Infectious Disease Modelling 10 (2025) 1019–1036

Contents lists available at ScienceDirect

Infectious Disease Modelling

journal homepage: www.keaipublishing.com/idm

The role of spontaneous clearance on fractional analysis of HBV

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ARTICLE INFO

Article history:
Received 29 January 2025
Received in revised form 13 May 2025
Accepted 23 May 2025
Available online 27 May 2025
Handling Editor: Dr Yiming Shao

Keywords:
Hepatitis B virus
Fractal fractional model
Fixed point theory
Memory-dependent
Basic reproduction number
Stability

ABSTRACT

Hepatitis B virus (HBV) remains a persistent global health concern, with recent research advancing our understanding of its transmission dynamics and potential interventions. The present study proposes a mathematical model of Hepatitis B Virus (HBV) epidemics using fractional calculus, with a special emphasis on the influence of spontaneous clearance across diverse population groups. Using the Atangana-Baleanu derivative, the model accounts for the complications of vertical and horizontal transmission, therapy, immunisation, and spontaneous clearance. Numerical simulations with different fractional orders demonstrate how spontaneous clearance affects the dynamics of susceptible, chronic, treated, and recovered populations. The findings indicate that in vulnerable populations, increasing spontaneous clearance reduces vulnerability because people either clear the illness naturally or gain resistance.However, in chronic populations, spontaneous clearance is insufficient for complete recovery without treatment. The combination of therapy and spontaneous clearance improves the treated population, demonstrating the beneficial effects of both medical intervention and natural immunity. Furthermore, increased spontaneous clearance boosts the restored population, demonstrating the immune system's ability to eliminate the virus over time. The fractional-order framework captures the memory effect of illness development, revealing how healing is time-dependent and how immune responses have a long-term impact. This study emphasises the need of combining spontaneous clearance with medical therapies to improve HBV management and public health consequences. Hepatitis B virus (HBV) remains a persistent global health concern, with recent research advancing our understanding of its transmission dynamics and potential interventions. This study presents a fractional mathematical model of HBV infection, employing the Atangana-Baleanu derivative with Mittag-Leffler kernels to capture memory-dependent and nonlocal transmission processes. The model integrates vertical and horizontal transmission pathways, treatment strategies, immunization efforts, and spontaneous clearance, providing a nuanced perspective compared to classical models. Stability conditions are analyzed through fixed-point theory, revealing the global stability of both disease-free and endemic states under specific values of the basic reproduction number R_0. Numerical simulations demonstrate the model's effectiveness in capturing the complex dynamics of HBV, with fractional-order parameters enhancing prediction accuracy. This approach offers valuable insights

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Peer review under the responsibility of KeAi Communications Co., Ltd.

https://doi.org/10.1016/j.idm.2025.05.009

into optimizing public health interventions and treatment strategies for managing HBV infections effectively.

1. Introduction

Hepatitis B virus (HBV) remains a persistent global health concern, and recent literature has significantly advanced our understanding of the virus, its transmission dynamics, and potential interventions. Key studies have shed light on various aspects, including molecular epidemiology, transmission patterns, and advancements in therapeutic approaches. Recent research has employed molecular epidemiology to characterize the genetic diversity of HBV strains, providing valuable insights into transmission patterns and the evolution of the virus. Studies by (Kramvis & Genotypes, 2014; Liu et al., 2021) have contributed to our understanding of HBV genotypes, variants, and their implications for disease progression.

Advancements in understanding the transmission dynamics of HBV have informed preventive strategies. Studies by (Ott et al., 2012) and (Lee & Gong, 2018) have explored the global epidemiology of HBV, emphasizing the importance of region-specific prevention efforts and vertical transmission challenges. Meanwhile (Van Herck et al., 2018), has highlighted the role of vaccination in specific age groups as a critical preventive measure. The application of mathematical models to study the spread of HBV has gained prominence (Liu et al., 2019a). utilized fractional-order modeling to capture the complexities of HBV transmission, considering factors such as vertical and horizontal transmission, treatment response, immunization effects, and spontaneous clearance. These models contribute to predicting the impact of interventions and informing public health policies.

Recent literature has also focused on therapeutic approaches for managing HBV infections. Studies by (Lampertico et al., 2017) and (Yuen et al., 2018) have explored novel antiviral treatments and addressed challenges such as drug resistance and long-term outcomes. These advancements are crucial in improving patient outcomes and reducing the burden of chronic HBV infections.

As Hepatitis B virus (HBV) continues to pose a substantial global health burden, affecting millions and contributing to severe liver-related complications. A comprehensive understanding of HBV dynamics necessitates an exploration of not only the traditional vertical and horizontal transmission pathways but also the intricate interactions involving treatment, immunization, and spontaneous clearance. Mathematical modeling, a powerful tool in epidemiology, offers a unique opportunity to unravel these complexities and inform evidence-based interventions.

The mathematical modeling of infectious diseases, particularly within the framework of fractional calculus, has become increasingly relevant in recent years. This holds for the study of the Hepatitis B virus (HBV), a global health concern affecting millions worldwide. The utilization of fractional calculus offers a powerful tool to capture the complex dynamics of HBV infection, encompassing aspects such as transmission, immune response, and treatment.

The traditional models of infectious diseases, often based on ordinary differential equations (ODEs), have been instrumental in understanding general transmission patterns (Kermack & McKendrick, 1927). However, these models have limitations in capturing the intricate dynamics of diseases characterized by memory-dependent processes, non-local interactions, and complex heterogeneities in the host population (Podlubny, 1999). In response to these challenges, fractional calculus has emerged as a versatile and effective mathematical framework for modeling the complexities of infectious diseases.

The application of fractional calculus to HBV dynamics allows for a more accurate representation of the virus's behaviour within the host population. Atangana-Baleanu fractional derivatives, introduced by (Atangana & Baleanu, 2016), present a novel approach to describe memory-dependent processes with non-local and non-singular kernel properties. These derivatives have shown promise in various scientific fields and provide a unique avenue for capturing the nuances of HBV dynamics. Studies have successfully employed fractional-order models to investigate the spread of HBV within populations, considering factors such as vertical and horizontal transmission pathways (Liu et al., 2019b). These models offer a more realistic representation of the infection's dynamics, considering the fractional nature of the processes involved in transmission and recovery. Additionally, the use of fractional calculus in modeling the immune response to HBV infection has provided insights into the memory effects inherent in the host's defence mechanisms (Liu & Liao, 2018). demonstrated the application of fractional calculus in capturing the complexities of the immune response and the subsequent impact on the overall dynamics of HBV infection. Furthermore, the incorporation of treatment strategies within fractional models adds another layer of realism to the understanding of HBV dynamics. Treatment interventions, such as antiviral therapies, have been integrated into fractional models to assess their effectiveness in controlling the spread of the virus within populations (Sun et al., 2019).

Traditional epidemiological models have significantly contributed to our comprehension of HBV transmission dynamics, emphasizing vertical transmission from mother to child and horizontal transmission within populations (Lavanchy, 2004). However, to comprehensively capture the full spectrum of HBV dynamics, it is imperative to extend these models to incorporate additional factors such as testing and treatment regimens, immune response, risk factors, and the phenomenon

of spontaneous clearance. Recent advancements in mathematical modeling, particularly in the context of fractional calculus, provide a framework to address this need. Fractional calculus enables the incorporation of memory-dependent processes, offering a more realistic portrayal of the complex interactions involved in infectious diseases (Baleanu et al., 2012).

In recent times, fractional calculus has found application across various domains in science and engineering. For example, fractional differential equations (FDE) are employed in fields such as robotics, economics, finance, potential field modeling, dam hydraulics, signal processing, control theory, heat transfer, diffusion problems, and the modeling of nonlocal epidemics. Several authoritative monographs on FDE and its diverse applications are detailed in references (Yavuz et al., 2023; iqbal et al., 2023a; Ain & Chu, 2023; Uçar, 2023; Ahmad et al., 2023; Khan et al., 2022; Liu et al., 2022; Shyamsunder et al., 2022; Din & Abidin, 2022; Simelane & Dlamini, 2021; Khan et al., 2021; Gul et al., 2021; Zhong et al., 2021; iqbal et al., 2023b; Oludoun et al., 2021).

The study conducted by (Yavuz et al., 2023) in 2023 explores various frameworks and perspectives utilized in the mathematical modeling of infectious diseases. It presents a novel modeling paradigm proven to be advantageous in modeling intricate scientific systems for biological purposes. The paper outlines essential methodologies, strategies, and connections among these approaches. Furthermore, it delves into the significance, fundamental characteristics, and methodologies of mathematical modeling. This theoretical examination offers valuable insights for researchers in epidemic and pandemic modeling (iqbal et al., 2023a). introduced a novel and impactful modeling approach to elucidate the behaviors of the Hepatitis-B virus. Their comprehensive study involved mathematical modeling, equilibrium analysis, stability assessment, and numerical simulations using the Adams–Bashforth numerical scheme. Additionally, they employed a parameter estimation method to determine model parameters, identify the optimal curve, and conducted stability and sensitivity analyses, revealing that the fractional derivative order significantly influences the dynamical process of the constructed Hepatitis-B model (iqbal et al., 2023a). explored the transmission of poliovirus in the human population, employing a classical model initially, which was subsequently transformed into a fractal fractional epidemic model with the existence of the solution ensured through fixed point theory, and the points of equilibria for the model were determined." In their 2022 paper (Ahmad et al., 2023), examined the temporal dynamics of hepatitis B, exploring the impact of various infectious periods. They introduced a model that considers the roles of acute and chronic infection stages, employing fractional theory and the Caputo–Fabrizio (CF) operator for fractionalization. The authors conducted qualitative analyses on both integer and fractional-order models, demonstrating the feasibility and uniqueness of solutions to the proposed fractional-order model to establish the epidemic problem's credibility (Din & Abidin, 2022). introduced a saturated incidence nonlinear fractional order model for Hepatitis B virus (HBV), employing Caputo fractional derivatives, and utilized the Adams-Bashforth-Moulton technique to numerically solve the epidemic model, investigating its dynamics, basic properties, and stability conditions through the fractional Routh-Hurwitz criterion and matrix algebra. The study by (Khan et al., 2021) examines the effects of spontaneous clearance in acute cases, recovery leading to full immunity in chronic cases, and reduction of risk factors on a model of hepatitis B virus (HBV). The findings reveal that treatment, spontaneous clearance, and risk factor reduction significantly influence the transmission and regulation of HBV. Through simulations, the study underscores the effectiveness of these measures, offering a robust control strategy for HBV transmission. In their 2021 study (Gul et al., 2021), developed a mathematical model to examine the acquisition and transmission dynamics of the hepatitis B virus, aiming to pinpoint strategies for mitigating its impact. The paper thoroughly explores the significance of testing and treatment in managing HBV dynamics. Their findings highlight the effectiveness of early testing during acute and chronic unaware phases for optimal virus management (Zhong et al., 2021). explored the dynamics of hepatitis B, examining various infection phases and multiple transmission routes. They devised a model using fractional calculus, employing the Caputo–Fabrizio operator for fractionalization. The study includes an examination of existence and uniqueness through fixed point theory, demonstrating that the proposed model exhibits a bounded and positive solution. The basic reproductive number for steady-state analysis was also determined, establishing the local and global asymptotic stability of the fractional-order epidemiological model under specific conditions Similarly (Oludoun et al., 2021), explored the development of the HBV model, incorporating the Caputo derivative for a more generalized approach; through fractional stability analysis, they established local asymptotic stability for disease-free scenarios with $R_0 < 1$ and demonstrated global asymptotic stability for the same condition. Additionally, the study revealed the emergence of an endemic equilibrium when $R_0 > 1$, indicating a distinctive and unique equilibrium point.

In this study, we employ Atangana-Baleanu fractional derivatives to model the diverse dynamics of HBV, encompassing vertical and horizontal transmission, treatment response, immunization effects, and spontaneous clearance.Traditional integer-order differential models describe the rate of change of biological processes assuming no memory, where the current rate of change depends solely on the present state. However, in real biological systems particularly chronic infections like HBV past states often exert a lingering influence. The fractional-order derivative, specifically the Atangana-Baleanu derivative with a non-singular Mittag-Leffler kernel, enables the incorporation of this memory effect. It models the immune system's capacity to remember past infections and treatments, which can influence current progression, spontaneous clearance, and treatment response. This makes the fractional-order approach particularly suitable for capturing the non local, time-dependent dynamics of HBV infection and recovery. Hence, our modeling approach integrates these components to offer a holistic understanding of HBV dynamics, considering the impact on prevalence in different age groups, the dynamics of transmission within populations, antiviral treatment effectiveness, the influence of immunization programs, and spontaneous clearance among infected individuals.

This research aims to develop a comprehensive mathematical model using Belanu-Atangana fractional derivatives to describe the intricate dynamics of Hepatitis B infection. Incorporating fractional calculus enhances prediction accuracy and captures the long-term behavior of the disease. The study explores the implications of fractional order on the model's stability and sensitivity, providing valuable insights into the underlying mechanisms of Hepatitis B progression.

2. Mathematical framework/fixed point reduction

The mathematical framework developed by (Oludoun et al., 2021), which introduced crucial dynamics for comprehending HBV epidemics, is expanded upon in this section. The Mittag-Leffler kernel is included in the analysis, allowing for a more accurate exploration of HBV outbreaks and uncovering insights. The goal is to create a better knowledge of the parameters driving HBV transmission and control by using the Mittag-Leffler kernel, which will ultimately result in more effective disease management and prevention methods.

The following equation system described by (Oludoun et al., 2021), is critically investigated using the fixed-point reduction technique. These equations capture the complex dynamics of HBV epidemics, including critical elements that influence disease propagation and population dynamics. The intricacy of HBV epidemic transmission is reduced to a more comprehensible form by employing this method, which enables a deeper understanding of the mechanisms causing epidemics. In addition to paving the path for more effective disease control and intervention techniques, this methodology clarifies the key factors of HBV transmission.

In this model ξ is the birth rate, α is the percentage of the population that has been successfully immunised, and γ is the likelihood that children born to carrier mothers would develop a chronic state, it is assumed that susceptible individual S enters the population at time t at a constant rate $\xi(1-\alpha)(1-\gamma_c)$. People die at the same natural mortality rate, μ, regardless of their class. People with HBV who are receiving therapy are thought to be non-contagious. The force of infecton of HBV is at a rate of λ_s. A fraction of acute HBV infected persons η become chronic carriers and require treatment at a rate σ, while the remaining proportion $1-\eta$ clear the virus on their own. The duration of the acute phase is represented by $\frac{1}{\omega}$. While the remaining proportion $1-\kappa$ becomes susceptible, a portion of the treated HBV individuals κ recover with full immunity, while some are in the process of recovering in the treated population at a rate of ν and treatment time is given as ρ. Individuals on treatment ρ can become infected again at a rate $\nu\rho$ if they stop receiving treatment at a rate ϵ, and those in the process of healing in the treated population at a rate ν if they engage in or are exposed to high-risk habits.

The following system of equations are considered for the fixed point reduction as introduced by (Oludoun et al., 2021)

$$\frac{dS}{dt} = \xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R \tag{2.1}$$

$$\frac{dA}{dt} = \lambda_s S - (\omega+\mu)A \tag{2.2}$$

$$\frac{dC}{dt} = \eta\omega A + \xi(1-\alpha)\gamma_c + (1-\nu)\rho T - (\sigma+\mu)C \tag{2.3}$$

$$\frac{dT}{dt} = \sigma C - (\rho+\mu)T \tag{2.4}$$

$$\frac{dR}{dt} = \xi\alpha + \kappa\nu\rho T - (\epsilon+\mu)R \tag{2.5}$$

Atangana-Baleanu derivatives, expressed as B integral, are applied to develop a mathematical model that captures the intricate dynamics of HBV epidemics. While the model uses Atangana-Baleanu fractional derivatives on the left-hand side to reflect memory-dependent evolution of each state variable, we retain conventional rate expressions on the right-hand side to maintain interpretability and parameter consistency. Each RHS term represents biologically instantaneous transitions (e.g., infection, recovery, treatment initiation). However, due to the action of the fractional operator, the overall system behavior inherently incorporates memory effects through convolution with the Mittag-Leffler kernel. This formulation provides a balance between biological realism and mathematical tractability. This system integrates essential factors influencing disease transmission and population behavior, providing a more accurate depiction of epidemic processes. The use of B-fractional derivatives allows for a deeper understanding of complex transmission patterns, offering valuable insights for designing more effective strategies for disease control and intervention. The resulting system of equations is as follows:

$${}^{B}D_t^{\nu} S(t) = \xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R \tag{2.6}$$

$${}^{B}D_t^{\nu} A(t) = \lambda_s S - (\omega+\mu)A \tag{2.7}$$

$$^{B}D_t^{\upsilon}C(t) = \eta\omega A + \xi(1-\alpha)\gamma_c + (1-\nu)\rho T - (\sigma+\mu)C \tag{2.8}$$

$$^{B}D_t^{\upsilon}T(t) = \sigma C - (\rho+\mu)T \tag{2.9}$$

$$^{B}D_t^{\upsilon}R(t) = \xi\alpha + \kappa\nu\rho T - (\epsilon+\mu)R \tag{2.10}$$

These equations are subject to the subsequent initial conditions:

$$S = S_0 \geq 0, A = A_0 \geq 0, C = C_0 \geq 0, T = T_0 \geq 0, R = R_0 \geq 0 \forall \in (0,1].$$

The model's degree of freedom is increased for improved curve fitting by integrating the fractional order parameters. The properties of this fractional order differential operator aid in future prediction and in moderating the dynamics of disease.

The goal of this study is to identify the solutions for the system (2.1)–(2.10). Therefore, the system is defined as follows:

$$Q_1(t,S,A,C,T,R) = \xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R \tag{2.11}$$

$$Q_2(t,S,A,C,T,R) = \lambda_s S - (\omega+\mu)A \tag{2.12}$$

$$Q_3(t,S,A,C,T,R) = \eta\omega A + \xi(1-\alpha)\gamma_c + (1-\nu)\rho T - (\sigma+\mu)C \tag{2.13}$$

$$Q_4(t,S,A,C,T,R) = \sigma C - (\rho+\mu)T \tag{2.14}$$

$$Q_5(t,S,A,C,T,R) = \xi\alpha + \kappa\nu\rho T - (\epsilon+\mu)R \tag{2.15}$$

The convergent set of equations is then obtained by using the B integral as demonstrated in (iqbal et al., 2023b).

$$S(t) - S(0) = \frac{1-\upsilon}{B(\upsilon)}Q_1(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)} \times \int_0^t (t-\varphi)^{(\upsilon-1)} Q_1(\varphi,S,A,C,T,R)d\varphi \tag{2.16}$$

$$A(t) - A(0) = \frac{1-\upsilon}{B(\upsilon)}Q_2(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)} \times \int_0^t (t-\varphi)^{(\upsilon-1)} Q_2(\varphi,S,A,C,T,R)d\varphi \tag{2.17}$$

$$C(t) - C(0) = \frac{1-\upsilon}{B(\upsilon)}Q_3(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)} \times \int_0^t (t-\varphi)^{(\upsilon-1)} Q_3(\varphi,S,A,C,T,R)d\varphi \tag{2.18}$$

$$T(t) - T(0) = \frac{1-\upsilon}{B(\upsilon)}Q_4(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)} \times \int_0^t (t-\varphi)^{(\upsilon-1)} Q_4(\varphi,S,A,C,T,R)d\varphi \tag{2.19}$$

$$R(t) - R(0) = \frac{1-\upsilon}{B(\upsilon)}Q_5(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)} \times \int_0^t (t-\varphi)^{(\upsilon-1)} Q_5(\varphi,S,A,C,T,R)d\varphi \tag{2.20}$$

Next, the closed balls for the operators of the convergent sets above are constructed. Consider five closed balls with radius ψ and centres S_0, A_0, C_0, T_0, R_0 in the space of continuous functions, and let Θ be the space of all continuous functions.

Let $\|.\|$ denote the supremum norm defined on the domain, such that:

$$Br(S_0) = [S, S \in \Theta[0,p]; \|S - S_0\| \leq \psi] \tag{2.21}$$

$$\Rightarrow \|S\| \leq (\psi + S_0) \tag{2.22}$$

$$Br(A_0) = [A, A \in \Theta[0,p]; \|A - A_0\| \leq \psi] \tag{2.23}$$

$$\Rightarrow \|A\| \leq (\psi + A_0) \tag{2.24}$$

$$Br(C_0) = [C, C \in \Theta[0,p]; \|C - C_0\| \leq \psi] \tag{2.25}$$

$$\Rightarrow \|C\| \leq (\psi + C_0) \tag{2.26}$$

$$Br(T_0) = [T, T \in \Theta[0,p]; \|T - T_0\| \leq \psi] \tag{2.27}$$

$$\Rightarrow \|T\| \leq (\psi + T_0) \tag{2.28}$$

$$Br(R_0) = [R, R \in \Theta[0,p]; \|R - R_0\| \leq \psi] \tag{2.29}$$

$$\Rightarrow \|R\| \leq (\psi + R_0) \tag{2.30}$$

3. Existence of the solution

This section establishes the conditions necessary for the existence of solutions to the fractional differential equations presented in the system (2.6) to (2.10). To achieve this, a rigorous mathematical framework is employed, utilizing multiple fixed-point theorems. Specifically, the existence of solutions is analyzed through the Banach fixed-point theorem, which guarantees a unique solution under contraction conditions, and Schauder's fixed-point theorem, which addresses the existence of solutions in spaces lacking compactness. The methodology involves verifying essential mathematical properties, including the Lipschitz condition, which ensures bounded growth behavior, and the self-mapping property, which confirms that the system's operator maps elements within the same functional space. Additionally, the relative compactness of the solution space is examined to ensure that sequences of approximate solutions converge to an actual solution. These collectively validate the mathematical consistency and solvability of the model, reinforcing the applicability of the fractional framework to accurately represent the dynamics of HBV transmission and control.

3.1. Mapping of fixed-point operators

Each operator maps the ball into itself. For (2.11),

$$S(t) = S(0) + \frac{1-\upsilon}{B(\upsilon)} Q_1(t, S, A, C, T, R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)} \times \int_0^t (t-\varphi)^{(\upsilon-1)} Q_1(\varphi, S, A, C, T, R) d\varphi \tag{3.1}$$

Replacing

$$Q_1(t, S, A, C, T, R) = \xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R \tag{3.2}$$

$$\begin{aligned} S(t) &= S(0) + \frac{1-\upsilon}{B(\upsilon)} [\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R] + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)} \\ &\int_0^t (t-\varphi)^{(\upsilon-1)} [\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R] d\varphi \end{aligned} \tag{3.3}$$

applying norm on both sides of equation (3.3) to get:

$$\begin{aligned} \|S(t) - S(0)\| &\leq \left|\frac{1-\upsilon}{B(\upsilon)}\right| \|\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R\| + \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right| \\ &\int_0^t |(t-\varphi)|^{(\upsilon-1)} \|\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R\| d\varphi \end{aligned} \tag{3.4}$$

$$\begin{aligned} \|S(t) - S(0)\| &\leq \left|\frac{1-\upsilon}{B(\upsilon)}\right| [|\xi\|(1-\alpha)\|(1-\gamma_c)| - |\lambda_s|\|S\| + |(1-\eta)\|\omega|\|A\| - |\mu|\|S\| + |(1-\kappa)\|\nu\|\rho|\|T\| \\ &+ |\epsilon|\|R\| + \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right| \xi\|(1-\alpha)\|(1-\gamma_c)| - |\lambda_s|\|S\| + |(1-\eta)\|\omega|\|A\| - |\mu|\|S\| + |(1-\kappa)\|\nu\|\rho|\|T\| \\ &+ |\epsilon|\|R\| d\varphi \end{aligned} \tag{3.5}$$

taking into account that $Q_1(t, S, A, C, T, R) = \xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R$, so that,

$$\|S(t) - S(0)\| \leq \left|\frac{1-\upsilon}{B(\upsilon)}\right| U^*(\psi) + \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right| \int_0^t |(t-\varphi)|^{(\upsilon-1)} U^*(\psi) d\varphi \tag{3.6}$$

where

$$|\xi\|(1-\alpha)\|(1-\gamma_c)| - |\lambda_s|(\psi+\Theta) + |(1-\eta)\|\omega|(\psi+\Theta) - |\mu|(\psi+\Theta) + |(1-\kappa)\|\nu\|\rho|(\psi+\Theta) + |\epsilon|(\psi+\Theta) = U^*(\psi)$$

$\therefore$

In the case $t > \varphi$ and $\varphi > t$ the integral $\int_0^t |(t-\varphi)|^{(\upsilon-1)} d\varphi = \frac{p^*}{\upsilon}$. Thus, the self mapping results as $\|S(t) - S(0)\| \leq \psi$.

$$\left|\frac{1-\upsilon}{B(\upsilon)}\right| U^*(\psi) + \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right| U^*(\psi) \frac{p^*}{\upsilon} \leq \psi \tag{3.7}$$

$$\left|\frac{1-\upsilon}{B(\upsilon)}\right| + \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right| \frac{p^*}{\upsilon} \leq \frac{\psi}{U^*(\psi)} \tag{3.8}$$

$$p \leq \left[\left|B(\upsilon)\Gamma(\upsilon)\right|\left(\frac{\psi}{U^*(\psi)} - \left|\frac{1-\upsilon}{B(\upsilon)}\right|\right)\right]^{\frac{1}{\upsilon}} \tag{3.9}$$

The condition for self-mapping is shown in equation (3.9). In order for p to be definite, it is necessary to have that

$$\psi > \frac{(1-\upsilon)U^*(\psi)}{B(\upsilon)} \tag{3.10}$$

Similarly, an inferable estimates for A, C, T, and R are as follows.

$$p \leq \left[\left|B(\upsilon)\Gamma(\upsilon)\right|\left(\frac{\psi}{\Xi^*(\psi)} - \left|\frac{1-\upsilon}{B(\upsilon)}\right|\right)\right]^{\frac{1}{\upsilon}} \tag{3.11}$$

$$\psi > \frac{(1-\upsilon)\Xi^*(\psi)}{B(\upsilon)} \tag{3.12}$$

$$p \leq \left[\left|B(\upsilon)\Gamma(\upsilon)\right|\left(\frac{\psi}{\Pi^*(\psi)} - \left|\frac{1-\upsilon}{B(\upsilon)}\right|\right)\right]^{\frac{1}{\upsilon}} \tag{3.13}$$

$$\psi > \frac{(1-\upsilon)\Pi^*(\psi)}{B(\upsilon)} \tag{3.14}$$

$$p \leq \left[\left|B(\upsilon)\Gamma(\upsilon)\right|\left(\frac{\psi}{\Omega^*(\psi)} - \left|\frac{1-\upsilon}{B(\upsilon)}\right|\right)\right]^{\frac{1}{\upsilon}} \tag{3.15}$$

$$\psi > \frac{(1-\upsilon)\Omega^*(\psi)}{B(\upsilon)} \tag{3.16}$$

$$p \leq \left[\left|B(\upsilon)\Gamma(\upsilon)\right|\left(\frac{\psi}{\Delta^*(\psi)} - \left|\frac{1-\upsilon}{B(\upsilon)}\right|\right)\right]^{\frac{1}{\upsilon}} \tag{3.17}$$

$$\psi > \frac{(1-\upsilon)\Delta^*(\psi)}{B(\upsilon)} \tag{3.18}$$

Theorem 3.1. *The operator vectors* (S, A, C, T, R) *maps the closed set in the event that the inequalities in* (3.1-3.18) *hold true.*

3.2. Relative compact mapping

For the relative compact mapping, recall that:

$$S(t) = S(0) + \frac{1-\upsilon}{B(\upsilon)}Q_1(t, S, A, C, T, R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^t (t-\varphi)^{(\upsilon-1)}Q_1(\varphi, S, A, C, T, R)d\varphi \tag{3.19}$$

Taking into account the group of functions i.e:

$$S_i(t) = S(0) + \frac{1-\upsilon}{B(\upsilon)}Q_1(t, S_i, A, C, T, R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^t (t-\varphi)^{(\upsilon-1)}Q_1(\varphi, S_i, A, C, T, R)d\varphi \tag{3.20}$$

and,

$$S_i(t^*) = S(0) + \frac{1-\upsilon}{B(\upsilon)}Q_1(t^*, S_i, A, C, T, R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^{t^*} (t^*-\varphi)^{(\upsilon-1)}Q_1(\varphi, S_i, A, C, T, R)d\varphi \tag{3.21}$$

Equation (3.21) subtracted from (3.20) gives:

$$S_i(t) - S_i(t^*) = \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\left[\int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*} (t^*-\varphi)^{(\upsilon-1)}d\varphi\right]Q_1(\varphi, S_i, A, C, T, R) \tag{3.22}$$

$$S_i(t) - S_i(t^*) = \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\left[\int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi\right] \times(\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R) \tag{3.23}$$

Considering the norm of both sides

$$\|S_i(t) - S_i(t^*)\| = \left\|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\left[\int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi\right] (\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R)\right\| \tag{3.24}$$

$$\|S_i(t) - S_i(t^*)\| \le \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right|\left|\int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi\right| \times |(\xi(1-\alpha)\|(1-\gamma_c)| - |\lambda_s|\|S\| + |(1-\eta)\|\omega|\|A\| - |\mu|\|S\| + |(1-\kappa)||\nu||\rho|\|T\| + |\epsilon|\|R\|) \tag{3.25}$$

The self-mapping analysis demonstrated that $|\xi\|(1-\alpha)\|(1-\gamma_c)| - |\lambda_s|(\psi+\Theta) + |(1-\eta)\|\omega|(\psi+\Theta) - |\mu|(\psi+\Theta) + |(1-\kappa)\|\nu\|\rho|(\psi+\Theta) + |\epsilon|(\psi+\Theta) = U^*(\psi)$. Substituting this expression into equation (3.25) yields:

$$\|S_i(t) - S_i(t^*)\| \le \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right|\left|\int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi\right| U^*(\psi) \tag{3.26}$$

Now, assume $Z = \int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi$. At first when t^* lies in the interval $(0, t)$, then:

$$\begin{aligned}
Z_1 &= \int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi \\
&= \int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi + \int_t^{t^*}(t-\varphi)^{(\upsilon-1)}d\varphi - \int_t^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi \\
&= \int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi - \int_{t^*}^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_t^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi \\
&= \int_0^t (t-\varphi)^{(\upsilon-1)}d\varphi - \int_0^t (t^*-\varphi)^{(\upsilon-1)}d\varphi - \int_t^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi \\
&= -\left[\int_0^t (t^*-\varphi)^{(\upsilon-1)} - (t-\varphi)^{(\upsilon-1)}d\varphi\right] - \int_t^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi \\
&= -\left[\int_0^t (t^*-\varphi)^{(\upsilon-1)} - (t-\varphi)^{(\upsilon-1)}d\varphi + \int_t^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi\right]
\end{aligned} \tag{3.27}$$

By substituting equation (3.27) into (3.26), to get

$$\|S_i(t) - S_i(t^*)\| \le \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right|\left|-\left[\int_0^t (t^*-\varphi)^{(\upsilon-1)} - (t-\varphi)^{(\upsilon-1)}d\varphi + \int_t^{t^*}(t^*-\varphi)^{(\upsilon-1)}d\varphi\right]\right| U^*(\psi) \tag{3.28}$$

$$\|S_i(t) - S_i(t^*)\| \le \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right| \int_0^t \left|(t^*-\varphi)^{(\upsilon-1)} - (t-\varphi)^{(\upsilon-1)}\right|d\varphi + \int_t^{t^*}\left|(t^*-\varphi)^{(\upsilon-1)}\right|d\varphi U^*(\psi) \tag{3.29}$$

It is evident that $f(t) = (t-\varphi)^{(\upsilon-1)}$ satisfies the conditions of the mean value theorem. The function $f(t) = (t-\varphi)^{(\upsilon-1)}$ is continuous on the interval $[t^*, t]$ and differentiable on (t^*, t), implying the existence of some $d \in [t^*, t]$.

$\Rightarrow \frac{(t-\varphi)^{\upsilon-1} - (t^*-\varphi)^{\upsilon-1}}{(t-t^*)} = f(d)$

$$(t-\varphi)^{\upsilon-1} - (t^*-\varphi)^{\upsilon-1} = (t-t^*)(\upsilon-1)(t-d)^{\upsilon-2}$$

taking the norm of above equation,

$$\|(t-\varphi)^{\upsilon-1}-(t^*-\varphi)^{\upsilon-1}\| \leq \|(t^*-t)\|\left|(\upsilon-1)(t-d)^{\upsilon-2}\right| \tag{3.30}$$

Assume $W=(\upsilon-1)(t-d)^{\upsilon-2}$, so that

$$\|(t^*-\varphi)^{\upsilon-1}-(t-\varphi)^{\upsilon-1}\| \leq |(t^*-t)|W \tag{3.31}$$

Substitute equation (3.30) into (3.29) to get:

$$\|S_i(t)-S_i(t^*)\| \leq \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right|\left[\int_0^t W|(t^*-t)|d\varphi+\int_t^{t^*}\left|(t^*-\varphi)^{(\upsilon-1)}\right|d\varphi\right]U^*(\psi) \tag{3.32}$$

$$\|S_i(t)-S_i(t^*)\| \leq \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right|\left[W|(t^*-t)|\int_0^t d\varphi+\int_t^{t^*}\left|(t^*-\varphi)^{(\upsilon-1)}\right|d\varphi\right]U^*(\psi) \tag{3.33}$$

$$\|S_i(t)-S_i(t^*)\| \leq \left|\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\right|\left[W|(t^*-t)t|+\frac{\left|(t^*-t)^{(\upsilon)}\right|}{\upsilon}\right]U^*(\psi) \tag{3.34}$$

$$\|S_i(t)-S_i(t^*)\| \leq \left[\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}U^*(\psi)Wt+\frac{\left|(t^*-t)^{(\upsilon-1)}\right|}{B(\upsilon)\Gamma(\upsilon)}U^*(\psi)\right]|(t^*-t)| \tag{3.35}$$

As $|(t^*-t)|\to 0$, then $\|S_i(t)-S_i(t^*)\| \to 0$
So, if $|(t^*-t)| \leq \delta, \|S_i(t)-S_i(t^*)\| \leq X\gamma=\varepsilon$ where $X\gamma=\varepsilon$, then

$$\|S_i(t)-S_i(t^*)\| < \epsilon \tag{3.36}$$

Where,

$$X=\left[\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}U^*(\psi)Wt+\frac{\left|(t^*-t)^{(\upsilon-1)}\right|}{B(\upsilon)\Gamma(\upsilon)}U^*(\psi)\right] \tag{3.37}$$

The same will follow when t lies between (0, t^*), to obtain

$$X_1=\left[\frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}U^*(\psi)Wt^*+\frac{\left|(t^*-t)^{(\upsilon-1)}\right|}{B(\upsilon)\Gamma(\upsilon)}U^*(\psi)\right] \tag{3.38}$$

$S_i(t)$ is equicontinuous, as demonstrated by equation (3.36), and there is a uniformly convergent subsequence $S_{ij}t$ of $S_i(t)$, according to the Arzela-Ascoli Theorem. Therefore, $S(t)$ is comparatively compact. It is also possible to demonstrate the equicontinous nature of the other fixed point operator A, C, T, R and hence conclude that Arzela-Ascoli theorem is true and applicable.

3.2.1. Positivity and boundedness

Lemma. *For any* $(S(t),A(t),C(t),T(t),R(t)) \in R_+^5$ *for* $t>0$, *the solutions related to the model* (2.6)-(2.10) *are bounded and positive.*

Proof. *It is necessary to show that the vector field point* R_+5 *exists on every plane that borders the positive orthant in order to prove that the solution to the model is non-negative. Considering the model* (2.6)–(2.10)

$${}^{B}D_t^{\upsilon}S(t)|_{S=0}=\xi(1-\alpha)(1-\gamma_c)-\lambda_s S+(1-\eta)\omega A-\mu S+(1-\kappa)\nu\rho T+\epsilon R \tag{3.39}$$

$${}^{B}D_t^{\upsilon}A(t)|_{A=0}=\lambda_s S-(\omega+\mu)A \tag{3.40}$$

$${}^{B}D_t^{\upsilon}C(t)|_{C=0}=\eta\omega A+\xi(1-\alpha)\gamma_c+(1-\nu)\rho T-(\sigma+\mu)C \tag{3.41}$$

$${}^{B}D_t^{\upsilon}T(t)|_{T=0}=\sigma C-(\rho+\mu)T \tag{3.42}$$

$$ {}^{B}D_t^{v} R(t)|_{R=0} = \xi\alpha + \kappa\nu\rho T - (\epsilon+\mu)R \tag{3.43} $$

The solution is:

$$ N(t) \leq \frac{\xi}{\mu} + ke^{-ut}. \tag{3.44} $$

Considering that for each $t > 0$, the Mittag-Leffler function is bounded. This suggests that $lim_{t\to\infty} \leq \frac{\xi}{\mu}$. As a result, the solutions to systems (2.6) through (2.11) are biologically invariant throughout the area and are shown as follows: $\tau = (S,A,C,T,R) \in R_+^5 : N \leq \frac{\xi}{\mu}$. As every term in the system (2.6)–(2.10) is non-negative, the solution is bounded.

4. The equilibrium points analysis

4.1. Analyzing the disease free equilibrium point

The model disease free equilibrium (DFE) denoted as E_0 exists and is given as:

$$ E_0 = \left[\frac{\xi(1-\alpha\mu)}{\mu}, 0, 0, 0, 0\right] \tag{4.1} $$

4.2. Analyzing the endemic equilibrium point

The model endemic equilibrium (EE)denoted as E^* exists and is given as:

$$ E^* = \left[-\frac{(\mu+\omega)\left(\xi\gamma(\mu+\rho)\rho(\alpha-1)\rho\sigma+\mu^2+\mu\rho+\mu\sigma\right)}{L}, \frac{S^*}{\Lambda(\mu+\omega)}, -\frac{(\mu+\rho)\eta\omega\Lambda(\alpha\mu-\epsilon-\mu)}{L}, \frac{C^*}{\sigma(\mu+\rho)}, \frac{H}{L}\right] \tag{4.2} $$

Consequentially, the basic reproduction number R_0 gives:

$$ R_0 = \frac{\beta\xi(1-\alpha)}{\mu(\omega+\mu)} - \frac{\beta\xi(1-\alpha)\eta\omega}{\mu(\omega+\mu)(\xi(1-\alpha)\gamma+(\sigma+\mu))} \tag{4.3} $$

4.3. Stability analysis of equilibrium points

4.3.1. Local stability of the disease free equilibrium point

The disease-free equilibrium is locally stable in the Jacobian matrix shown as:

$$ J = \begin{bmatrix} -\beta A - \xi\beta - \mu & (1-\eta)\omega - \beta S & \xi\gamma(\alpha-1) - \xi\beta S & (1-\kappa)v\rho & \epsilon \\ \beta A + \xi\beta C & -\omega - \mu + \beta S & \xi\beta S & 0 & 0 \\ 0 & \omega\eta & \xi\gamma(1-\alpha) - \sigma - \mu & (1-v)\rho & 0 \\ 0 & 0 & \sigma & -\rho - \mu & 0 \\ 0 & 0 & 0 & \kappa v\rho & -\epsilon - \mu \end{bmatrix} \tag{4.4} $$

4.3.2. Global stability of the disease free equilibrium point

Theorem 4.1. *The disease-free equilibrium in model*(2.6)-(2.10) *is globally asymptotically stable if* $R_0 < 1$. ***Proof*** *Consider the Lyapunov function* Γ_1: $\Xi \to \varphi$ *which can be defined as*:

$$ \Gamma_1 = \left(S - S_0 - S_0 ln\frac{S}{S_0}\right) + A + C + T + R, $$

$$ \forall (S, A, C, T, R) \in \Xi^5 $$

Then,

$$ {}^{B}D_t^{\nu}\Gamma_1 \leq \left(1-\frac{S_0}{S}\right)^{B} D_t^{\nu}S + {}^{B}D_t^{\nu}A + {}^{B}D_t^{\nu}C + {}^{B}D_t^{\nu}T + {}^{B}D_t^{\nu}R \tag{4.5} $$

$$ {}^{B}D_t^{\nu}\Gamma_1 \leq \left(\frac{S-S_0}{S}\right)^{B} D_t^{\nu}S + {}^{B}D_t^{\nu}A + {}^{B}D_t^{\nu}C + {}^{B}D_t^{\nu}T + {}^{B}D_t^{\nu}R \tag{4.6} $$

$$ {}^{B}D_t^{\nu}\Gamma_1 \leq \left(\frac{S-S_0}{S}\right)(\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R) \tag{4.7} $$

$$ +(\lambda_s S - (\omega+\mu)A) + (\eta\omega A + \xi(1-\alpha)\gamma_c + (1-\nu)\rho T - (\sigma+\mu)C) + (\sigma C - (\rho+\mu)T) \tag{4.8} $$

$$ +(\xi\alpha + \kappa\nu\rho T - (\epsilon+\mu)R) \tag{4.9} $$

$$ {}^{B}D_t^{\nu}\Gamma_1 \leq (S-S_0)\left(\frac{\xi(1-\alpha)(1-\gamma_c)}{S}\right) - \lambda_s S + \omega A - \eta\omega A - \mu S + \nu\rho T - \kappa\nu\rho T + \epsilon R\Bigg) \tag{4.10} $$

$$ +(\lambda_s S - (\omega A + \mu A) + (\eta\omega A + \xi(1-\alpha)\gamma_c + \rho T - \nu\rho T - \sigma C + \mu C) + (\sigma C - \rho T + \mu T) \tag{4.11} $$

$$ +(\xi\alpha + \kappa\nu\rho T - \epsilon R + \mu R) \tag{4.12} $$

$$ {}^{B}D_t^{\nu}\Gamma_1 \leq (S-S_0)\left(\frac{\xi(1-\alpha)(1-\gamma_c)}{S}\right) - \mu S - \mu A\Bigg) + (\xi(1-\alpha)\gamma_c - \mu C - \mu T) + (\xi\alpha + \mu R) \tag{4.13} $$

$$ {}^{B}D_t^{\nu}\Gamma_1 \leq (S-S_0)\left(\frac{1}{S}-\frac{1}{S_0}\right)\xi(1-\alpha)(1-\gamma_c - \mu S - \mu A) + (\xi(1-\alpha)\gamma_c - \mu C - \mu T) + (\xi\alpha + \mu R) \tag{4.14} $$

$$ {}^{B}D_t^{\nu}\Gamma_1 \leq \left(\frac{(S-S_0)^2}{SS_0}\right)\xi(1-\alpha)(1-\gamma_c - \mu S - \mu A) + (\xi(1-\alpha)\gamma_c - \mu C - \mu T) + (\xi\alpha + \mu R) \tag{4.15} $$

The computations above demonstrate that ${}^{B}D_t^{\nu}\Gamma_1 < 0$ if $R_0 < 1$.

The disease-free equilibrium is therefore asymptotically stable globally as presented by (Oludoun et al., 2021).

4.3.3. Global stability of the endemic equilibrium point

Theorem 4.2. *The endemic equilibrium is globally asymptotically stable if* $R_0 > 1$.

Proof. *Considering the Lyapunov function* Γ_2: $\Xi \to \varphi$, *where*

$$ \Gamma_2 = \left(S - S^* - S^* ln\frac{S}{S^*}\right) + \left(A - A^* - A^* ln\frac{A}{A^*}\right) + \left(C - C^* - C^* ln\frac{C}{C^*}\right) + \left(T - T^* - T^* ln\frac{T}{T^*}\right) + \left(R - R^* - R^* ln\frac{R}{R^*}\right), \forall (S, A, C, T, R) \in R^5. \tag{4.16} $$

Then,

$$ {}^{B}D_t^{\nu}\Gamma_2 \leq \frac{S-S^*}{S}{}^{B} D_t^{\nu}S + \frac{A-A^*}{A}{}^{B} D_t^{\nu}A + \frac{C-C^*}{C}{}^{B} D_t^{\nu}C + \frac{T-T^*}{T}{}^{B} D_t^{\nu}T + \frac{R-R^*}{R}{}^{B} D_t^{\nu}R, \tag{4.17} $$

$$ {}^{B}D_t^{\nu}\Gamma_2 \leq \frac{S-S^*}{S}((\xi(1-\alpha)(1-\gamma_c) - \lambda_s S + (1-\eta)\omega A - \mu S + (1-\kappa)\nu\rho T + \epsilon R)) + \frac{A-A^*}{A}(\lambda_s S - (\omega+\mu)A) + \frac{C-C^*}{C}\eta\omega A + \xi(1-\alpha)\gamma_c + (1-\nu)\rho T - (\sigma+\mu)C\Big) + \frac{T-T^*}{T}(\sigma C - (\rho+\mu)T) + \frac{R-R^*}{R}(\xi\alpha + \kappa\nu\rho T - (\epsilon+\mu)R) \tag{4.18} $$

$$
\begin{aligned}
{}^{B}D_t^{\upsilon}\Gamma_2 \leq (S-S^*)\left[\frac{\xi(1-\alpha)(1-\gamma_c)}{S}-\lambda_s+\frac{(1-\eta)\omega A}{S}-\mu+\frac{(1-\kappa)\nu\rho T}{S}+\frac{\epsilon R}{S}\right]+(A-A^*)\left[\frac{\lambda_s S}{A}-(\omega+\mu)\right]+\\
(C-C^*)\left[\frac{\eta\omega A}{C}+\frac{\xi(1-\alpha)\gamma_c}{C}+\frac{(1-\nu)\rho T}{C}-(\sigma+\mu)\right]+(T-T^*)\left[\frac{\sigma C}{T}-(\rho+\mu)\right]+\\
(R-R^*)\left[\frac{(\xi\alpha+\kappa\nu\rho)T}{R}-(\epsilon+\mu)\right]
\end{aligned}
\tag{4.19}
$$

$$
\begin{aligned}
{}^{B}D_t^{\upsilon}\Gamma_2 \leq (S-S^*)\left[\frac{\xi(1-\alpha)(1-\gamma_c)}{S}-\frac{\xi(1-\alpha)(1-\gamma_c)}{S^*}-\lambda_s+\lambda_s+\frac{(1-\eta)\omega A}{S}-\frac{(1-\eta)\omega A^*}{S^*}-\mu+\right.\\
\left.\frac{(1-\kappa)\nu\rho}{S}-\frac{(1-\kappa)\nu\rho T^*}{S^*}+\frac{\epsilon R}{S}-\frac{\epsilon R^*}{S^*}\right]+(A-A^*)\left[\frac{\lambda_s S}{A}-\frac{\lambda_s S}{A^*}-\omega+\omega\right]+\\
(C-C^*)\left[\frac{\eta\omega A}{C}-\frac{\eta\omega A^*}{C^*}+\frac{\xi(1-\alpha)\gamma_c}{C}-\frac{\xi(1-\alpha)\gamma_c}{C^*}+\frac{(1-\nu)\rho T}{C}-\frac{(1-\nu)\rho T^*}{C^*}-\sigma+\sigma\right]\\
+(T-T^*)\left[\frac{\sigma C}{T}-\frac{\sigma C^*}{T^*}-\rho+\rho\right]+(R-R^*)\left[\frac{(\xi\alpha+\kappa\nu\rho)T}{R}-\frac{(\xi\alpha+\kappa\nu\rho)T}{R^*}-\epsilon+\epsilon\right]
\end{aligned}
\tag{4.20}
$$

$$
\begin{aligned}
{}^{B}D_t^{\upsilon}\Gamma_2 \leq -\frac{(S-S^*)^2}{SS^*}\xi(1-\alpha)(1-\gamma_c)-\frac{(A-A^*)^2}{AA^*}\lambda_s S-\frac{(C-C^*)^2}{CC^*}\xi(1-\alpha)\gamma_c-\frac{(T-T^*)^2}{TT^*}\sigma C-\\
\left.\frac{(R-R^*)^2}{RR^*}\xi\alpha+\kappa\nu\rho\right)T
\end{aligned}
\tag{4.21}
$$

The equations above show that ${}^{B}D_t^{\upsilon}\Gamma_2 \leq 0$ for $R_0 > 1$.

Therefore, the endemic equilibrium is globally asymptotically stable.

4.4. Analysis of the model with Mittag-Leffler Kernel

The following problem will be considered with the Atangana-Baleanu derivative:

$$
{}^{B}D_t^{\upsilon}S(t) = \xi(1-\alpha)(1-\gamma_c)-\lambda_s S+(1-\eta)\omega A-\mu S+(1-\kappa)\nu\rho T+\epsilon R \tag{4.22}
$$

$$
{}^{B}D_t^{\upsilon}A(t) = \lambda_s S-(\omega+\mu)A \tag{4.23}
$$

$$
{}^{B}D_t^{\upsilon}C(t) = \eta\omega A+\xi(1-\alpha)\gamma_c+(1-\nu)\rho T-(\sigma+\mu)C \tag{4.24}
$$

$$
{}^{B}D_t^{\upsilon}T(t) = \sigma C-(\rho+\mu)T \tag{4.25}
$$

$$
{}^{B}D_t^{\upsilon}R(t) = \xi\alpha+\kappa\nu\rho T-(\epsilon+\mu)R \tag{4.26}
$$

Simplifying further as:

$$
Q_1(t,S,A,C,T,R) = \xi(1-\alpha)(1-\gamma_c)-\lambda_s S+(1-\eta)\omega A-\mu S+(1-\kappa)\nu\rho T+\epsilon R, \forall t \geq 0 \tag{4.27}
$$

$$
Q_2(t,S,A,C,T,R) = \lambda_s S-(\omega+\mu)A, \forall t \geq 0 \tag{4.28}
$$

$$
Q_3(t,S,A,C,T,R) = \eta\omega A+\xi(1-\alpha)\gamma_c+(1-\nu)\rho T-(\sigma+\mu)C, \forall t \geq 0 \tag{4.29}
$$

$$
Q_4(t,S,A,C,T,R) = \sigma C-(\rho+\mu)T, \forall t \geq 0 \tag{4.30}
$$

$$
Q_5(t,S,A,C,T,R) = \xi\alpha+\kappa\nu\rho T-(\epsilon+\mu)R, \forall t \geq 0 \tag{4.31}
$$

to have:

$$
\frac{B(\upsilon)}{1-\upsilon}\frac{d}{dt}\int_0^t S(\varphi)E_x\left(\frac{-\upsilon}{1-\upsilon}(t-\varphi)^{\upsilon}\right)d\varphi = Q_1(t,S,A,C,T,R) \tag{4.32}
$$

$$
\frac{B(\upsilon)}{1-\upsilon}\frac{d}{dt}\int_0^t A(\varphi)E_x\left(\frac{-\upsilon}{1-\upsilon}(t-\varphi)^{\upsilon}\right)d\varphi = Q_2(t,S,A,C,T,R) \tag{4.33}
$$

$$
\frac{B(\upsilon)}{1-\upsilon}\frac{d}{dt}\int_0^t C(\varphi)E_x\left(\frac{-\upsilon}{1-\upsilon}(t-\varphi)^{\upsilon}\right)d\varphi = Q_3(t,S,A,C,T,R) \tag{4.34}
$$

$$\frac{B(\upsilon)}{1-\upsilon}\frac{d}{dt}\int_0^t T(\varphi)E_x\Big(\frac{-\upsilon}{1-\upsilon}(t-\varphi)^{\upsilon}\Big)d\varphi = Q_4(t,S,A,C,T,R) \tag{4.35}$$

$$\frac{B(\upsilon)}{1-\upsilon}\frac{d}{dt}\int_0^t R(\varphi)E_x\Big(\frac{-\upsilon}{1-\upsilon}(t-\varphi)^{\upsilon}\Big)d\varphi = Q_5(t,S,A,C,T,R) \tag{4.36}$$

By applying the B integral to get,

$$S(t)-S(0) = \frac{1-\upsilon}{B(\upsilon)}Q_1(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^t (t-\varphi)^{(\upsilon-1)}Q_1(\varphi,S,A,C,T,R)d\varphi \tag{4.37}$$

$$A(t)-A(0) = \frac{1-\upsilon}{B(\upsilon)}Q_2(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^t (t-\varphi)^{(\upsilon-1)}Q_2(\varphi,S,A,C,T,R)d\varphi \tag{4.38}$$

$$C(t)-C(0) = \frac{1-\upsilon}{B(\upsilon)}Q_3(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^t (t-\varphi)^{(\upsilon-1)}Q_3(\varphi,S,A,C,T,R)d\varphi \tag{4.39}$$

$$T(t)-T(0) = \frac{1-\upsilon}{B(\upsilon)}Q_4(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^t (t-\varphi)^{(\upsilon-1)}Q_4(\varphi,S,A,C,T,R)d\varphi \tag{4.40}$$

$$R(t)-R(0) = \frac{1-\upsilon}{B(\upsilon)}Q_5(t,S,A,C,T,R) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^t (t-\varphi)^{(\upsilon-1)}Q_5(\varphi,S,A,C,T,R)d\varphi \tag{4.41}$$

Discretizing the equations at t_{m+1} to have:

$$S^{m+1} = S(0) + \frac{1-\upsilon}{B(\upsilon)}Q_1\left(t_{m+1},S^m,A^m,C^m,T^m,R^m\right) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^{t_{m+1}} (t_{m+1}-\varphi)^{(\upsilon-1)}Q_1(\varphi,S,A,C,T,R)d\varphi \tag{4.42}$$

$$A^{m+1} = A(0) + \frac{1-\upsilon}{B(\upsilon)}Q_2\left(t_{m+1},S^m,A^m,C^m,T^m,R^m\right) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^{t_{m+1}} (t_{m+1}-\varphi)^{(\upsilon-1)}Q_2(\varphi,S,A,C,T,R)d\varphi \tag{4.43}$$

$$C^{m+1} = C(0) + \frac{1-\upsilon}{B(\upsilon)}Q_3\left(t_{m+1},S^m,A^m,C^m,T^m,R^m\right) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^{t_{m+1}} (t_{m+1}-\varphi)^{(\upsilon-1)}Q_3(\varphi,S,A,C,T,R)d\varphi \tag{4.44}$$

$$T^{m+1} = T(0) + \frac{1-\upsilon}{B(\upsilon)}Q_4\left(t_{m+1},S^m,A^m,C^m,T^m,R^m\right) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^{t_{m+1}} (t_{m+1}-\varphi)^{(\upsilon-1)}Q_4(\varphi,S,A,C,T,R)d\varphi \tag{4.45}$$

$$R^{m+1} = R(0) + \frac{1-\upsilon}{B(\upsilon)}Q_5\left(t_{m+1},S^m,A^m,C^m,T^m,R^m\right) + \frac{\upsilon}{B(\upsilon)\Gamma(\upsilon)}\int_0^{t_{m+1}} (t_{m+1}-\varphi)^{(\upsilon-1)}Q_5(\varphi,S,A,C,T,R)d\varphi \tag{4.46}$$

Hence by the method shown in (iqbal et al., 2023b), we obtain:

$$\begin{aligned} S^{m+1} &= S(0) + \frac{1-\upsilon}{B(\upsilon)}Q_1\left(t_{m+1},S^m,A^m,C^m,T^m,R^m\right) + \frac{\upsilon}{B(\upsilon)} \\ &\sum_{k=0}^{m}\left[\frac{h^{\upsilon}Q_1\left(t_k,S^m,A^m,C^m,T^m,R^m\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^{\upsilon}(m-k+2+\upsilon)-(m-k)^{\upsilon}(m-k+2+2\upsilon)\right)\right] \\ &-\frac{\upsilon}{B(\upsilon)}\sum_{k=0}^{m}\left[\frac{h^{\upsilon}Q_1\left(t_{k-1},S^{m-1},A^{m-1},C^{m-1},T^{m-1},R^{m-1}\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^{\upsilon+1}-(m-k)^{\upsilon}(m-k+1+\upsilon)\right)\right] \end{aligned} \tag{4.47}$$

$$\begin{aligned} A^{m+1} &= A(0) + \frac{1-\upsilon}{B(\upsilon)}Q_2\left(t_{m+1},S^m,A^m,C^m,T^m,R^m\right) + \frac{\upsilon}{B(\upsilon)} \\ &\sum_{k=0}^{m}\left[\frac{h^{\upsilon}Q_2\left(t_k,S^m,A^m,C^m,T^m,R^m\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^{\upsilon}(m-k+2+\upsilon)-(m-k)^{\upsilon}(m-k+2+2\upsilon)\right)\right] \\ &-\frac{\upsilon}{B(\upsilon)}\sum_{k=0}^{m}\left[\frac{h^{\upsilon}Q_2\left(t_{k-1},S^{m-1},A^{m-1},C^{m-1},T^{m-1},R^{m-1}\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^{\upsilon+1}-(m-k)^{\upsilon}(m-k+1+\upsilon)\right)\right] \end{aligned} \tag{4.48}$$

$$C^{m+1} = A(0) + \frac{1-\upsilon}{B(\upsilon)} Q_3\left(t_{m+1}, S^m, A^m, C^m, T^m, R^m\right) + \frac{\upsilon}{B(\upsilon)}$$
$$\sum_{k=0}^{m}\left[\frac{h^\upsilon Q_3\left(t_k, S^m, A^m, C^m, T^m, R^m\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^\upsilon(m-k+2+\upsilon) - (m-k)^\upsilon(m-k+2+2\upsilon)\right)\right] \tag{4.49}$$
$$-\frac{\upsilon}{B(\upsilon)}\sum_{k=0}^{m}\left[\frac{h^\upsilon Q_3\left(t_{k-1}, S^{m-1}, A^{m-1}, C^{m-1}, T^{m-1}, R^{m-1}\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^{\upsilon+1} - (m-k)^\upsilon(m-k+1+\upsilon)\right)\right]$$

$$T^{m+1} = A(0) + \frac{1-\upsilon}{B(\upsilon)} Q_4\left(t_{m+1}, S^m, A^m, C^m, T^m, R^m\right) + \frac{\upsilon}{B(\upsilon)}$$
$$\sum_{k=0}^{m}\left[\frac{h^\upsilon Q_4\left(t_k, S^m, A^m, C^m, T^m, R^m\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^\upsilon(m-k+2+\upsilon) - (m-k)^\upsilon(m-k+2+2\upsilon)\right)\right] \sum_{k=0}^{m}\left[\frac{h^\upsilon Q_4\left(t_{k-1}, S^{m-1}, A^{m-1}, C^{m-}\right.}{\Gamma(\upsilon+2)}\right.$$
$$-\upsilon B(\upsilon) \tag{4.50}$$

$$R^{m+1} = A(0) + \frac{1-\upsilon}{B(\upsilon)} Q_5\left(t_{m+1}, S^m, A^m, C^m, T^m, R^m\right) + \frac{\upsilon}{B(\upsilon)}$$
$$\sum_{k=0}^{m}\left[\frac{h^\upsilon Q_5\left(t_k, S^m, A^m, C^m, T^m, R^m\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^\upsilon(m-k+2+\upsilon) - (m-k)^\upsilon(m-k+2+2\upsilon)\right)\right] \tag{4.51}$$
$$-\frac{\upsilon}{B(\upsilon)}\sum_{k=0}^{m}\left[\frac{h^\upsilon Q_5\left(t_{k-1}, S^{m-1}, A^{m-1}, C^{m-1}, T^{m-1}, R^{m-1}\right)}{\Gamma(\upsilon+2)}\left((m+1-k)^{\upsilon+1} - (m-k)^\upsilon(m-k+1+\upsilon)\right)\right]$$

5. Results and discussion

This section takes a novel method to investigating HBV epidemics, utilizing a mathematical model based on vital dynamics, as described in (Oludoun et al., 2021) as shown in Fig. 1. The goal is to investigate the effects of various fractional orders on HBV dynamics and provide insights into how changing parameters affect disease transmission and control. The various fractional orders employed in the numerical simulations are explained here, with reference to the initial conditions and parameter values stated in Oludoun et al.'s study (Oludoun et al., 2021).

Fig. 2 demonstrates the behaviour of a susceptible population under different fractional orders; including spontaneous clearance provides a biological mechanism that could reduce the susceptible count over time. Initially, exposure to the virus

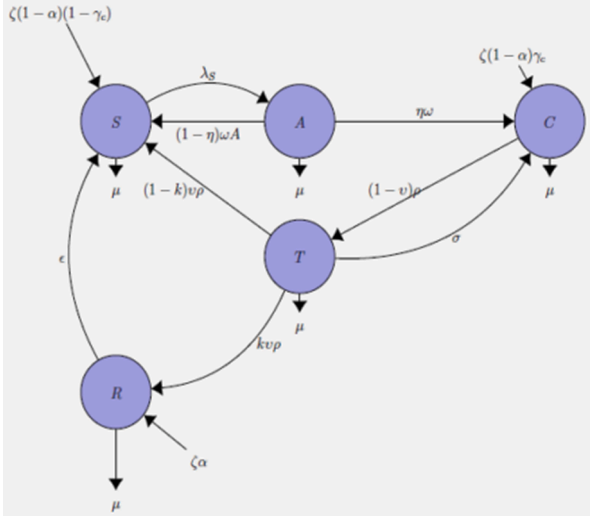

Fig. 1. Schematic diagram of mathematical modeling of HBV with vital dynamics.

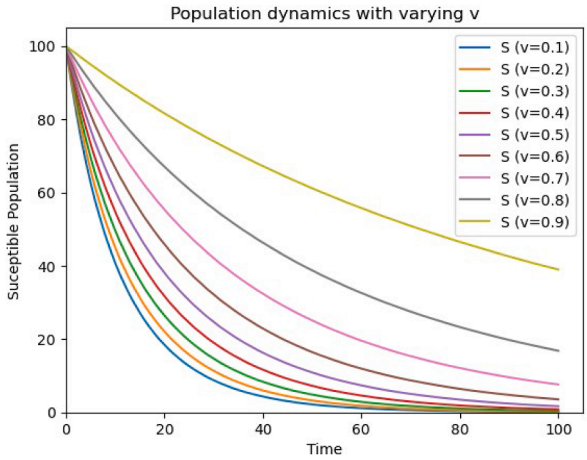

Fig. 2. Simulation of the effects of fractional order on Susceptible population of HBV dynamics.

may increase the number of susceptible individuals; however, as time passes, those with stronger immune systems may spontaneously clear the infection, resulting in a decrease in the susceptible group. The fractional-order model captures the memory effect or hereditary aspects of the population's reaction, indicating how previous interactions with the virus influence future susceptibility. From a biological perspective, this might mean that those who have recovered from the virus might become partially immune or resistant, which would reduce the number of new infections. As clearance rises, the vulnerable population would consequently decrease more quickly, indicating a change in the dynamics of the epidemic. As fewer people become susceptible to infection, this process may eventually lead to herd immunity or a slower spread of the epidemic. Because different populations have different immune responses, this complex relationship between fractional orders and spontaneous clearance might help model epidemics more accurately.

The behaviour of a chronic population under different fractional orders is depicted in Fig. 3, which demonstrates that the chronic population grows as spontaneous clearance values rise. A biological explanation for this seemingly paradoxical result is that in people who have progressed to the chronic stage of infection, spontaneous clearance is insufficient to eradicate the virus. At this point, the virus has often established itself in the host, resisting immune reactions and remaining in the body for an extended period of time. Additional therapeutic measures, such as antiviral medications or immune-modulating therapies, are frequently necessary for chronic infections like hepatitis B or HIV in order to successfully lower the viral load and encourage recovery. The rise in the chronic population, accompanied by increasing spontaneous clearance rates, may represent the biological truth that, without effective treatment, people remain in the chronic phase for longer periods of time. Chronic infection may persist as a result of the immune system mounting a partial response that restricts viral replication but leaves the virus. Past infection history influences illness progression in a fractional-order model that takes memory and genetic effects into account. The protracted character of chronic illnesses, in which the

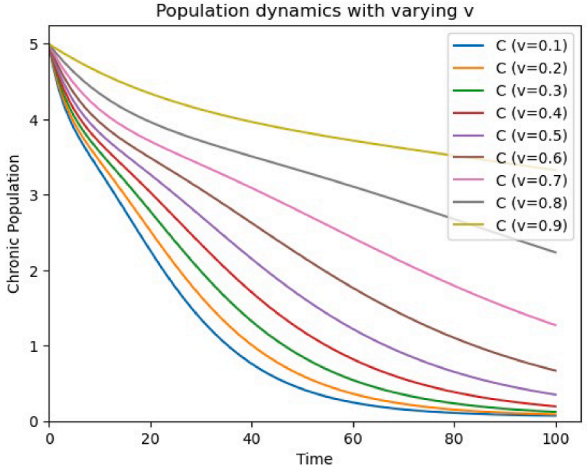

Fig. 3. Simulation of the effects of fractional order on Chronic population of HBV dynamics.

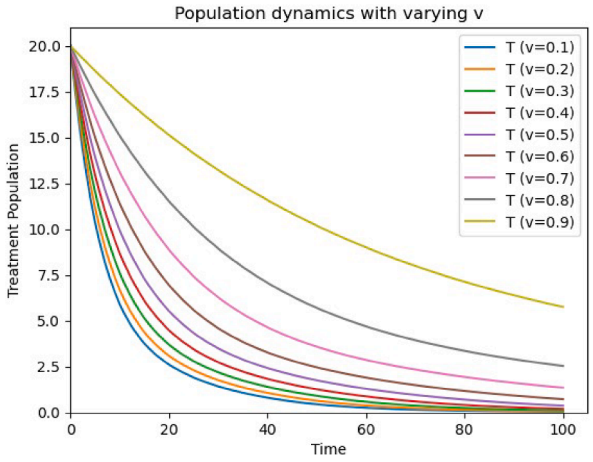

Fig. 4. Simulation of the effects of fractional order on Treated population of HBV dynamics.

immune system's capacity to eradicate the virus gradually deteriorates, is reflected in this model. Therefore, without proper treatment, people are likely to continue in the chronic phase, contributing to a bigger chronic population, even though spontaneous clearance may reduce the progression of the disease.

Fig. 4 depicts the behaviour of a treated population under different fractional orders, demonstrating that as spontaneous clearance values grow, so does the treated population. This is scientifically explained by the fact that in people at the chronic stage of infection, spontaneous clearance is not enough to eradicate the virus. The virus's capacity to elude immune reactions and endure inside the host is what defines chronic infections. Treatment measures, including antiviral treatments or immune-stimulating drugs, are therefore required to help eradicate the infection. Although the body's immune response may help control the infection, it is insufficient to eradicate the virus on its own without medical assistance, as evidenced by the reported rise in the treated group with better rates of spontaneous clearance. By directly preventing viral reproduction or strengthening the immune system's ability to fight the virus, treatment speeds up viral clearance. This rise suggests that treatment and spontaneous clearance work together to assist people move from the chronic period to recovery in a fractional-order model that takes memory and the historical development of infection into consideration. Over time, the population's response to treatment is influenced by biological memory, or the lasting effects of previous illnesses; treated people gain from both therapeutic interventions and any residual. Hence, the rise in the treated population reflects how treatment supplements the body's ability to clear the virus, particularly in chronic cases where spontaneous clearance alone would be insufficient for complete recovery.

Fig. 5 depicts the behaviour of a recovered population under various fractional orders, and the plot demonstrates that raising the values of spontaneous clearance results in an increase in the recovered population. More people are able to fight off the virus and reach a fully healed state as the ability for spontaneous clearance rises.This rise in recovery is especially apparent in immune-responsive populations, where spontaneous clearance successfully stops the virus from developing a

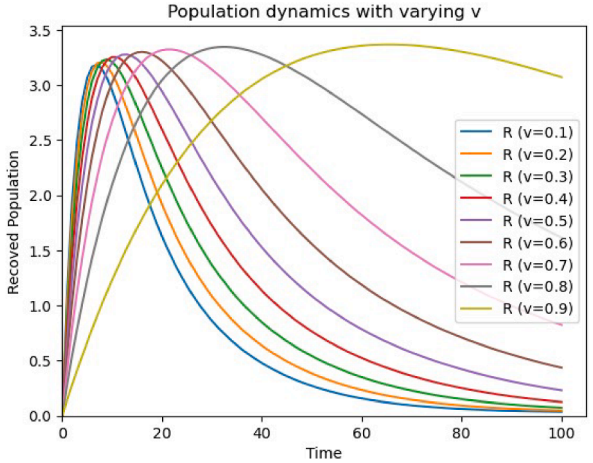

Fig. 5. Simulation of the effects of fractional order on Recovered population of HBV dynamics.

chronic, long-term presence. The memory effect and genetic characteristics of disease progression are incorporated into fractional-order models, which represent the long-term dynamics of how the immune system retains previous infections and progressively improves its ability to eradicate the virus. Individuals who have been exposed to the virus or have gained partial immunity may recover more quickly when spontaneous clearance rates are higher, contributing to a larger recovered population.The biological interpretation of this link is that innate immunological strength and the body's capacity to generate a sustained, effective response are important factors in a population's capacity to recover from illness, in addition to treatment or intervention. Consequently, a higher spontaneous clearance speeds up recovery, moving more people from the chronic or infected groups to the recovered group and ultimately aiding in an overall reduction in disease burden.

The fractional order $\upsilon \in (0, 1]$ plays a critical role in modulating how strongly past states influence the current behavior of the system. Biologically, lower values of υ reflect stronger memory effects, representing populations where immune responses or chronic disease progression are influenced by long-term exposure or treatment history. For instance, in HBV, memory effects may arise from persistent viral presence or latent immune activation. As $\upsilon \to 1$, the system approximates a classical memoryless (ODE) model, while lower values reflect chronic conditions with long-term consequences.

6. Conclusion

In conclusion, this study on mathematical modelling of HBV epidemics, which is based on vital dynamics as described in (Oludoun et al., 2021), provides a better understanding of the intricate interplay between infection progression and spontaneous clearance across diverse population groups. The findings show that under fractional-order models, which reflect the historical memory of disease progression, spontaneous clearance is crucial in determining the dynamics of susceptible, chronic, treated, and recovered populations. In the susceptible population, spontaneous clearance steadily reduces the number of people susceptible to HBV infection as they either clear the virus naturally or develop resistance, lowering overall vulnerability. Furthermore, the combination of treatment and spontaneous clearance greatly increases the treated population, demonstrating that medical interventions and the immune system's natural response work together to reduce persistent infection. The importance of spontaneous clearance is most visible in the recovered population, where greater clearance values result in higher recovery rates, demonstrating the immune system's ability to remove the infection over time, especially when it adapts to the virus. The use of fractional-order models, which include memory effects, emphasises that recovery is a time-dependent process impacted by prior exposure and the body's adaptive immunological response.

This study reveals that spontaneous clearance, when considered under a fractional-order framework, provides nuanced insights into HBV dynamics. In contrast to classical models, the incorporation of memory effects allows us to observe how past immune responses can modulate future susceptibility, treatment success, or failure. Notably, the increase in chronic infection despite rising clearance probabilities underscores that spontaneous clearance alone is insufficient in long-term viral suppression for chronic cases highlighting the importance of sustained treatment. The simulations demonstrate that even as spontaneous clearance improves, its effects are more pronounced in the susceptible and recovered populations than in chronic carriers. This observation reflects a biological truth: spontaneous clearance is often more effective in acute phases or early infection. By explicitly modeling these interactions, the fractional framework offers richer predictive power for designing intervention strategies that combine immunization, early diagnosis, and timely antiviral treatment. While promising, the model omits age structure, spatial heterogeneity, and reinfection pathways, which could affect HBV persistence. Future work could extend this model to address these complexities, possibly integrating Bayesian inference for parameter calibration against empirical data.

CRediT authorship contribution statement

O.Y. Oludoun: Writing – review & editing, Writing – original draft, Conceptualization. **O. Abiodun:** Formal analysis. **B. Gbadamosi:** Software, Data curation. **J.K. Oladejo:** Methodology. **E.I. Akinola:** Investigation. **O.N. Emuoyibofarhe:** Writing – review & editing. **O. Adebimpe:** Writing – review & editing.

Declaration of competing interest

The authors declare that there are no conflict of interest and funding available for this research

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