

Modeling and Analysis of SEIR Model of Hepatitis-B Virus via Fibonacci Wavelets

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Abstract. This study aims to examine the dynamics of susceptible, exposed, infected, and recovered individuals in the context of hepatitis-B model. This is achieved by developing an efficient numerical scheme based on Fibonacci wavelets for solving the hepatitis-B model. The proposed method employs operational matrices derived from Fibonacci wavelets to convert the given system of differential equations into a system of algebraic equations, solved via the Newton-Raphson method in MATLAB. The effects of hepatocyte death and virus production rates on healthy cells, infected cells, and viral load are analyzed to capture key aspects of disease progression. Comparative analysis with ND solve and Haar wavelet methods demonstrates that the proposed method yields accurate and efficient results. The numerical findings confirm the potential of Fibonacci wavelets as a reliable computational tool for studying infectious disease dynamics.

1. INTRODUCTION

Hepatitis B virus (HBV) is a significant global health concern, affecting over 350 million individuals worldwide. The infection poses serious risks, including liver fibrosis, hepatocellular carcinoma, and other severe liver diseases [1]. The World Health Organisation estimated in 2019 that approximately 296 million people were chronically infected, with 1.5 million new cases occurring annually [2]. The virus belongs to the Hepadnaviridae family and carries a circular, partially double-stranded DNA genome. HBV primarily spreads through exposure to infected blood,

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mother-to-child transmission, unprotected sexual contact, and organ transplantation. After entering the body, the virus specifically targets the liver, causing dysfunction and potentially leading to long-term complications. Despite effective vaccines and antiviral treatments in high-endemic areas, migration and population dynamics continue to influence infection rates in low-endemic regions [3]. While acute infections may be asymptomatic, chronic infections significantly increase the risk of liver cirrhosis and liver cancer. Treatment for chronic HBV includes antiviral medications aimed at slowing disease progression, while acute cases often require supportive care [4–6].

The fundamental model of viral infection dynamics includes three key variables: uninfected target cells S , infected cells E , and free virus particles I [9, 10]:

$$\left. \begin{aligned} \frac{dS}{dt} &= \alpha - d_T S - \varphi IS \\ \frac{dE}{dt} &= \varphi IS - \delta E \\ \frac{dI}{dt} &= -cI + rE \end{aligned} \right\} \quad (1.1)$$

In this framework, uninfected cells become infected upon interaction with virus particles at a rate proportional to φSI , while infected cells die at a rate δE . Free virus particles are produced by infected cells at a rate rE , and they are cleared from the system at a rate cI . The average lifespan of an infected cell is $1/\delta$, and for free virus particles, it is $1/c$. On average, each infected cell generates r/δ virus particles during its lifetime [15]. Uninfected cells are generated at a constant rate λ and die at a natural rate $d_T S$, leading to a stable population of uninfected cells in the absence of infection. The model (1.1) was later extended by Nowak et al. [7] by incorporating the immune response, specifically the activity of cytotoxic T lymphocytes (CTLs), represented by the variable R . CTLs recognise and eliminate infected cells, thus contributing to the reduction of viral load. Their proliferation is stimulated by the presence of infected cells, growing at a rate sER , while they decay at a rate σR in the absence of stimulation. Infected cells are eliminated by CTLs at a rate sER . The parameters s and a describe the responsiveness of CTLs to infection and the rate at which they kill infected cells, respectively. By incorporating the immune response, the modified form of the model (1.1) becomes [7]:

$$\left. \begin{aligned} \frac{dS}{dt} &= \lambda - d_T S - \varphi IS \\ \frac{dE}{dt} &= \varphi IS - \delta E - aER \\ \frac{dI}{dt} &= -cI + rE \\ \frac{dR}{dt} &= sER - \sigma R \end{aligned} \right\} \quad (1.2)$$

This extended model captures essential aspects of virus-immune interactions and offers insight into how the immune system contributes to controlling infection.

For a better understanding of the complex immune responses, the model (1.2) is modified by Pang et al. [8] by introducing a specialized function that reflects both antigen-driven stimulation and the contribution of specific precursor CTL cells exported from the thymus. This replaces the previous CTL proliferation term, sER , used in model (1.2). Moreover, they also incorporate noncytolytic mechanisms by which CTLs contribute to the clearance of infected cells. These enhancements lead us to the following system of differential equations [8]:

$$\left. \begin{aligned} \frac{dS}{dt} &= \alpha - \varphi IS - d_T S + bER \\ \frac{dE}{dt} &= \varphi IS - (a + b)ER - \delta E \\ \frac{dI}{dt} &= -cI + rE \\ \frac{dR}{dt} &= \rho - \sigma R + \frac{sER}{\epsilon + E} \end{aligned} \right\}, \quad (1.3)$$

with initial conditions $S(0) = S_0$, $E(0) = E_0$, $I(0) = I_0$, and $R(0) = R_0$. Moreover, the detailed description of parameters used in the model (1.3) are tabulated in Table 1. The non-cytolytic removal of infected cells is represented by the term $\frac{sER}{\epsilon + E}$ which illustrates a saturating response as infected cell levels increase. Additionally, we assume that all parameters are strictly positive, and that the death rate of uninfected hepatocytes is not greater than that of infected ones; that is, $d_T \leq a$.

Parameter	Meaning
S_0	initial population of $S(t)$
E_0	initial population of $E(t)$
I_0	initial population of $I(t)$
R_0	initial population of $R(t)$
α	Hepatocytes production rate
d_T	Hepatocytes death rate
φ	transmission factor
δ	Death rate of infected hepatocytes
a	Return to uninfected cell rate
b	Infected cell killing rate by CTL response
r	Virus production rate
c	Decay rate
s	CTL stimulation rate
ϵ	Viral load
ρ	Antigenic stimulation rate in decay absence
σ	Export of precursor CTL cells

TABLE 1. Table of numerical values

The dynamics of this model describe the interaction between uninfected liver cells, infected cells, the hepatitis B virus (HBV), and cytotoxic T-lymphocyte (CTL) immune response. Uninfected cells become infected upon exposure to the virus, while the immune system, particularly CTL cells, works to eliminate infected cells and control viral replication. The model captures the interplay of these compartments to understand HBV progression and immune response effectiveness. This model has been rigorously studied from time to time through the inclusion of various parameters. For instance, Danane et al. [20] developed a fractional differential model incorporating antibody immunity and hepatocyte interactions, exploring HBV dynamics using fractional calculus. Mehmood et al. [19] introduced a non-linear saturated incidence rate in an epidemic model and applied Galerkin-Petrov and Runge-Kutta techniques for numerical solutions. Wang et al. [18] introduced a diffusive HBV model with logistic proliferation terms for both infected and uninfected cells. Hoang [17] proposed a non-standard finite difference method to study the long-term dynamics of HBV infection, demonstrating its advantages over traditional numerical methods. Kumar et al. [21] extended HBV modeling by incorporating an asymptomatic carrier class using fractional-order operators. Kumbinarasaiah et al. [16] have analyzed the immune response of CTL cells by modifying the fourth equation, which represents their interaction with infected cells. Other studies have applied stochastic approaches to analyze fluctuations in HBV persistence and immune responses, demonstrating how external factors such as environmental changes influence disease transmission and recovery [11–14].

Wavelet theory has made significant advancements in numerical analysis over the past few decades, leading to the development of efficient computational algorithms. Wavelet-based numerical methods have gained prominence over traditional approaches, attracting considerable interest from researchers in science and engineering. Among these, wavelet-based collocation methods have become widely recognised due to their straightforward implementation, ease of computation, and rapid convergence. Additionally, they are well-suited for computer-based applications, eliminating the need to compute the inverse of the wavelet matrix and reducing computational time by reusing major components of the wavelet operational matrix. Various types of linear and non-linear differential equations have been solved using different wavelet families, including Haar, Legendre, Chebyshev, Bernoulli, Euler, Hermite, Gegenbauer, and Fibonacci wavelets [26–33].

Keeping the exciting properties and prolific advantages of wavelet-based numerical methods over the other existing numerical methods in hindsight, we are deeply motivated to solve the hepatitis B infection model (1.3) by employing Fibonacci wavelet methods. To the best of our knowledge, there is no available evidence that wavelets have been applied to solve the hepatitis B infection model on the interval $[0, t]$. This study introduces an efficient numerical approach utilizing compactly supported Fibonacci wavelets to analyze the behavior of compartments in this model. The accuracy and effectiveness of the proposed method is validated by comparing the obtained results with other existing methods. The key contributions of this work include:

- Developing a new numerical method based on Fibonacci wavelets for solving the hepatitis B infection model.
- Constructing operational integration matrices based on Fibonacci wavelets using the Chen and Hsiao approach.
- Comparing the obtained results with the existing results available in open literature.
- Analyzing the impact of various parameters on the compartments of the model graphically.

The structure of this article is organized as follows. Section 2 presents the construction of the operational integration matrices for Fibonacci wavelets using the technique of Chen and Hsiao. Section 3 forms the central part of the study, where the hepatitis B infection model is solved. The main results appear in Section 4, which provides detailed numerical simulations. Finally, Section 5 offers concluding remarks.

2. WAVELETS AND FUNCTION APPROXIMATION

This section forms the central part of the article, as it develops the operational matrices of integration constructed through Fibonacci wavelets. To achieve this, we adopt the Chen-Hsiao procedure [22], which provides a systematic framework for generating these matrices and enables their use in converting differential equations into algebraic systems.

3.1. Basics of Wavelets

Wavelets form a broad family of functions produced by dilating and translating a basic generating function known as the *mother wavelet*. The property that formally characterizes a mother wavelet is the notion of *admissibility*, which guarantees that a signal can be accurately reconstructed from its wavelet transform. A function $\psi \in L^2(\mathbb{R})$ is considered an admissible wavelet if it satisfies

$$C_\psi = 2\pi \int_{\mathbb{R}} \frac{|(\mathcal{F}\psi)(\omega)|^2}{|\omega|} d\omega < \infty. \quad (2.1)$$

The condition in (2.1) ensures that $(\mathcal{F}\psi)(\omega)$ approaches 0 as $\omega \rightarrow 0$. When $(\mathcal{F}\psi)(\omega)$ is continuous, this requirement reduces to $(\mathcal{F}\psi)(0) = 0$, which is equivalent to

$$\int_{\mathbb{R}} \psi(t) dt = 0.$$

Thus, ψ must be an oscillatory function with zero mean, giving wavelets their natural localization in time and their characteristic wave-like appearance. This explains why the function ψ is commonly referred to as a *wavelet*.

By introducing translations and dilations to the mother wavelet $\psi(t)$, one obtains a collection of functions called *daughter wavelets*, defined by

$$\psi_{a,b}(t) = |a|^{-1/2} \psi\left(\frac{t-b}{a}\right), \quad a \in \mathbb{R}^+, b \in \mathbb{R}, \quad (2.2)$$

where a denotes the scale parameter and b represents the translation parameter. The parameter a determines the degree of compression or expansion of the wavelet: for $0 < a < 1$, the function

$\psi_{a,b}(t)$ becomes a compressed version of $\psi(t)$, while $a > 1$ produces a stretched or dilated version. The parameter b shifts the wavelet along the time axis.

A notable example of a continuous wavelet is the *Mexican Hat* (or Ricker) wavelet, defined by

$$\psi(t) = (1 - t^2) e^{-t^2/2}, \quad (2.3)$$

which is derived as the second derivative of the Gaussian function. Its smooth shape and strong localization properties make it useful in feature detection, geophysical analysis, and image processing.

On the other hand, the multiresolution analysis (MRA) based wavelets provide information on objects at different resolutions, such as small objects being captured at high resolution and large objects at low resolution. In an MRA, a function f is expressed as the limit of a sequence of approximations, each corresponding to a finer resolution level. The fundamental concept behind MRA is to break down the function space into a hierarchy of nested subspaces $V_j \subset V_{j+1}$, where each subspace represents a different level of detail. This nested structure ensures that the subspace V_{j+1} contains all rescaled versions of functions from V_j . Through this framework, a function can be analyzed across multiple scales, with each subspace capturing specific features of f . Mathematically, an MRA is defined by a collection $V_j : j \in \mathbb{Z}$ of closed subspaces of $L^2(\mathbb{R})$ satisfying the following conditions:

- (i). Nestedness: The subspaces are nested such that $V_j \subset V_{j+1}$; for all $j \in \mathbb{Z}$
- (ii). Density: The union of all subspaces is dense in $L^2(\mathbb{R})$, i.e., $\overline{\cup_{j \in \mathbb{Z}} V_j} = L^2(\mathbb{R})$
- (iii). Separation: The intersection of all subspaces contains only the zero function: $\cap_{j \in \mathbb{Z}} V_j = 0$.
- (iv). Scaling: A function $g(\tau) \in V_j$ if and only if its scaled version $g(2\tau) \in V_{j+1}$; for every $j \in \mathbb{Z}$
- (v). Orthonormal basis: There exists a scaling function $\psi \in V_0$ such that the set $\psi(t - k), k \in \mathbb{Z}$ forms an orthonormal basis for V_0 .

A classic example of a wavelet system arising from a multiresolution analysis is the *Meyer wavelet*, known for its smoothness and compact support in the frequency domain. Beyond this, many other wavelet families are constructed within the MRA framework, including the Franklin wavelet, Shannon wavelet, Battle-Lemarie wavelet, Chebyshev and Legendre wavelets, Gegenbauer wavelet, as well as more recent families such as the Fibonacci and Vieta-Fibonacci wavelets [23].

In the sequel, we aim to construct the operational matrices of integration associated with Fibonacci wavelets to obtain the numerical solution of the hepatitis B virus model. To begin with, we present an overview of the fundamental notion of wavelets

2.1. Fibonacci Wavelets and Function Approximation

This subsection is devoted to developing the Fibonacci wavelets by making use of the Fibonacci polynomials. For any $t \in \mathbb{R}^+$, these polynomials are defined recursively through

$$\mathcal{F}_{m+2}(t) = t\mathcal{F}_{m+1}(t) + \mathcal{F}_m(t), \quad (2.4)$$

together with the initial conditions $\mathcal{F}_0(t) = 0$ and $\mathcal{F}_1(t) = 1$. An equivalent closed-form representation of the Fibonacci polynomials is

$$\mathcal{F}_{m-1}(t) = \frac{\lambda^m - \mu^m}{\lambda - \mu}, \quad m \geq 1, \quad (2.5)$$

where λ and μ denote the roots of the characteristic equation $\eta^2 - t\eta - 1 = 0$ associated with the recurrence relation. A further useful expression for these polynomials is their power-series form [24]:

$$\mathcal{F}_m(t) = \sum_{i=0}^{\lfloor m/2 \rfloor} \binom{m-i}{i} t^{m-2i}, \quad m \geq 0. \quad (2.6)$$

The Fibonacci wavelets defined on the interval $[0, 1]$ are introduced in [25] as

$$\psi_{n,m}(t) = \begin{cases} \frac{2^{(k-1)/2}}{\sqrt{W_m}} \mathcal{F}_m(2^{k-1}t - n + 1), & \frac{n-1}{2^{k-1}} \leq t < \frac{n}{2^{k-1}}, \\ 0, & \text{otherwise,} \end{cases} \quad (2.7)$$

where k and n denote the resolution level and translation index, respectively, with $k = 1, 2, \dots$ and $n = 1, 2, \dots, 2^{k-1}$. In (2.7), the function $\mathcal{F}_m(t)$ represents the Fibonacci polynomial of degree m , obtained from (2.6). The coefficient $1/\sqrt{W_m}$ provides normalization and is computed as

$$\begin{aligned} W_m &= \int_0^1 F_m(t) F_m(t) dt, \quad m = 0, 1, \dots, M-1, \\ &= \sum_{i=0}^{\lfloor m/2 \rfloor} \sum_{j=0}^{\lfloor m/2 \rfloor} \binom{m-i}{i} \binom{m-j}{j} (2(-i-j) + n + m + 1)^{-1}. \end{aligned} \quad (2.8)$$

For example, choosing $k = 2$ and $M = 3$ yields the Fibonacci wavelet family

$$\left. \begin{aligned} \psi_{1,0}(t) &= \sqrt{2}, \\ \psi_{1,1}(t) &= 2\sqrt{6}t, \\ \psi_{1,2}(t) &= \sqrt{\frac{15}{14}}(1 + 4t^2), \end{aligned} \right\} t \in \left[0, \frac{1}{2}\right) \quad (2.9)$$

$$\left. \begin{aligned} \psi_{2,0}(t) &= \sqrt{2}, \\ \psi_{2,1}(t) &= \sqrt{6}(2t - 1), \\ \psi_{2,2}(t) &= \sqrt{\frac{30}{7}}(2t^2 - 2t + 1), \end{aligned} \right\} t \in \left[\frac{1}{2}, 1\right)$$

Any function $g(t) \in L^2[0, 1]$ may be approximated by the Fibonacci wavelet expansion

$$g(t) \approx g_{k,M}(t) = \sum_{n=1}^{2^{k-1}} \sum_{m=0}^{M-1} h_{n,m} \psi_{n,m}(t), \quad (2.10)$$

where the coefficients $h_{n,m}$ are obtained from

$$h_{n,m} = \langle g, \psi_{n,m} \rangle = \int_0^1 g(t) \psi_{n,m}(t) dt. \quad (2.11)$$

The matrix representation corresponding to (2.10) is

$$G = C^T \Psi(t), \quad (2.12)$$

where the coefficient vector H is arranged as

$$C = [c_{1,0}, c_{1,1}, \dots, c_{1,M-1}, c_{2,0}, c_{2,1}, \dots, c_{2,M-1}, \dots, c_{2^{k-1},0}, \dots, c_{2^{k-1},M-1}]^T. \quad (2.13)$$

The Fibonacci wavelet matrix $\Psi(t)$ of size $1 \times 2^{k-1}M$ is

$$\Psi(t) = [\psi_{1,0}, \psi_{1,1}, \dots, \psi_{1,M-1}, \psi_{2,0}, \dots, \psi_{2,M-1}, \dots, \psi_{2^{k-1},0}, \dots, \psi_{2^{k-1},M-1}]^T. \quad (2.14)$$

The collocation points used for constructing the Fibonacci wavelet approximations are chosen as

$$t_\ell = \frac{2\ell - 1}{2^k M}, \quad \ell = 1, 2, \dots, 2^{k-1}M. \quad (2.15)$$

2.2. Operational Matrices of Integration via Fibonacci Wavelets

To derive the operational matrices of integration corresponding to the Fibonacci wavelets in (2.7), we follow the Chen-Hsiao procedure [22], which approximates

$$\int_0^t \psi_{n,m}(s) ds \approx Q \psi_{n,m}(t), \quad (2.16)$$

where Q is the operational matrix of integration of size $2^{k-1}M \times 2^{k-1}M$.

For the specific case $k = 2$ and $M = 3$, integrating the wavelets in (2.9) at the collocation points in (2.15) leads to

$$\begin{aligned} \int_0^t \psi_{1,0}(x) dx &= \left(0, \frac{\sqrt{3}}{6}, 0, \frac{1}{2}, 0, 0\right)^T \Psi_{6 \times 6}(t), \\ \int_0^t \psi_{1,1}(x) dx &= \left(-\frac{\sqrt{3}}{4}, 0, \frac{\sqrt{35}}{10}, \frac{\sqrt{3}}{4}, 0, 0\right)^T \Psi_{6 \times 6}(t), \\ \int_0^t \psi_{1,2}(x) dx &= \left(-\frac{29\sqrt{105}}{1680}, \frac{\sqrt{35}}{35}, \frac{1}{4}, \sqrt{\frac{5}{21}}, 0, 0\right)^T \Psi_{6 \times 6}(t), \\ \int_0^t \psi_{2,0}(x) dx &= \left(0, 0, 0, 0, \frac{\sqrt{3}}{6}, 0\right)^T \Psi_{6 \times 6}(t), \\ \int_0^t \psi_{2,1}(x) dx &= \left(0, 0, 0, -\frac{\sqrt{3}}{4}, 0, \frac{\sqrt{35}}{10}\right)^T \Psi_{6 \times 6}(t), \\ \int_0^t \psi_{2,2}(x) dx &= \left(0, 0, 0, -\frac{29\sqrt{105}}{1680}, \frac{\sqrt{35}}{35}, \frac{1}{4}\right)^T \Psi_{6 \times 6}(t). \end{aligned}$$

Using (2.16), this yields

$$\int_0^t \Psi_{6 \times 1}(x) dx \approx Q_{6 \times 6} \Psi_6(t), \quad (2.17)$$

where the matrix $Q_{6 \times 6}$ is given by

$$\Psi_{6 \times 6} = \begin{pmatrix} 0 & \frac{\sqrt{3}}{6} & 0 & \frac{1}{2} & 0 & 0 \\ -\frac{\sqrt{3}}{4} & 0 & \frac{\sqrt{35}}{10} & \frac{\sqrt{3}}{4} & 0 & 0 \\ -\frac{29\sqrt{105}}{1680} & \frac{\sqrt{35}}{35} & \frac{1}{4} & \sqrt{\frac{5}{21}} & 0 & 0 \\ 0 & 0 & 0 & 0 & \frac{\sqrt{3}}{6} & 0 \\ 0 & 0 & 0 & -\frac{\sqrt{3}}{4} & 0 & \frac{\sqrt{35}}{10} \\ 0 & 0 & 0 & -\frac{29\sqrt{105}}{1680} & \frac{\sqrt{35}}{35} & \frac{1}{4} \end{pmatrix}. \quad (2.18)$$

3. FIBONACCI WAVELET METHOD FOR SOLVING THE SEIR MODEL OF HEPATITIS-B

In this section, we aim to solve the SEIR model of hepatitis-B given by the equation using Fibonacci wavelets, incorporating key factors such as the hepatitis B virus (HBV), cytotoxic T lymphocyte (CTL) immune response, birth and death rates, and the dynamics between infected and uninfected hepatocytes. By applying Fibonacci wavelets, we efficiently capture the complex interactions between these components, enabling an efficient numerical solution that reflects the transmission and progression of the infection. To solve the SEIR epidemic model, it is essential to refer to the SEIR model of hepatitis-B, which is expressed as:

$$\left. \begin{aligned} \frac{dS}{d\tau} &= \alpha - \phi IS - d_T S + bER \\ \frac{dE}{d\tau} &= \phi IS - (a + b)ER - \delta E \\ \frac{dI}{d\tau} &= -cI + rE \\ \frac{dR}{d\tau} &= \rho - \sigma R + \frac{sER}{\epsilon + E} \end{aligned} \right\}, \quad (3.1)$$

Expressing the highest derivatives appearing in the model (3.1) in terms of the Fibonacci wavelet basis gives:

$$\left. \begin{aligned} \frac{dS}{dt} &= C_1^T \Psi(\tau) \\ \frac{dE}{dt} &= C_2^T \Psi(\tau) \\ \frac{dI}{dt} &= C_3^T \Psi(\tau) \\ \frac{dR}{dt} &= C_4^T \Psi(\tau) \end{aligned} \right\}, \quad (3.2)$$

where C_1^T , C_2^T , C_3^T , and C_4^T are the unknown Fibonacci wavelet coefficients. On integrating both sides of equation (3.2) with respect to t , we obtain

$$\left. \begin{aligned} S(t) &= S(0) + C_1^T Q(\tau) \\ E(t) &= E(0) + C_2^T Q(\tau) \\ I(t) &= I(0) + C_3^T Q(\tau) \\ R(t) &= R(0) + C_4^T Q(\tau) \end{aligned} \right\}. \quad (3.3)$$

By substituting (3.2) and (3.3) into equation (3.1), the following system of algebraic equations is obtained:

$$\left. \begin{aligned} C_1^T \Psi(\tau) &= \alpha - \varphi \left(I(0) + C_3^T Q(\tau) \right) \left(S(0) + C_1^T Q(\tau) \right) - d_T \left(S(0) + C_1^T Q(\tau) \right) + b \left(E(0) + C_2^T Q(\tau) \right) \\ &\quad \left(R(0) + C_4^T Q(\tau) \right) \\ C_2^T \Psi(\tau) &= \varphi \left(I(0) + C_3^T Q(\tau) \right) \left(S(0) + a Q(\tau) \right) + (a+b) \left(E(0) + C_2^T Q(\tau) \right) \left(R(0) + C_4^T Q(\tau) \right) - \delta C_2^T Q(\tau) \\ C_3^T \Psi(\tau) &= -c \left(I(0) + C_3^T Q(\tau) \right) + r \left(E(0) + C_2^T Q(\tau) \right) \\ C_4^T \Psi(\tau) &= \rho - \sigma \left(R(0) + C_4^T Q(\tau) \right) - \gamma \left(I(0) + c Q(\tau) \right) + \frac{s \left(E(0) + C_2^T Q(\tau) \right) \left(R(0) + C_4^T Q(\tau) \right)}{\epsilon + \left(E(0) + C_2^T Q(\tau) \right)} \end{aligned} \right\}. \quad (3.4)$$

After simplifying the expression (3.4) and utilizing the collocation points (2.15), we arrive at:

$$\left. \begin{aligned} C_1^T \Psi(\tau) &+ \varphi \left(I(0) S(0) + I(0) C_1^T Q(\tau) + S(0) C_3^T Q(\tau) + (C_3^T Q(\tau))(C_1^T Q(\tau)) \right) \\ &+ d_T \left(S(0) + C_1^T Q(\tau) \right) - b \left(E(0) R(0) + E(0) C_4^T Q(\tau) + R(0) C_2^T Q(\tau) + (C_2^T Q(\tau))(C_4^T Q(\tau)) \right) - \alpha = 0 \\ C_2^T \Psi(\tau) &- \varphi \left(I(0) S(0) + a I(0) Q(\tau) + S(0) C_3^T Q(\tau) + a (C_3^T Q(\tau)) Q(\tau) \right) \\ &- (a+b) \left(E(0) R(0) + E(0) C_4^T Q(\tau) + R(0) C_2^T Q(\tau) + (C_2^T Q(\tau))(C_4^T Q(\tau)) \right) + \delta C_2^T Q(\tau) = 0 \\ C_3^T \Psi(\tau) &+ c I(0) + c C_3^T Q(\tau) - r E(0) - r C_2^T Q(\tau) = 0 \\ C_4^T \Psi(\tau) &+ \sigma R(0) + \sigma C_4^T Q(\tau) + \gamma I(0) + \gamma c Q(\tau) \\ &- \frac{s \left(E(0) R(0) + E(0) C_4^T Q(\tau) + R(0) C_2^T Q(\tau) + (C_2^T Q(\tau))(C_4^T Q(\tau)) \right)}{\epsilon + E(0) + C_2^T Q(\tau)} - \rho = 0 \end{aligned} \right\}. \quad (3.5)$$

Using the collocation points $\tau_\ell = \frac{2\ell-1}{2^{k+1}M}$, $\ell = 1, 2, \dots, 2^k M$, the preceding system of equations is transformed into its corresponding discrete form:

$$\left. \begin{aligned}
& C_1^T \Psi(\tau_\ell) + \varphi \left(I(0)S(0) + I(0)C_1^T Q(\tau_\ell) + S(0)C_3^T Q(\tau_\ell) + (C_3^T Q(\tau_\ell))(C_1^T Q(\tau_\ell)) \right) \\
& \quad + d_T \left(S(0) + C_1^T Q(\tau_\ell) \right) - b \left(E(0)R(0) + E(0)C_4^T Q(\tau_\ell) + R(0)C_2^T Q(\tau_\ell) + (C_2^T Q(\tau_\ell))(C_4^T Q(\tau_\ell)) \right) - \alpha = 0 \\
& C_2^T \Psi(\tau_\ell) - \varphi \left(I(0)S(0) + aI(0)Q(\tau_\ell) + S(0)C_3^T Q(\tau_\ell) + a(C_3^T Q(\tau_\ell))Q(\tau_\ell) \right) \\
& \quad - (a+b) \left(E(0)R(0) + E(0)C_4^T Q(\tau_\ell) + R(0)C_2^T Q(\tau_\ell) + (C_2^T Q(\tau_\ell))(C_4^T Q(\tau_\ell)) \right) + \delta C_2^T Q(\tau_\ell) = 0 \\
& C_3^T \Psi(\tau_\ell) + cI(0) + cC_3^T Q(\tau_\ell) - rE(0) - rC_2^T Q(\tau_\ell) = 0 \\
& C_4^T \Psi(\tau_\ell) + \sigma R(0) + \sigma C_4^T Q(\tau_\ell) + \gamma I(0) + \gamma c Q(\tau_\ell) \\
& \quad - \frac{s \left(E(0)R(0) + E(0)C_4^T Q(\tau_\ell) + R(0)C_2^T Q(\tau_\ell) + (C_2^T Q(\tau_\ell))(C_4^T Q(\tau_\ell)) \right)}{\epsilon + E(0) + C_2^T Q(\tau_\ell)} - \rho = 0
\end{aligned} \right\}. \quad (3.6)$$

In equation (3.6) the only unknowns are C_1^T , C_2^T , C_3^T , and C_4^T . By solving this equation using Newton's iterative method in MATLAB, we can determine these unknown Fibonacci wavelet coefficients. After determining the unknown coefficients C_1^T , C_2^T , C_3^T , and C_4^T substituting them into equation (3.2) yields the required solutions $S(\tau)$, $E(\tau)$, $I(\tau)$ and $R(\tau)$ through the Fibonacci wavelet representation.

4. RESULTS AND DISCUSSION

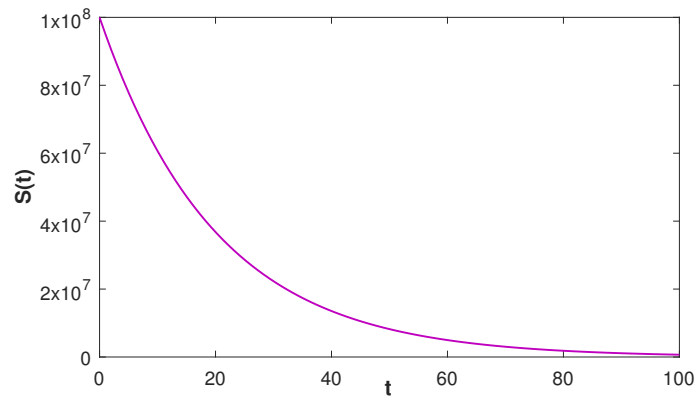
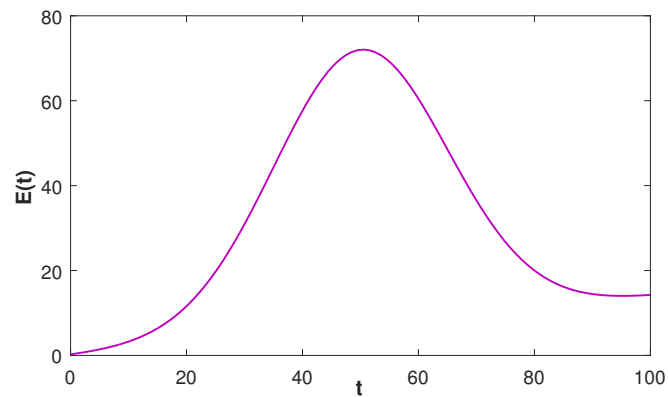
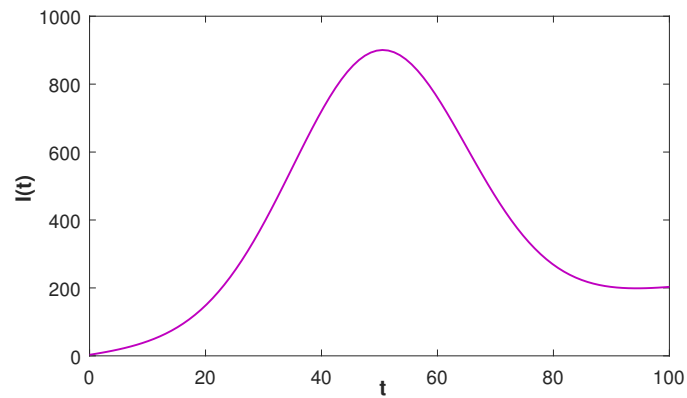
In this section, we aim to provide numerical simulations both graphically and in tabular form for different groups in the SEIR model of hepatitis-B: susceptible, exposed, infected, and recovered individuals. Numerical simulations provide a visual representation to understand how these groups change over time. We analyze the disease dynamics of these groups using Fibonacci wavelets, which are graphically illustrated in Figures 1-4. The numerical simulation is carried out for $M = 8$ and $k = 3$, and the numerical values of the parameters involved in the proposed SEIR epidemic model of hepatitis-B virus are given in Table 2.

Figure 1 illustrates the decline in the number of susceptible individuals as time progresses. It shows that over time, the susceptible population gradually decreases. Figures 2 and 3 illustrate the number of exposed and infected individuals, respectively. These figures suggest a classic epidemic curve where, at first, there is a rapid increase due to high rates of transmission or interaction among individuals, leading to a maximum population level (or peak). This peak marks where infection or exposure rates are highest, possibly due to factors like maximum susceptible population or optimal conditions for the spread. After reaching the peak, the figures show a gradual decline, which can be attributed to factors such as depletion of susceptible individuals, increased immunity (natural or acquired), or interventions that slow down the spread. Figure 4 depicts that the number of recovered individuals increases over time.

Parameter	Numerical Value
S_0	10^8
E_0	10^{-2}
I_0	0
R_0	100
α	1
d_T	$0.01 - 9 \times 10^{-4}$
φ	$1 \times 10^{-10} - 6.6 \times 10^{-9}$
δ	-0.19
a	0.17
b	-7.88
r	-162.6
c	-44.5
s	0.4
ϵ	0.0003
ρ	1.7×10^8
σ	427.1

TABLE 2. Table of numerical values

The sensitivity analysis reveals the impact of varying parameters on population dynamics. Increasing the hepatocyte death rate reduces the uninfected hepatocytes, infected hepatocytes, and HBV levels, as shown in Figures 5 to 7. Similarly, raising the virus production rate decreases uninfected hepatocytes as depicted in Figure 8. Figure 9 highlights the effect of the infection rate on the exposed population, while Figure 10 illustrates how the decay rate influences the infected population. Finally, to demonstrate the precision and accuracy of the proposed method, we present a comparison of the obtained results in Tables 3-6 with those from ND Solve and Haar wavelets found in the existing literature.

FIGURE 1. Plot of susceptible class via Fibonacci wavelets with time t .FIGURE 2. Plot of exposed class via Fibonacci wavelets with time t .FIGURE 3. Plot of infected class via Fibonacci wavelets with time t .

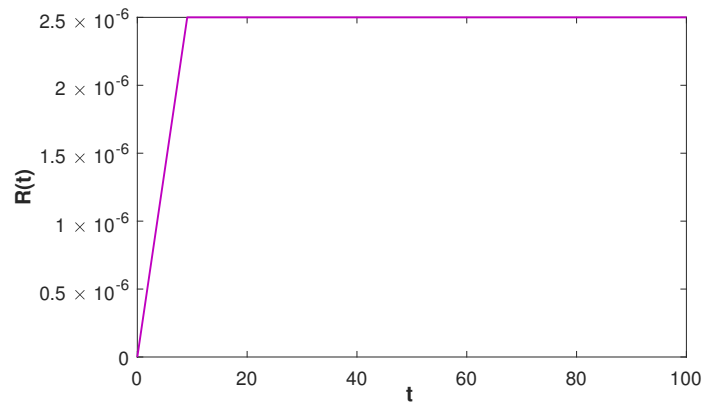
FIGURE 4. Plot of recovered class via Fibonacci wavelets with time t .

TABLE 3. Comparison of the proposed method and other numerical methods for the susceptible class.

$t \downarrow$	ND Solve [16]	HWM [16]	FWM
0.1	9.9610×10^7	9.9610×10^7	9.9610×10^7
0.2	9.9223×10^7	9.9223×10^7	9.9224×10^7
0.3	9.8836×10^7	9.8836×10^7	9.8837×10^7
0.4	9.8452×10^7	9.8452×10^7	9.8451×10^7
0.5	9.8068×10^7	9.8068×10^7	9.8068×10^7
0.6	9.7687×10^7	9.7687×10^7	9.7689×10^7
0.7	9.7306×10^7	9.7306×10^7	9.7306×10^7
0.8	9.6928×10^7	9.6928×10^7	9.6927×10^7
0.9	9.6550×10^7	9.6550×10^7	9.6549×10^7
1	9.6175×10^7	9.6175×10^7	9.6176×10^7

TABLE 4. Comparison of the proposed method and other numerical methods for the exposed class.

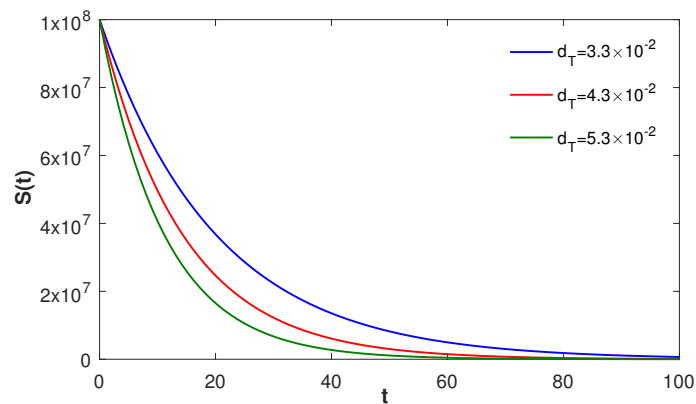
$t \downarrow$	ND Solve [16]	HWM [16]	FWM
0.1	0.0446	0.0446	0.0446
0.2	0.0767	0.0767	0.0765
0.3	0.1067	0.1067	0.1067
0.4	0.1348	0.1348	0.1349
0.5	0.1611	0.1611	0.1611
0.6	0.1859	0.1859	0.1859
0.7	0.2093	0.2093	0.2093
0.8	0.2315	0.2315	0.2314
0.9	0.2526	0.2526	0.2527
1	0.2728	0.2728	0.2728

TABLE 5. Comparison of the proposed method and other numerical methods for the infected class.

$t \downarrow$	ND Solve [16]	HWM [16]	FWM
0.1	9.3752	9.3752	9.375
0.2	8.8191	8.8191	8.8191
0.3	8.3251	8.3251	8.3252
0.4	7.8875	7.8875	7.8875
0.5	7.5011	7.5011	7.5011
0.6	7.1612	7.1612	7.1612
0.7	6.8636	6.8636	6.8636
0.8	6.6045	6.6045	6.6046
0.9	6.3804	6.3804	6.3804
1	6.1882	6.1882	6.1882

TABLE 6. Comparison of the proposed method and other numerical methods for the recovered class.

$t \downarrow$	ND Solve [16]	HWM [16]	FWM
0.1	$2.512352947048 \times 10^{-6}$	$2.512351221919 \times 10^{-6}$	$2.5123197781 \times 10^{-6}$
0.2	$2.5123529470648 \times 10^{-6}$	$2.5123561550655 \times 10^{-6}$	$2.5123321988404 \times 10^{-6}$
0.3	$2.5123529470713 \times 10^{-6}$	$2.5123497392000 \times 10^{-6}$	$2.5123491119278 \times 10^{-6}$
0.4	$2.5123529470747 \times 10^{-6}$	$2.5123546719901 \times 10^{-6}$	$2.5123546540367 \times 10^{-6}$
0.5	$2.5123529470786 \times 10^{-6}$	$2.5123529470854 \times 10^{-6}$	$2.5123529001487 \times 10^{-6}$
0.6	$2.5123529470794 \times 10^{-6}$	$2.5123512222818 \times 10^{-6}$	$2.5123512566201 \times 10^{-6}$
0.7	$2.5123529470802 \times 10^{-6}$	$2.5123561544618 \times 10^{-6}$	$2.5123401694333 \times 10^{-6}$
0.8	$2.512352947080 \times 10^{-6}$	$2.5123497398272 \times 10^{-6}$	$2.5123657169383 \times 10^{-6}$
0.9	$2.512352947080 \times 10^{-6}$	$2.512354671663 \times 10^{-6}$	$2.512346084416 \times 10^{-6}$
1	$2.512352947081 \times 10^{-6}$	$2.51237563827 \times 10^{-6}$	$2.51244318972 \times 10^{-6}$

FIGURE 5. Effect of hepatocyte death rate on $S(t)$.

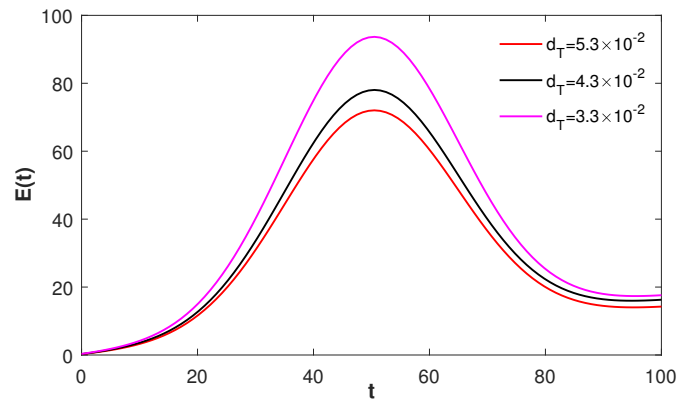


FIGURE 6. Impact of hepatocyte death Rate on $E(t)$.

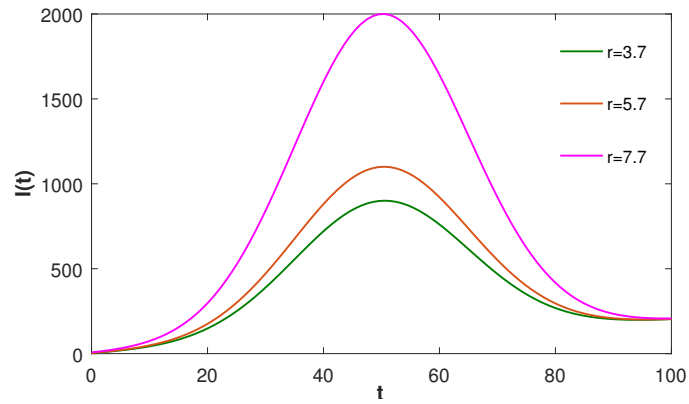


FIGURE 7. Impact of hepatocyte death rate on the $I(t)$.

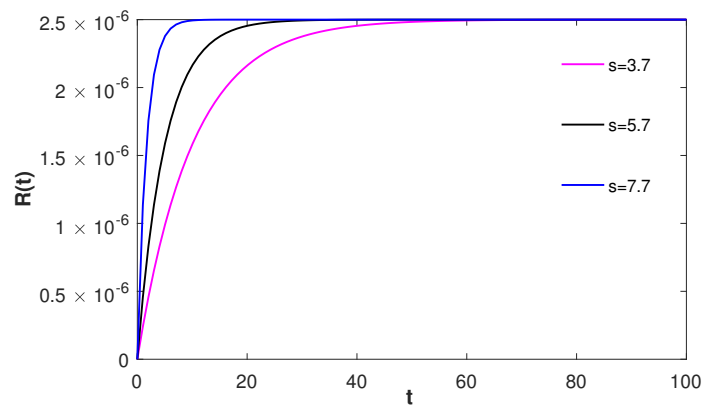
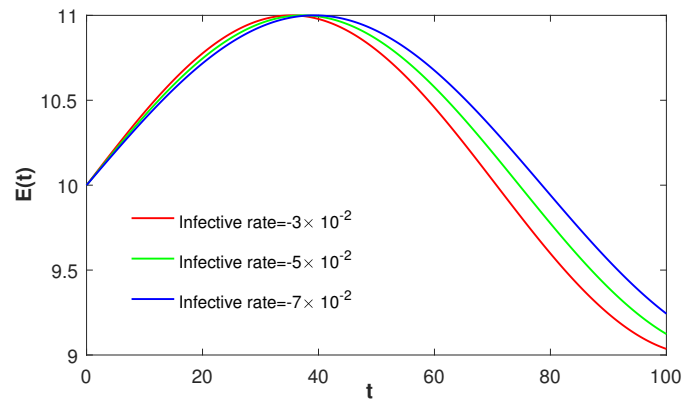
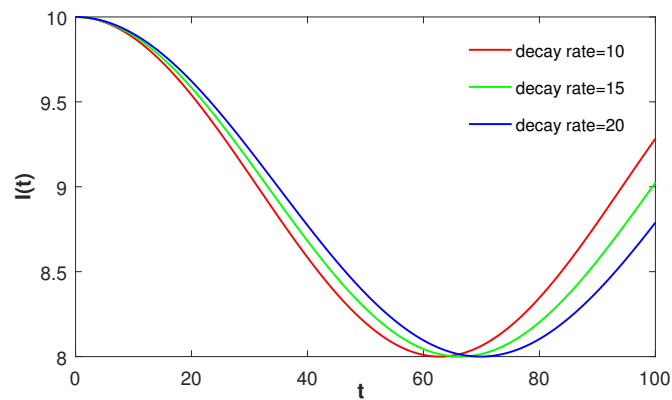


FIGURE 8. Impact of virus production rate on $R(t)$.

FIGURE 9. Impact of infective rate on $E(t)$.FIGURE 10. Impact of decay Rate on $I(t)$.

5. CONCLUSION

An efficient numerical scheme based on Fibonacci wavelets has been developed to investigate the dynamics of the hepatitis B virus. The main advantage of the proposed method is that it converts the given model into a system of nonlinear algebraic equations, which can be solved using any classical method, such as Newton-Raphson's method, the *fsolve* command in MATLAB, Broyden's iterative method, and many more. The method successfully captures the influence of hepatocyte death and virus production rates on uninfected cells, infected cells, and viral populations. Numerical results confirm that the proposed scheme achieves results consistent with the established methods, such as ND solve and Haar wavelets, while offering enhanced efficiency and accuracy. These outcomes demonstrate the effectiveness of Fibonacci wavelets in modelling complex biological systems and suggest their broader applicability in infectious disease dynamics.

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