023

Thomson, B. (2005). European Monitoring Centre for Drugs and Drug Addiction. Hepatitis C and injecting drug use: Impact, costs and policy options (2004). EMCDDA Monograph Series no. 72004. *Clinical Microbiology and Infection*, *11*(8), 685. doi:10.1111/j.1469-0691.2005.01191.x

Tilahun, G. T., Makinde, O. D., & Malonza, D. (2017). Modelling and optimal control of typhoid fever disease with cost-effective strategies. *Computational and Mathematical Methods in Medicine*, *2017*, 2324518. doi:10.1155/2017/2324518

Umare, A., Seyoum, B., Gobena, T., & Haile Mariyam, T. (2016). Hepatitis B virus infections and associated factors among pregnant women attending antenatal care clinic at Deder Hospital, Eastern Ethiopia. *PLoS One*, *11*(11), e0166936. doi:10.1371/journal.pone.0166936

Utoyo, M. I., & Sa'adah, A. (2018). Analysis of fractional order mathematical model of hematopoietic stem cell gene-based therapy. *International Journal of Mathematics and Mathematical Sciences*, *2018*. doi:10.1155/2018/6180892

Van den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences*, *180*(1–2), 29–48. doi:10.1016/S0025-5564(02)00108-6

Wiah, E. N., Makinde, O. D., & Adetunde, I. A. (2015). A fractional-order HBV infection model with constant vaccination strategy. *Commun. Math. Biol. Neurosci*, *2015*.

World Health Organization (2020). Hepatitis b. Retrieved from https://www.who.int/news-room/fact-sheets/detail/hepatitis-b

World Health Organization and others (2012). *Prevention and control of viral hepatitis infection: Framework for global action*. Technical report, World Health Organization.

World Health Organization and others. (2017). *Global hepatitis report 2017*. World Health Organization.

Zhang, S., Guo, H., & Smith, R. (2018). Dynamical analysis for a hepatitis B transmission model with immigration and infection age. *Mathematical Bioscience Engineering*, *15*(6), 1291–1313. doi:10.3934/mbe.2018060

Zhang, T., Wang, K., & Zhang, X. (2015). Modeling and analyzing the transmission dynamics of HBV epidemic in Xinjiang. *Plos One*, *10*(9), e0138765. doi:10.1371/journal.pone.0138765

Zou, L., Zhang, W., & Ruan, S. (2010). Modeling the transmission dynamics and control of hepatitis B virus in China. *Journal of Theoretical Biology*, *262*(2), 330–338. doi:10.1016/j.jtbi.2009.09.035