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# FUZZY FRACTIONAL DERIVATIVE MODEL TO ASSESS THE DYNAMICS OF HEPATITIS B INFECTION

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Abstract. Hepatitis B virus infection shall remain a public health concern in many developed and developing countries. In this paper, we formulate and analyse a simple fuzzy fractional model of HBV infection to assess the dynamics of the disease using fractional-order differential equations. To analyse the effect of the initial transmission of the disease, we computed the basic reproduction number $\mathcal{R}_0$, and used it to perform stability analysis. The results show that the disease-free and the endemic equilibrium are globally stable with respect to the value of $\mathcal{R}_0$. Numerical simulations were performed to study the variations of each sub-population with respect to time at different order ($\alpha$). In general, results for the fractional model show that as the order ($\alpha$) increases, the population of the susceptible and exposed individuals decreases. In contrast, the other sub-populations increase with an increase in $\alpha$. Further results from the numerical analysis show that increase in $\alpha$, decreases the diameter of the fuzzy triangular solutions for the susceptible and exposed individuals in the fuzzy fractional model.

## 1. Introduction

Hepatitis B is a viral infection that attacks the liver and can cause both acute and chronic disease [1]. The virus that cause hepatitis B is a member of the family *hepadnavirus* [2]. According to WHO [1], the hepatitis virus (HBV) is most commonly transmitted from mother to child during birth and delivery, as well as through contact with blood or other body fluids during sex with an infected partner, unsafe injections or exposures to sharp instruments. WHO estimates that 296 million people were living with chronic hepatitis B infection in 2019, with 1.5 million new infections each year, and in 2019, hepatitis B resulted approximately 820,000 deaths, mostly from cirrhosis and hepatocellular carcinoma (primary liver cancer).

HBV infection can either be acute or chronic. According to WHO [1], acute hepatitis B occurs within the first 6 months after exposure to HBV. Adults, are able to clear the virus without treatment. Those who clear the virus develops long-life immune. On other hand, chronic hepatitis B is a lifelong infection with the HBV. The risk to develop a chronic hepatitis B depends on the age at which a person become exposed to HBV. Less than 5% cases of hepatitis B infection acquired in adulthood leads to chronic hepatitis, while about 95% cases of infection in childhood leads to chronic hepatitis, forming a basis for strengthening and prioritizing infant and childhood vaccination.

The burden of HBV infection is very high in Africa and other part of the world leading to a major global health concern [3]. Mathematical models developed from different types of

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differential equations have been used to describe the transmission dynamics of several infectious diseases and the possible control mechanisms available for the disease. Modeling the dynamics of HBV infection have been a major concern for epidemiologists. The first attempt to model HBV infection was by Anderson & May [4] who deterministically illustrated the effects of carriers on the transmission of HBV. Following that, Anderson et al., [5] and Williams et al., [6] developed mathematical models which included heterogeneous mixing with respect to age and sexual activity.

Several types of mathematical models which been used to describe the spread of HBV infection, including the deterministic models, stochastic models, and fractional order models. Deterministic models for HBV which uses ordinary differential equations includes Mpeshe and Nyerere [7, 8], and Zada et al. [9] just to mention a few. Stochastic models for HBV which uses stochastic differential equations includes Din, A., & Li, Y. [10, 12] and [11]. However, ordinary differential equations does not depend on the previous history of the systems, that is, they do not have memory.

The evolution and control of epidemic processes in human population cannot be considered without memory effect [13]. 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. Fractional-order differential equations are potential tools to describe the effect of memory and hence used to model infectious diseases. Fractional-order differential equations models of HBV includes [14–17] and [18] just to mention a few.

The concept of fuzziness is very important in disease modeling because the infection occur in fuzzy environment, and its decision to control the disease is fuzzy. Fuzziness is well established through the idea of fuzzy set which involve assigning to each possible individual in the population a value representing its degree of membership. For example, a fuzzy set representing our concept of infection can assign a degree of membership of 1 to a high infection, 0.5 to a medium infection, and 0 to a low infection. Fuzzy sets representing linguistic concepts such as low, medium, and high, are often employed to define states of a variable called fuzzy variable [19].

Modelling with fuzzy sets and logic has motivated many researchers in science and social sciences including epidemiology. In this paper, we present a fuzzy fractional-order model of HBV infection in order to study its dynamics. The paper begins with presentation of some fuzzy concepts followed by the model formulation and a description of parameters used in the model. The feasibility solution, basic reproduction number, and global stability analysis of the model are also discussed. Numerical simulations of the model are also established to study the behaviour of the disease over a certain time period.

## 2. Model Formulation

2.1. **The Model.** The model considers only human populations with natural and disease-dependent death rate for human. The population consists of susceptible humans ($S$), exposed humans $E$, acutely infected humans ($I_a$), chronic carriers ($I_c$), and recovered humans ($R$). Table 1 shows the model parameters and their description as they have been used in this work.

TABLE 1. Parameters and their description

| Parameter | Description |
|---|---|
| $b$ | recruitment rate in human due to births |
| $\mu$ | natural death rate of human |
| $\varepsilon$ | rate of infection in human |
| $\beta_a$ | transmission rate of acute HBV |
| $\beta_c$ | transmission rate of chronic HBV |
| $\gamma_a$ | recovery rate of acute HBV |
| $\gamma_c$ | recovery rate of chronic carrier HBV |
| $\omega$ | failure rate to clear acute HBV |
| $d$ | disease induced death rate of human |
| $q$ | vertical transmission rate in human |

The mode of transmission of HBV in human is shown by Figure 1. Several assumptions have been made in formulating this model as in Mpeshe and Nyerere [7], including following: $bq < \mu + d + \gamma_c$ so that carriers would not increase rapidly; $\beta_a < \beta_c$ because many infected individuals are likely to be unaware of their condition and hence continue with their regular behaviour; acute may become chronic carriers if they fail to clear the infection, and that a chronic carrier mother may give birth to a chronic carrier child; both acutely and chronically infected individuals can transmit HBV; and screening and treatment may help some chronic carriers to recover.

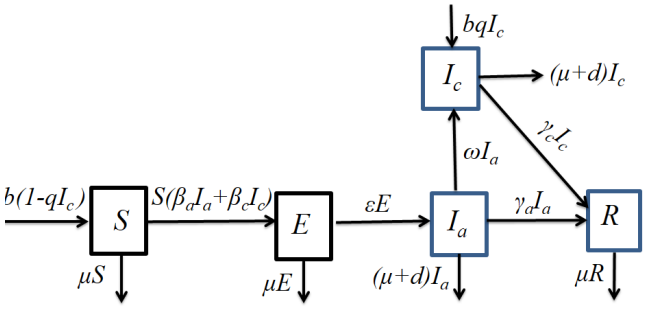



FIGURE 1. Flow diagram for the HPB model

Using the parameters in Table 1 and Figure 1, an SEIR model is derived using first order nonlinear ordinary differential equations as follows:

$$\frac{dS}{dt} = b(1 - qI_c) - \mu S - (\beta_a I_a + \beta_c I_c)S, \tag{1a}$$

$$\frac{dE}{dt} = (\beta_a I_a + \beta_c I_c)S - (\varepsilon + \mu)E, \tag{1b}$$

$$\frac{dI_a}{dt} = \varepsilon E - (\mu + d + \omega + \gamma_a)I_a, \tag{1c}$$

$$\frac{dI_c}{dt} = bqI_c + \omega I_a - (\mu + d + \gamma_c)I_c, \tag{1d}$$

$$\frac{dR}{dt} = \gamma_c I_c + \gamma_a I_a - \mu R. \tag{1e}$$

The fractional-order derivative model using Caputo derivative is now defined as

$$
\begin{aligned}
&{}_0^C D_t^\alpha S = b(1-qI_c) - \mu S - (\beta_a I_a + \beta_c I_c)S, && \text{(2a)}\\
&{}_0^C D_t^\alpha E = (\beta_a I_a + \beta_c I_c)S - (\varepsilon+\mu)E, && \text{(2b)}\\
&{}_0^C D_t^\alpha I_a = \varepsilon E - (\mu + d + \omega + \gamma_a)I_a, && \text{(2c)}\\
&{}_0^C D_t^\alpha I_c = bqI_c + \omega I_a - (\mu + d + \gamma_c)I_c, && \text{(2d)}\\
&{}_0^C D_t^\alpha R = \gamma_c I_c + \gamma_a I_a - \mu R. && \text{(2e)}
\end{aligned}
$$

where ${}_0^C D_t^\alpha$ is the Caputo derivative of order $\alpha \in (0,1)$, with initial conditions $S(0) = S_0 \geq 0$, $E(0) = E_0 \geq 0$, $I_a(0) = I_{a0} \geq 0$, $I_c(0) = I_{c0} \geq 0$, $R(0) = R_0 \geq 0$. The order $\alpha$ indicates the index of memory in the system.

The fuzzy fractional-order derivative model in Caputo sense is now defined as

$$
\begin{aligned}
&{}_0^C D_t^\alpha \tilde{S} = b(1-q\tilde{I}_c) - \mu\tilde{S} - (\beta_a \tilde{I}_a + \beta_c \tilde{I}_c)\tilde{S}, && \text{(3a)}\\
&{}_0^C D_t^\alpha \tilde{E} = (\beta_a \tilde{I}_a + \beta_c \tilde{I}_c)\tilde{S} - (\varepsilon+\mu)\tilde{E}, && \text{(3b)}\\
&{}_0^C D_t^\alpha \tilde{I}_a = \varepsilon\tilde{E} - (\mu + d + \omega + \gamma_a)\tilde{I}_a, && \text{(3c)}\\
&{}_0^C D_t^\alpha \tilde{I}_c = bq\tilde{I}_c + \omega\tilde{I}_a - (\mu + d + \gamma_c)\tilde{I}_c, && \text{(3d)}\\
&{}_0^C D_t^\alpha \tilde{R} = \gamma_c \tilde{I}_c + \gamma_a \tilde{I}_a - \mu\tilde{R}. && \text{(3e)}
\end{aligned}
$$

with initial conditions $S(0) = \tilde{k}(r)S_0$, $E(0) = \tilde{k}(r)E_0$, $I_a(0) = \tilde{k}(r)I_{a0}$, $I_c(0) = \tilde{k}(r)I_{c0}$, $R(0) = \tilde{k}(r)R_0$, and $\tilde{k}(r) = (u_l(r), u_c(r)) = (r-1, 1-r)$ for $0 \leq r \leq 1$.

2.2. **Feasible Region and Equilibria.** To show that the model solution exist and is in the positive region $\mathbb{R}_+^5$, we show that ${}_0^C D_t^\alpha x_i \geq 0$ in the region $\mathbb{R}_+^5$. Using the model system (3) we have

$$
\begin{aligned}
&{}_0^C D_t^\alpha \tilde{S}|_{\tilde{S}=0} = b > 0, && \text{(4a)}\\
&{}_0^C D_t^\alpha \tilde{E}|_{\tilde{E}=0} = (\beta_a \tilde{I}_a + \beta_c \tilde{I}_c)\tilde{S} \geq 0, && \text{(4b)}\\
&{}_0^C D_t^\alpha \tilde{I}_a|_{\tilde{I}_a=0} = \varepsilon\tilde{E} \geq 0, && \text{(4c)}\\
&{}_0^C D_t^\alpha \tilde{I}_c|_{\tilde{I}_c=0} = \omega\tilde{I}_a \geq 0, && \text{(4d)}\\
&{}_0^C D_t^\alpha \tilde{R}|_{\tilde{R}=0} = \gamma_c \tilde{I}_c + \gamma_a \tilde{I}_a \geq 0. && \text{(4e)}
\end{aligned}
$$

Hence, the model solution is feasible and positive in $\Omega = (\tilde{S}, \tilde{E}, \tilde{I}_a, \tilde{I}_c, \tilde{R}) \geq 0 \in \mathbb{R}_+^5$.

Further more, from (3) we find that

$$
{}_0^C D_t^\alpha \tilde{N} = b - \mu\tilde{N} - d(\tilde{I}_a + \tilde{I}_c) \leq b - \mu\tilde{N}. \tag{5}
$$

Solving this inequality as $t \to \infty$ gives,

$$
\tilde{N}(t) \leq \frac{b}{\mu}. \tag{6}
$$

Hence, the model solution is positively invariant in $\mathbb{R}_+^5$, This means that the model solution will remain in the feasible region $\Omega$ if it starts in $\Omega$.

2.3. **Equilibrium Points and Basic Reproduction Number.** To determine the disease-free and endemic equilibrium points, we set the left-hand side of (3) equal to zero. For $\tilde{E} = \tilde{I}_a = \tilde{I}_c = \tilde{R} = 0$, the disease-free equilibrium is

$$E_0 = (\frac{b}{\mu}, 0, 0, 0, 0), \tag{7}$$

If $\tilde{E} \neq 0$, $\tilde{I}_a \neq 0$, $\tilde{I}_c \neq 0$, and $\tilde{R} \neq 0$, the endemic equilibrium is $E^* = (\tilde{S}^*, \tilde{E}^*, \tilde{I_a}^*, \tilde{I_c}^*, \tilde{R}^*)$ where,

$$\tilde{S}^* = \frac{(\varepsilon+\mu)(\mu+d+\omega+\gamma_a)(-bq+\mu+d+\gamma_c)}{\varepsilon\beta_a(-bq+\mu+d+\gamma_c)+\varepsilon\beta_c\omega}, \tag{8a}$$

$$\tilde{E}^* = \frac{1}{\varepsilon}(\mu+d+\omega+\gamma_a)\tilde{I}_a^*, \tag{8b}$$

$$\tilde{I}_a^* = \frac{(-bq+\mu+d+\gamma_c)(b-\mu\tilde{S}^*)}{bq\omega+\beta_a\tilde{S}^*(-bq+\mu+d+\gamma_c)+\beta_c\omega\tilde{S}^*}, \tag{8c}$$

$$\tilde{I}_c^* = \frac{\omega\tilde{I}_a^*}{-bq+\mu+d+\gamma_c} \tag{8d}$$

$$\tilde{R}^* = \frac{1}{\mu}(\gamma_c\tilde{I}_c^* + \gamma_a\tilde{I}_a^*) \tag{8e}$$

For $E^*$ to exist in the feasible region $\Omega$, the condition $0 < \tilde{S}^* < \frac{b}{\mu}$, or equivalently, $\frac{b}{\mu}\frac{1}{\tilde{S}^*} \geq 1$ is sufficiently necessary. Thus, define the basic reproduction number $\mathcal{R}_0$ by

$$\mathcal{R}_0 = \frac{b}{\mu}\frac{1}{\tilde{S}^*}, \tag{9}$$

then

$$\mathcal{R}_0 = \frac{b}{\mu}\frac{\varepsilon\beta_a(-bq+\mu+d+\gamma_c)+\varepsilon\beta_c\omega}{(\varepsilon+\mu)(\mu+d+\omega+\gamma_a)(-bq+\mu+d+\gamma_c)}, \tag{10}$$

## 3. Stability of the Disease-Free Equilibrium

In assessing the stability of equilibrium points, we omit from the analysis the equation involving $\tilde{R}$ for its value can be obtained when the values of $\tilde{S}$, $\tilde{E}$, $\tilde{I}_a$ and $\tilde{I}_c$ are known. Thus, we reduce the model system (3) to

$${}_0^C D_t^\alpha \tilde{S} = b(1-q\tilde{I}_c) - \mu\tilde{S} - (\beta_a\tilde{I}_a + \beta_c\tilde{I}_c)\tilde{S}, \tag{11a}$$

$${}_0^C D_t^\alpha \tilde{E} = (\beta_a\tilde{I}_a + \beta_c\tilde{I}_c)\tilde{S} - (\varepsilon+\mu)\tilde{E}, \tag{11b}$$

$${}_0^C D_t^\alpha \tilde{I}_a = \varepsilon\tilde{E} - (\mu+d+\omega+\gamma_a)\tilde{I}_a, \tag{11c}$$

$${}_0^C D_t^\alpha \tilde{I}_c = bq\tilde{I}_c + \omega\tilde{I}_a - (\mu+d+\gamma_c)\tilde{I}_c. \tag{11d}$$

with the feasible region $\Omega = \{(\tilde{S}, \tilde{E}, \tilde{I}_a, \tilde{I}_c) \geq 0 \in \mathbb{R}_+^4 : \tilde{S}+\tilde{E}+\tilde{I}_a+\tilde{I}_c \leq \frac{b}{\mu}.\}$

**Theorem 1.** *The disease-free equilibrium of the HBV model (11) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

*Proof.* In this case, we show that the Jacobian matrix $J(E_0)$ of the fuzzy fractional derivative HBV model (11) at $E_0 = (\frac{b}{\mu}, 0, 0)$ has negative eigenvalues. Further computations show that

$J(E_0)$ of the fuzzy fractional derivative HBV model at $E_0$ is

$$J(E_0) = \begin{bmatrix} -\mu & 0 & -\beta_a \frac{b}{\mu} & -bq - \beta_c \frac{b}{\mu} \\ 0 & -(\varepsilon+\mu) & \beta_a \frac{b}{\mu} & \beta_c \frac{b}{\mu} \\ 0 & \varepsilon & -(\mu+d+\omega+\gamma_a) & 0 \\ 0 & 0 & \omega & bq-(\mu+d+\gamma_c) \end{bmatrix} \tag{12}$$

From $J(E_0)$ we find that one of the eigenvalue is $\lambda_1 = -\mu$. The remaining eigenvalues are the eigenvalues of the reduced $3 \times 3$ matrix

$$J^*(E_0) = \begin{bmatrix} -(\varepsilon+\mu) & \beta_a \frac{b}{\mu} & \beta_c \frac{b}{\mu} \\ \varepsilon & -(\mu+d+\omega+\gamma_a) & 0 \\ 0 & \omega & bq-(\mu+d+\gamma_c) \end{bmatrix}. \tag{13}$$

Observe that the remaining matrix $J^*(E_0)$ is a Metzler stable matrix whose eigenvalues are all negative. Hence, the Jacobian matrix $J(E_0)$ has all its eigenvalues negative, and thus, the disease-free equilibrium is locally asymptotically stable. □

**Theorem 2.** *The disease-free equilibrium is of the HBV model (11) is globally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

*Proof.* In this case, we define a Lyapunov function $V$ and show that ${}_0^C D_t^\alpha V \leq 0$. Now, consider the Lyapunov function

$$V = \omega_1 \tilde{E} + \omega_2 \tilde{I}_a + \omega_3 \tilde{I}_c \tag{14}$$

The fuzzy fractional Caputo derivative of $V$ is then

$${}_0^C D_t^\alpha V = \omega_1 \, {}_0^C D_t^\alpha \tilde{E} + \omega_2 \, {}_0^C D_t^\alpha \tilde{I}_a + \omega_3 \, {}_0^C D_t^\alpha \tilde{I}_c \tag{15}$$

From the model system (11) we have

$$\begin{aligned} {}_0^C D_t^\alpha V &= \omega_1[(\beta_a \tilde{I}_a + \beta_c \tilde{I}_c)\tilde{S} - (\varepsilon+\mu)\tilde{E}] + \omega_2[\varepsilon\tilde{E} - (\mu+d+\omega+\gamma_a)\tilde{I}_a] \\ &\quad + \omega_3[bq\tilde{I}_c + \omega\tilde{I}_a - (\mu+d+\gamma_c)\tilde{I}_c] \\ &\leq \omega_1[(\beta_a \tilde{I}_a + \beta_c \tilde{I}_c)\tilde{S}^* - (\varepsilon+\mu)\tilde{E}] + \omega_2[\varepsilon\tilde{E} - (\mu+d+\omega+\gamma_a)\tilde{I}_a] \\ &\quad + \omega_3[bq\tilde{I}_c + \omega\tilde{I}_a - (\mu+d+\gamma_c)\tilde{I}_c] \\ &= \omega_1\beta_a\tilde{I}_a\tilde{S}^* + \omega_3\omega\tilde{I}_a - (\mu+d+\omega+\gamma_a)\tilde{I}_a - \omega_1(\varepsilon+\mu)\tilde{E} \\ &\quad + \omega_2\varepsilon\tilde{E} + \omega_1\beta_c\tilde{I}_c\tilde{S}^* - \omega_3(-bq+\mu+d+\gamma_c)\tilde{I}_c \end{aligned} \tag{16}$$

If we choose $\omega_1 = \varepsilon(-bq+\mu+d+\gamma_c)$, $\omega_2 = (\varepsilon+\mu)(-bq+\mu+d+\gamma_c)$, $\omega_3 = \varepsilon\beta_c\tilde{S}^*$, and simplify the equation, we have

$$\begin{aligned} {}_0^C D_t^\alpha V &\leq \varepsilon(-bq+\mu+d+\gamma_c)\beta_a\tilde{I}_a\tilde{S}^* + \varepsilon\beta_c\omega\tilde{I}_a\tilde{S}^* \\ &\quad - (\varepsilon+\mu)(-bq+\mu+d+\gamma_c)(\mu+d+\omega+\gamma_a)\tilde{I}_a \\ &= (\varepsilon+\mu)(-bq+\mu+d+\gamma_c)(\mu+d+\omega+\gamma_a)\tilde{I}_a(\mathcal{R}_0 - 1) \end{aligned} \tag{17}$$

Since at disease-free equilibrium $\mathcal{R}_0 < 1$, then it follows that ${}_0^C D_t^\alpha V \leq 0$. Thus, the disease-free equilibrium is globally asymptotically stable at $\mathcal{R}_0 < 1$. □

## 4. Stability of the Endemic Equilibrium

The local stability of the disease-free equilibrium implies that the endemic equilibrium is also locally stable [20–22]. Therefore, in this section, we only establish the global stability of the endemic equilibrium.

**Theorem 3.** *The endemic equilibrium of the HBV model (11) is globally asymptotically stable if $\mathcal{R}_0 > 1$ and unstable if $\mathcal{R}_0 < 1$.*

*Proof.* To prove this theorem, we apply the Lyapunov functional approach for fractional differential equations.

**Definition 4** ( [21]). *Let $V(x_i(t)) = \sum_i^n \omega_i \phi_i(x_i(t))$ be a $C^1$ function defined on some domain in $\mathbb{R}_+^n$ and $x_i(t)$ is a solution of the model system ${}_0^C D_t^\alpha x_i(t) = f(x_i(t))$, $x_i(0) = x_0$, $\alpha \in (0, 1)$. Then the Caputo derivative of $V$ along $x_i(t)$ is given by*

$$ {}_0^C D_t^\alpha V(x_i(t)) = \sum_i^n \omega_i \, {}_0^C D_t^\alpha \phi_i(x_i(t)). \tag{18}$$

From the Definition 4, define $\phi_i(x_i(t))$ by $\phi_i(x_i(t)) = x_i(t) - x_i^* - x_i^* \ln \frac{x_i(t)}{x_i^*}$ where $x^*$ is the equilibrium point of the model system (11). Then, we have the following corollary:

**Corollary 5** ( [21]). *Let $x(t) \in \mathbb{R}^+$ be a continuous differentiable function . Then, for any $t \geq 0$, $\alpha \in (0, 1)$, and $x^* \geq 0$, we have*

$$\begin{aligned} {}_0^C D_t^\alpha V(x_i(t)) &= \sum_i^n {}_0^C D_t^\alpha (x_i(t) - x_i^* - x_i^* \ln \frac{x_i(t)}{x_i^*}) \\ &\leq \sum_i^n \omega_i (1 - \frac{x_i^*}{x_i(t)}) {}_0^C D_t^\alpha x_i(t). \end{aligned} \tag{19}$$

Now, consider the Lyapunov function

$$\begin{aligned} V = \omega_1 (\tilde{S} - \tilde{S}^* - \tilde{S}^* \ln \frac{\tilde{S}}{\tilde{S}^*}) + \omega_2 (\tilde{E} - \tilde{E}^* - \tilde{E}^* \ln \frac{\tilde{E}}{\tilde{E}^*}) \\ + \omega_3 (\tilde{I}_a - \tilde{I}_a{}^* - \tilde{I}_a{}^* \ln \frac{\tilde{I}_a}{\tilde{I}_a{}^*}) + \omega_4 (\tilde{I}_c - \tilde{I}_c{}^* - \tilde{I}_c{}^* \ln \frac{\tilde{I}_c}{\tilde{I}_c{}^*}). \end{aligned} \tag{20}$$

Applying the Definition 4 and Corollary 5, the Caputo derivative of $V$ is then

$$\begin{aligned} {}_0^C D_t^\alpha V \leq \omega_1 (1 - \frac{\tilde{S}^*}{\tilde{S}}) {}_0^C D_t^\alpha \tilde{S} + \omega_2 (1 - \frac{\tilde{E}^*}{\tilde{E}}) {}_0^C D_t^\alpha \tilde{E} + \omega_3 (1 - \frac{\tilde{I}_a{}^*}{\tilde{I}_a}) {}_0^C D_t^\alpha \tilde{I}_a \\ + \omega_4 (1 - \frac{\tilde{I}_c{}^*}{\tilde{I}_c}) {}_0^C D_t^\alpha \tilde{I}_c. \end{aligned} \tag{21}$$

From the model system (11) we have

$$\begin{aligned} {}_0^C D_t^\alpha V \leq \omega_1 (1 - \frac{\tilde{S}^*}{\tilde{S}}) [b(1 - q\tilde{I}_c) - \mu\tilde{S} - (\beta_a \tilde{I}_a + \beta_c \tilde{I}_c)\tilde{S}] \\ + \omega_2 (1 - \frac{\tilde{E}^*}{\tilde{E}}) [(\beta_a \tilde{I}_a + \beta_c \tilde{I}_c)\tilde{S} - (\varepsilon + \mu)\tilde{E}] \\ + \omega_3 (1 - \frac{\tilde{I}_a{}^*}{\tilde{I}_a}) [\varepsilon \tilde{E} - (\mu + d + \omega + \gamma_a)\tilde{I}_a] \\ + \omega_4 (1 - \frac{\tilde{I}_c{}^*}{\tilde{I}_c}) [bq\tilde{I}_c + \omega \tilde{I}_a - (\mu + d + \gamma_c)\tilde{I}_c]. \end{aligned} \tag{22}$$

At endemic equilibrium, $b = bq\tilde{I}_c^* - \mu\tilde{S}^* - (\beta_a\tilde{I}_a^* + \beta_c\tilde{I}_c^*)\tilde{S}^*$, $(\varepsilon+\mu) = (\beta_a\tilde{I}_a^* + \beta_c\tilde{I}_c^*)\frac{\tilde{S}^*}{\tilde{E}^*}$, $(\mu + d + \omega + \gamma_a) = \varepsilon\frac{\tilde{E}^*}{\tilde{I}_a{}^*}$, and $-bq + (\mu + d + \gamma_c) = \omega\frac{\tilde{I}_a^*}{\tilde{I}_c^*}$. Thus,

$$
\begin{aligned}
{}_0^C D_t^\alpha V \leq\ & \omega_1(1-\frac{\tilde{S}^*}{\tilde{S}})[bq\tilde{I}_c^* - \mu\tilde{S}^* - (\beta_a\tilde{I}_a^* + \beta_c\tilde{I}_c^*)\tilde{S}^* - bq\tilde{I}_c - \mu\tilde{S} - (\beta_a\tilde{I}_a + \beta_c\tilde{I}_c)\tilde{S}] \\
& + \omega_2(1-\frac{\tilde{E}^*}{\tilde{E}})[(\beta_a\tilde{I}_a + \beta_c\tilde{I}_c)\tilde{S} - (\beta_a\tilde{I}_a^* + \beta_c\tilde{I}_c^*)\frac{\tilde{S}^*}{\tilde{E}^*}\tilde{E}] \\
& + \omega_3(1-\frac{I_a^*}{I_a})[\varepsilon\tilde{E} - \varepsilon\frac{\tilde{E}^*}{\tilde{I}_a{}^*}\tilde{I}_a] + \omega_4(1-\frac{\tilde{I}_a{}^*}{\tilde{I}_c})[\omega\tilde{I}_a - \omega\frac{\tilde{I}_a^*}{\tilde{I}_c^*}\tilde{I}_c].
\end{aligned} \tag{23}
$$

Further simplification gives

$$
{}_0^C D_t^\alpha V \leq -\omega_1\mu\tilde{S}(1-\frac{\tilde{S}^*}{\tilde{S}})^2 + F(P) \tag{24}
$$

where $P = (\tilde{S},\ \tilde{E},\ \tilde{I}_a,\ \tilde{I}_c) \geq 0$ and

$$
\begin{aligned}
F(P) =\ & \omega_1(1-\frac{\tilde{S}^*}{\tilde{S}})(1-\frac{\tilde{I}_c}{\tilde{I}_c^*})bq\tilde{I}_c^* \\
& + [\omega_1(1-\frac{\tilde{S}^*}{\tilde{S}})(1-\frac{\tilde{S}\tilde{I}_a}{\tilde{S}^*\tilde{I}_a^*}) + \omega_2(1-\frac{\tilde{E}^*}{\tilde{E}})(\frac{\tilde{S}\tilde{I}_a}{\tilde{S}^*\tilde{I}_a^*} - \frac{\tilde{E}}{\tilde{E}^*})]\beta_a\tilde{I}_a^*\tilde{S}^* \\
& + [\omega_1(1-\frac{\tilde{S}^*}{\tilde{S}})(1-\frac{\tilde{S}\tilde{I}_c}{\tilde{S}^*\tilde{I}_c^*}) + \omega_2(1-\frac{\tilde{E}^*}{\tilde{E}})(\frac{\tilde{S}\tilde{I}_c}{\tilde{S}^*\tilde{I}_c^*} - \frac{\tilde{E}}{\tilde{E}^*})]\beta_c\tilde{I}_c^*\tilde{S}^* \\
& + \omega_3(1-\frac{\tilde{I}_a{}^*}{\tilde{I}_a})(\frac{\tilde{E}}{\tilde{E}^*} - \frac{\tilde{I}_a}{\tilde{I}_a^*})\varepsilon\tilde{E}^* + \omega_4(1-\frac{\tilde{I}_c^*}{\tilde{I}_c})(\frac{\tilde{I}_a}{\tilde{I}_a^*} - \frac{\tilde{I}_c}{\tilde{I}_c^*})\omega\tilde{I}_a{}^*
\end{aligned} \tag{25}
$$

If we choose $\omega_2 = \omega_1$, $\omega_3 = \omega_1\frac{\beta_a\tilde{I}_a^*\tilde{S}^*}{\varepsilon\tilde{E}^*}$, and $\omega_4 = \omega_1\frac{\beta_c\tilde{I}_c^*\tilde{S}^*}{\omega\tilde{I}_a{}^*}$, and simplify further, gives

$$
\begin{aligned}
F(P) =\ & \omega_1(1-\frac{\tilde{S}^*}{\tilde{S}})(1-\frac{\tilde{I}_c}{\tilde{I}_c^*})bq\tilde{I}_c^* + \omega_1(3 - \frac{S^*}{S} - \frac{\tilde{I}_a^*\tilde{E}^*}{\tilde{I}_a\tilde{E}} - \frac{\tilde{S}\tilde{I}_a\tilde{E}^*}{\tilde{S}^*\tilde{I}_a^*\tilde{E}})\beta_a\tilde{I}_a^*\tilde{S}^* \\
& + \omega_1(3 - \frac{\tilde{S}^*}{\tilde{S}} - \frac{\tilde{E}}{\tilde{E}^*} - \frac{\tilde{I}_a}{\tilde{I}_a^*}(\frac{\tilde{I}_c^*}{\tilde{I}_c} - 1) - \frac{\tilde{S}\tilde{I}_c\tilde{E}^*}{\tilde{S}^*\tilde{I}_c^*\tilde{E}})\beta_c\tilde{I}_c^*\tilde{S}^*
\end{aligned} \tag{26}
$$

In general $(1-\frac{x^*}{x}) \geq 0$ and $(1-\frac{x}{x^*}) \leq 0$ if the $x^*$ is the equilibrium point, therefore, $((1-\frac{\tilde{S}^*}{\tilde{S}})(1-\frac{\tilde{I}_c}{\tilde{I}_c^*})) \leq 0$. Using the property of arithmetic mean ensures that $F(P)$ is non-positive in $P$. Thus, ${}_0^C D_t^\alpha V \leq 0$ in $P$ and is zero when $P = P^*$. Following the LaSalle's invariant principle, it is concluded that $P^*$ is globally asymptotically stable. □

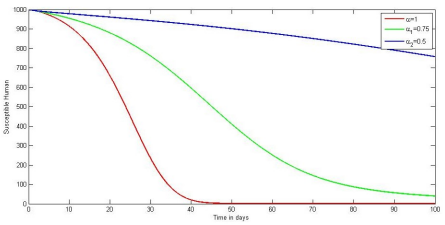
## 5. Numerical Simulations

In this section, numerical simulation are carried out using parameter values given in Table 2. Numerical simulation help to study the persistence of the disease when introduced in a closed or isolated system. The initial values used in to simulate the fractional-order model are $S = 1000$, $E = 10$, $I_a = 10$, $I_c = 1$, and $R = 1$. For the fuzzy fractional-order model $S(0) = E(0) = I_a(0) = I_c(0) = R(0) = 1$. According to WHO [1], the incubation period is 30 to 180 days. For the purpose of simulation we use 30 days and therefore, $\varepsilon = 1/30$ per day. According to the Southern Cross Medical Library (SCML), the recory from acute HBV infection is 4 to 8 weeks or several months. For the purpose of simulation we use 4 weeks and therefore, $\gamma_a = 1/28$ per day. The parameters and their sources are shown in the Table 2.

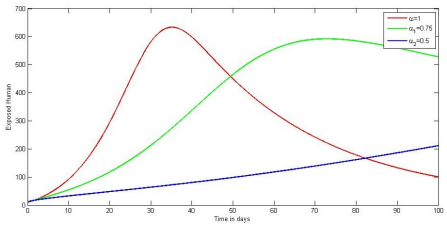
TABLE 2. Parameters and their description

| Parameter | Description | Range value | Source |
|---|---|---|---|
| $b$ | birth rate in human | 1 | assumed |
| $\varepsilon$ | rate of infection | 1/30 | [1] |
| $\mu$ | natural death rate of human | 0.00004521 | [22] |
| $d$ | disease induced death rate of human | 0.0013 | [23] |
| $\beta_a$ | transmission rate of acute HBV | 0.00015 | [7] |
| $\beta_c$ | transmission rate of chronic HBV | 0.0025 | [7] |
| $\gamma_a$ | recovery rate of acute HBV | 1/28 | [24] |
| $\gamma_c$ | recovery rate of chronic carrier HBV | 1/180 | assumed |
| $\omega$ | failure rate to clear acute HBV | 0.05 | [7] |
| $q$ | vertical transmission rate in human | 0.001 | [7] |

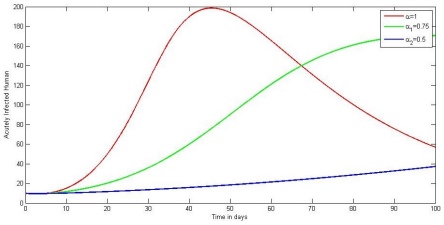
The graph in Figure 2 shows the dynamical behaviour of state variables for different values of fractional order derivative $\alpha$ for time over 100 days. From Figure 2a, we observe that the population of susceptible individuals decreases as the value of order ($\alpha$) increases. However, there is a rapid increase in exposed individuals in the first 50 days for $\alpha = 1$, surpassing all orders, and then, it falls as shown in Figure 2b. The same results are exhibited for acute and chronic individuals as in Figure 2c and 2d. The population of recovered individuals appear to increase as $\alpha$ increases as shown in Figure 2e.



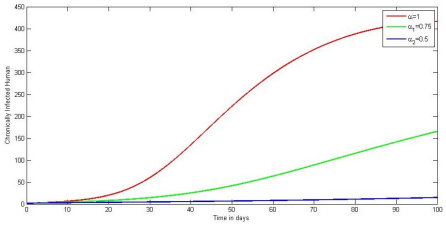
(A) Susceptible



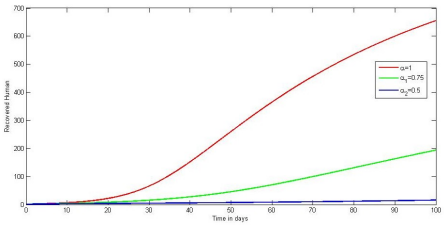
(B) Exposed



(C) Acute



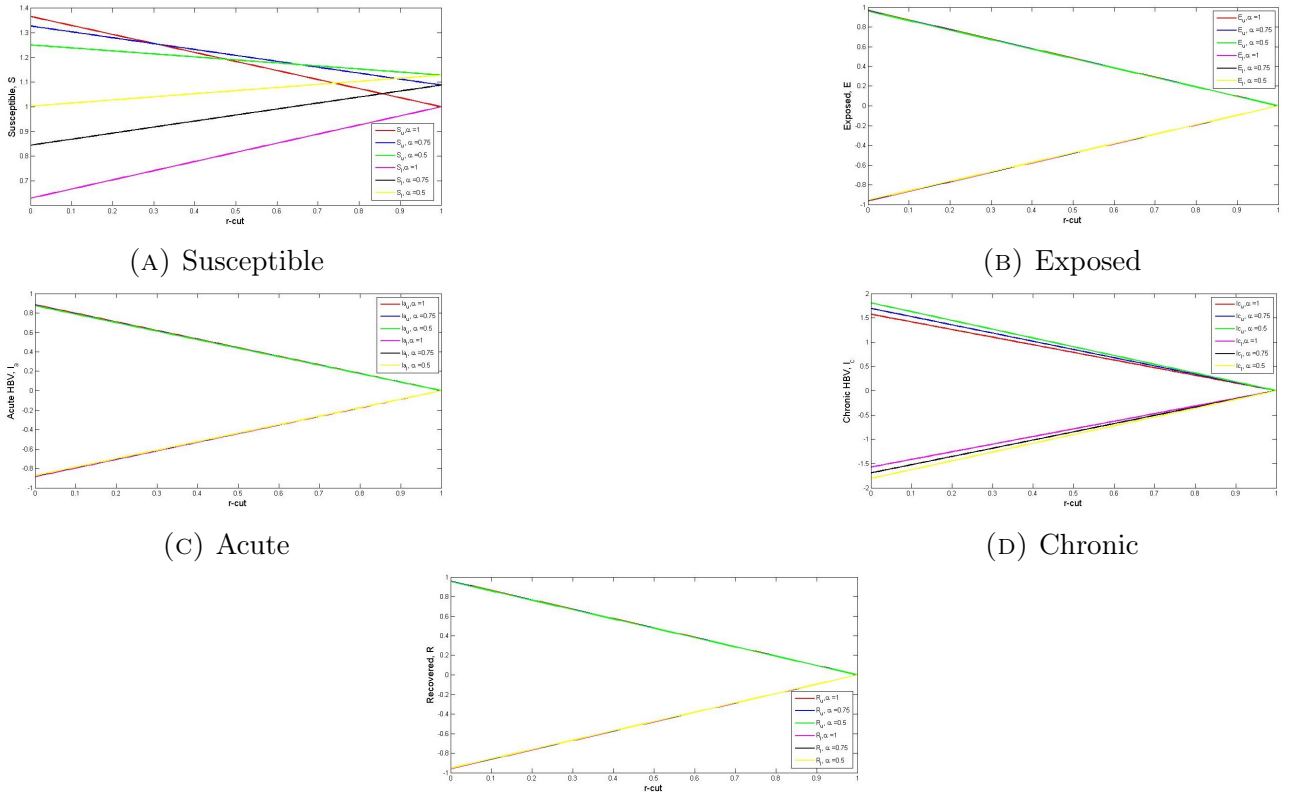
(D) Chronic



(E) Recovered

FIGURE 2. Three simple graphs

The graph in Figure 3, shows the dynamical behaviour of state variables for different values of the fuzzy fractional order derivative model to the variation of $r$-cut. It can be observed that the obtained graphs are fuzzy triangular functions. A variation in fractional order $\alpha$ also affects the dynamics of the fuzzy fractional model. When the order increases from 0.5 to 1, the diameter of the fuzzy solution for susceptible individuals decreases, while that of chronic individuals increases as in Figure 3a and 3d. The diameter for the exposed, acute and recovered individuals is almost the same as shown in Figure 3b, 3c, and 3e. When $\alpha = 1$, then the solution curves converge to the curves of integer order model.




(A) Susceptible

(B) Exposed

(C) Acute

(D) Chronic

(E) Recovered

FIGURE 3. Three simple graphs

## 6. DISCUSSION

In this paper, we used a fuzzy fractional order modeling approach in Caputo sense to investigate the dynamics of HBV infection. To study the effect of initial transmission of the disease we computed the basic reproduction number $\mathcal{R}_0$ of the model and used it to analyse the stability of the disease equilibrium points. Analysis of the equilibrium points indicate that the disease-free equilibrium of the model is globally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable otherwise. This means that there is a possibility of controlling the disease provided that $\mathcal{R}_0 < 1$. We also found that the disease-endemic equilibrium is globally asymptotically stable when $\mathcal{R}_0 > 1$, showing that the disease when introduced in the population can persist for a long time.

To analyse the variation of sub-population in the model with respect to time we performed numerical simulations for both the fractional order model and the fuzzy fractional-order model at different values of the order $\alpha$. The results of numerical simulations of the fractional-order

model as in Figure 2 show that, whenever there is an increase in $\alpha$ the susceptible and exposed population decreases, while the population of acute, chronic, and recovered individuals increases as $\alpha$ increases. The results of numerical simulations of the fuzzy fractional-order model as shown as in Figure 3 exhibit fuzzy triangular solutions with increase in diameter as $\alpha$ decreases for the susceptible individuals, while for chronic individuals the diameter increases as $\alpha$ increases.

## 7. Conclusion

HBV infection will remain a potential threat to many countries in the world because of its nature of infection. The virus can cause chronic infection and set people at high risk of death from cirrhosis and liver cancer. HBV infection occur in fuzzy environment, and hence placing much concern to application of fuzziness in modeling its dynamics. The results of the analysis show that the disease is persistent when introduced in the society causing threat to human health. Effective educational campaign about the dynamical transmission of the disease will help to make people on safe motherhood practices and the importance of attending clinics for screening and treatment where possible.

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