EUROPEAN JOURNAL OF PURE AND APPLIED MATHEMATICS
2025, Vol. 18, Issue 4, Article Number 6682
ISSN 1307-5543 – ejpam.com
Published by New York Business Global

Modeling Dengue Dynamics Using Classical, Caputo Fractional, and Fractal Derivatives

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Abstract. Dengue is a major problem in Sudan. Dengue spreads in Gedaref, Sudan. In this work, we compare classical, Caputo fractional derivative, and fractal SIAR-SI pandemic models. We investigate the disease-free and endemic equilibrium stability, two important stable states of the proposed dynamical model. This study identifies the factors guiding a disease's persistence or extinction in the population. The model is fitted using real data from Gedaref, Sudan, and the Markov Chain Monte Carlo method is used for estimating parameters. The computed $\mathcal{R}_0 = 3.27$, indicating that the disease is endemic. The classical model fits the real data better than the other methods. The fractional model, involving Caputo derivatives, explains memory effects. Thus, they provide a more realistic formulation of disease transmission. The fractal model with Hausdorff derivatives offers a new direction for complicated transmission dynamics and is worthy of further research. The fractional and fractal models outperform the classical model by treating long-term dependency with fractional derivatives and disease spread accurately with fractal operators.

2020 Mathematics Subject Classifications: 26A33, 34A08, 92D30

Key Words and Phrases: Dengue fever, mathematical modeling, fractional derivatives, fractals, epidemiology, forecasting

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DOI: https://doi.org/10.29020/nybg.ejpam.v18i4.6682

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1. Introduction

Dengue fever is a viral infection spread by mosquitoes. The infection usually occurs in warm and subtropical regions with a high population density. The virus has four serotypes: DENV-1, DENV-2, DENV-3, and DENV-4. Dengue fever can appear in various ways, ranging from minor signs such as fever, headache, and joint pain to severe complications such as dengue hemorrhagic fever and dengue shock syndrome [1–5].

Dengue fever is a worldwide health issue. It causes millions of reported cases annually in Asia, Africa, and South America [6]. Weather factors like temperature, humidity, and rainfall influence viral spread due to their direct effect on the breeding of mosquitoes and virus reproduction. Dengue fever burst into epidemic proportions in eastern Sudan in 2023, specifically in Gedaref state, with morbidity and mortality [7]. The extremely high dengue fever epidemics in Gedaref are due to the interaction between social, environmental, and economic determinants. Climate change and the intensification of seasonal rains create favorable breeding conditions for mosquitoes. However, water courses and wetland areas represent breeding sites that contribute to the spread of the epidemic. Inadequate healthcare impacts the timely detection and care of new cases, increasing outbreaks' severity. These linked determinants explain the region's chronic and endemic dengue fever epidemic.

In 2023, Gedaref State recorded one of Sudan's highest dengue fever incidence rates. Thus, it is critical to investigate epidemiological characteristics of the disease spread in the region [8–11]. Mathematical models are of central importance to comprehending and predicting infectious disease transmission to enable public health authorities to design effective disease surveillance systems, respond to outbreaks, and formulate policy. The models utilize accurate data and computer methods to understand infection transmission, intervention effectiveness, and environmental factors that affect disease transmission [12–14].

Scientists have devised various modelling methods, each with advantages and disadvantages. Classical Susceptible-Infected-Recovered (SIR) and Susceptible-Exposed-Infected-Recovered (SEIR) are among the most commonly used integer-order models. While providing a straightforward framework for understanding disease spread, these models fail to capture the complexity of actual epidemics [15]. Fractional and fractal models emerged and are used to address these limitations. These models incorporate memory effects and improve prediction accuracy [16, 17].

Fractional-order models enhance conventional frameworks by incorporating non-integer derivatives. They are of special use in long-term epidemic forecasting, particularly for diseases with latency in immune response or with long incubation periods [16]. Fractional-order models were found to increase accuracy in prediction, particularly for vector-borne disease, which is influenced by changing mosquito populations and environmental conditions [18, 19]. Studies on dengue showed that fractional order predicted epidemic patterns more precisely than traditional models [20]. Fractional models, however, cannot account for time variations [21–23]. Fractal models account for time variations, assuming that disease transmission occurs in irregular patterns influenced by population density and vector mobility. Expanding epidemiological modeling into non-Euclidean space allows fractal

models to represent disease transmission in complex ecosystems accurately [24].

Fractal models improved epidemic predictions, particularly in densely populated urban areas where human mobility and clustering are critical elements in disease spread [23]. Fractal surpassed traditional models in forecasting infection, as indicated by the study of the influenza epidemic that showed more congruence with empirical data [25].

The advantages of each modelling technique, integer, fractional, and fractal, are distinct. The integer models give a simple and practical foundation, whereas fractional models increase accuracy by incorporating memory effects, and fractal models introduce consciousness to improve predictions [26].

Due to these complementary qualities, there is an increasing interest in developing better and more flexible epidemiological models by incorporating integer, fractional, and fractal components in hybrid modelling strategies.

In this study, we aim to compare the performance of three modelling approaches in forecasting dengue fever outbreaks in Gedaref, Sudan, by integrating actual epidemiological data with computational approaches to determine the most accurate model. Specifically, we explore how the role of memory effects in fractional models and spatial heterogeneity in fractal models influence disease transmission dynamics and outbreak generation. Furthermore, the study assesses the predictability and applicability of integer, fractional, and fractal models in enhancing disease control interventions.

This research has six chapters, starting with the **Introduction**, which discusses dengue fever and its public health impact in Gedaref, Sudan, which was chosen for the investigation. In the **Classical describe** chapter, integer-order differential equations have been utilized to describe disease transmission. Theoretical analysis analyzes the model's behaviour and stability to understand the disease's dynamics. The chapter on fractional and fractal models is called **Non-Integer Calculus**. In the **Discussion**, classical, fractional, and fractal models are compared to actual data. Finally, the **Conclusion** summarises key findings and emphasizes the importance of sophisticated modeling in predicting illnesses.

2. Classical model

Let N_1 and N_2 denote the total populations of humans and mosquitoes, respectively. Dengue, transmitted by *Aedes aegypti*, has four distinct serotypes (DENV-1 to DENV-4). Infection grants serotype-specific immunity, but reinfection with a different serotype increases the risk of severe complications due to Antibody-Dependent Enhancement (ADE). The human population is divided into: S_1 (susceptible), I_1 (infected and infectious), A_1 (partially immune), and R_1 (fully immune). For mosquitoes: S_2 (susceptible) and I_2 (infected and infectious).

We assume constant total populations for humans and mosquitoes, i.e.,

$$N_1 = S_1 + I_1 + A_1 + R_1, \quad N_2 = S_2 + I_2,$$

With natural births balancing natural deaths. The parameters of the model in Table (1) and Figure((1)). The model equations are described below, where β_1 represents the transmission rate at which an infected mosquito infects susceptible humans.

$$
\begin{aligned}
\frac{dS_1}{dt} &= \mu_1 N_1 - \beta_1 \frac{S_1 I_2}{N_1} - \mu_1 S_1, \\
\frac{dI_1}{dt} &= \frac{\beta_1 I_2}{N_1} S_1 + \delta_1 A_1 - (\mu_1 + \gamma) I_1, \\
\frac{dA_1}{dt} &= p\gamma I_1 - (\mu_1 + \delta_1) A_1, \\
\frac{dR_1}{dt} &= (1-p)\gamma I_1 - \mu_1 R_1. \\
\frac{dS_2}{dt} &= \mu_2 N_2 - \beta_2 \frac{S_2 I_1}{N_1} - \mu_2 S_2, \\
\frac{dI_2}{dt} &= \beta_2 \frac{S_2 I_1}{N_1} - \mu_2 I_2.
\end{aligned}
\tag{1}
$$

System (1) is naturally appended with initial conditions:

$S_1(0) = N_1 - I_1(0) - A_1(0) - R_1(0)$, $I_1(0) = 8$, $A_1(0) = 0$, $R_1(0) = 0$, $S_2(0) = N_2 - I_2(0)$, $I_2(0) = 10$.

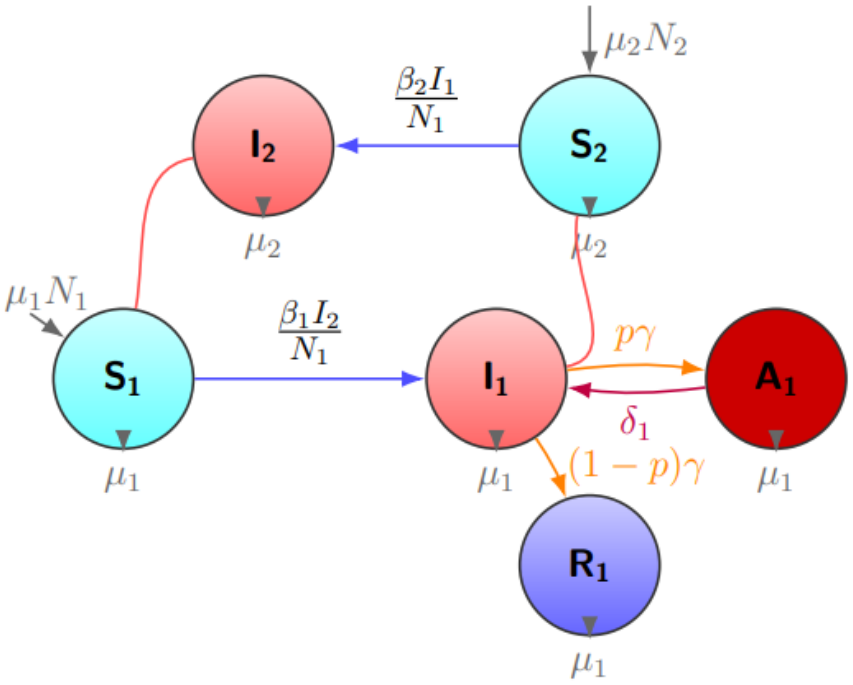


Figure 1: Enhanced schematic of Dengue transmission dynamics.

Table 1: Model parameters, values, and sources.

Parameter	**Interpretation**	**Value**	**Source**
μ_1	Birth/death rate (humans).	0.0000391	Fixed
μ_2	Birth/death rate (mosquitoes).	0.1	Fixed
β_1	Mosquito to human transmission rate.	0.1003	Fitted
β_2	Human to mosquito transmission rate.	0.1001	Fitted
γ	Human recovery rate.	0.2101	Fitted
p	Proportion of partial immunity.	0.8452	Fitted
δ_1	Reinfection rate.	0.0250	Fitted
N_1	Total humans.	2,208,385	Fixed
N_2	Total mosquitoes.	$3.5 \times N_1$	Assumed

Precise estimation of model parameters is crucial for adequately delineating dengue transmission patterns and forecasting capacities. This study applied the Markov Chain Monte Carlo (MCMC) technique [27, 28] to estimate parameter values derived from daily data about the dengue outbreak in Gedaref State, eastern Sudan, reported in 2023. The data were obtained from the reports of the Ministry of Health in Gedaref State, Sudan. The use of MCMC provides excellent accuracy in obtaining parameter values, enabling a more reliable assessment of dengue management efforts. Table 1 presents the estimated values.

3. Analysis of model

In this part, we verify that all state variables are nonnegative and bounded for all time. We also obtain the model equilibrium points and compute the basic reproduction number. Further, we explore bifurcation and stability properties.

3.1. Model non-negativity and boundedness

Proposition 1. *Let $S_{1,0}, I_{1,0}, A_{1,0}, R_{1,0}, S_{2,0}, I_{2,0}$ be non-negative initial conditions. Then the solution of* (1) *is non-negative for all $t \geq 0$.*

Proof. For $y \in \{S_1, I_1, A_1, R_1, S_2, I_2\}$, $\frac{dy}{dt}\Big|_{y=0} \geq 0$ ensures non-negativity.

Proposition 2. *The solution of* (1) *is bounded for all t.*

Proof. If N_1 and N_2 are bounded, all state variables remain bounded.

Thus, system (1) remains in the compact biologically feasible set:

$$\Omega = \left\{(S_1(t), I_1(t), A_1(t), R_1(t), S_2(t), I_2(t)) \subset \mathbb{R}^6_+ : N_1(t) + N_2(t) = N_{1,0} + N_{2,0}\right\}.$$

3.2. Model equilibria and basic reproduction number

As the total population $N_1(t) = S_1(t) + I_1(t) + A_1(t) + R_1(t)$ and $N_2(t) = S_2(t) + I_2(t)$ are constants, System (1) simplifies to:

$$\begin{aligned}
\frac{dS_1}{dt} &= \mu_1 N_1 - \frac{\beta_1}{N_1} S_1 I_2 - \mu_1 S_1, \\
\frac{dI_1}{dt} &= \frac{\beta_1}{N_1} S_1 I_2 - (\mu_1 + \gamma) I_1 + \delta_1 A_1, \\
\frac{dA_1}{dt} &= p\gamma I_1 - (\mu_1 + \delta_1) A_1, \\
\frac{dI_2}{dt} &= \frac{\beta_2}{N_1} (N_2 - I_2) I_1 - \mu_2 I_2.
\end{aligned} \tag{2}$$

This system has two equilibrium points: the disease-free equilibrium (DFE) $P_0 = (N_1, 0, 0, 0)$ and the endemic equilibrium (EE) $P^* = (S_1^*, I_1^*, A_1^*, S_2^*, I_2^*)$ with:

$$\begin{aligned}
S_1^* &= \frac{N_1 (N_1 N_2^{-1} \mu_1 \hat{\mathcal{R}}_0 + \beta_1)}{\hat{\mathcal{R}}_0 (N_1 N_2^{-1} \mu_1 + \beta_1)}, \\
I_1^* &= \left(\frac{\mu_1 N_1 (\mu_1 + \delta_1) \beta_1}{(\mu_1 + \gamma)(\mu_1 + \delta_1) - \delta_1 p \gamma} \right) \left(\frac{\hat{\mathcal{R}}_0 - 1}{\hat{\mathcal{R}}_0 (N_1 N_2^{-1} \mu_1 + \beta_1)} \right), \\
A_1^* &= \left(\frac{\mu_1 N_1 \beta_1 p \gamma}{(\mu_1 + \gamma)(\mu_1 + \delta_1) - \delta_1 p \gamma} \right) \left(\frac{\hat{\mathcal{R}}_0 - 1}{\hat{\mathcal{R}}_0 (N_1 N_2^{-1} \mu_1 + \beta_1)} \right), \\
I_2^* &= \frac{\mu_1 N_1 (\hat{\mathcal{R}}_0 - 1)}{N_1 N_2^{-1} \mu_1 \hat{\mathcal{R}}_0 + \beta_1}.
\end{aligned} \tag{3}$$

where $\hat{\mathcal{R}}_0$ is:

$$\hat{\mathcal{R}}_0 = \frac{\beta_1 \beta_2 N_2 (\mu_1 + \delta_1)}{N_1 \mu_2 [(\mu_1 + \gamma)(\mu_1 + \delta_1) - \delta_1 p \gamma]}. \tag{4}$$

We calculate the basic reproductive number, $\mathcal{R}_0$, using the next-generation matrix method. The Jacobian matrices are:

$$F = \begin{pmatrix} 0 & 0 & \beta_1 \\ 0 & 0 & 0 \\ \frac{\beta_2 N_2}{N_1} & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \mu_1 + \gamma & -\delta_1 & 0 \\ -p\gamma & \mu_1 + \delta_1 & 0 \\ 0 & 0 & \mu_2 \end{pmatrix}.$$

The basic reproduction number is the dominant eigenvalue of FV^{-1}:

$$\mathcal{R}_0 = \sqrt{\hat{\mathcal{R}}_0}. \tag{5}$$

3.3. Stability Analysis

Theorem 1. *(i) If $\mathcal{R}_0 < 1$, the disease-free equilibrium (DFE) of System (2) is locally asymptotically stable.*

(ii) If $\mathcal{R}_0 > 1$, the DFE is unstable.

Proof. The Jacobian matrix of System (2), denoted by J, is expressed as:

$$J = \begin{bmatrix} -\frac{\beta_1}{N_1} I_2 - \mu_1 & 0 & 0 & -\frac{\beta_1}{N_1} S_1 \\ \frac{\beta_1}{N_1} I_2 & -(\mu_1 + \gamma) & \delta_1 & \frac{\beta_1}{N_1} S_1 \\ 0 & p\gamma & -(\mu_1 + \delta_1) & 0 \\ 0 & \frac{\beta_2}{N_1}(N_2 - I_2) & 0 & -\frac{\beta_2}{N_1} I_1 - \mu_2 \end{bmatrix}.$$

At the disease-free equilibrium P_0, where $I_1 = I_2 = 0$, $S_1 = N_1$, and $S_2 = N_2$, the Jacobian matrix simplifies to:

$$J_{P_0} = \begin{bmatrix} -\mu_1 & 0 & 0 & -\beta_1 \\ 0 & -(\mu_1 + \gamma) & \delta_1 & \beta_1 \\ 0 & p\gamma & -(\mu_1 + \delta_1) & 0 \\ 0 & \frac{\beta_2 N_2}{N_1} & 0 & -\mu_2 \end{bmatrix}.$$

The eigenvalues of J_{P_0} are $\{\lambda_1, \lambda_2, \lambda_3, \lambda_4\}$, where:

$$\lambda_1 = -\mu_1, \quad \lambda_2 = -(\mu_1 + \gamma).$$

The remaining eigenvalues, λ_3 and λ_4, are determined by solving the following characteristic equations:

1. For λ_3, we have:

$$\lambda_3^2 + (\gamma + 2\mu_1 + \delta_1)\lambda_3 + \gamma\delta_1(1 - p) + \gamma\mu_1 + \mu_1(\delta_1 + \mu_1) = 0.$$

2. For λ_4, we solve:

$$\begin{aligned}\lambda_4^3 + (\delta_1 + \gamma + 2\mu_1 + \mu_2)\,\lambda_4^2 \\ + \Bigg[\mu_2(\delta_1+\mu_1) + (\delta_1+\mu_1)(\gamma+\mu_1) - p\gamma\delta_1 \\ + \mu_2(\mu_1+\gamma) - \frac{N_2\beta_1\beta_2}{N_1}\Bigg]\lambda_4 \\ + \frac{1}{\mu_2\left((\delta_1+\mu_1)(\gamma+\mu_1) - p\gamma\delta_1\right)}\left[1-\hat{\mathcal{R}}_0\right] = 0.\end{aligned}$$

To analyze the stability of the DFE, we examine the condition.$\mathcal{R}_0 < 1$. From the definition of $\mathcal{R}_0$, $\mathcal{R}_0 = \sqrt{\hat{\mathcal{R}}_0}$, where:

$$\hat{\mathcal{R}}_0 = \frac{\beta_1\beta_2 N_2(\mu_1+\delta_1)}{N_1\mu_2[(\mu_1+\gamma)(\mu_1+\delta_1) - \delta_1 p\gamma]}.$$

If $\mathcal{R}_0 < 1$, it follows that

$$\frac{\beta_1\beta_2 N_2}{N_1} < \mu_2(\mu_1+\gamma).$$

This ensures that all coefficients of the characteristic equations are positive, and using the Routh-Hurwitz criterion, all eigenvalues have negative real parts. Thus, the DFE is locally asymptotically stable when $\mathcal{R}_0 < 1$.

In contrast, if $\mathcal{R}_0 > 1$, according to Descartes' sign rule, the characteristic equation for λ_4 has at least one positive root. It causes the DFE to be unstable.

Theorem 2. *The disease-free equilibrium (DFE) is globally asymptotically stable when* $\mathcal{R}_0 < 1$.

Proof. To establish the global stability of the DFE, we analyze the subsystem corresponding to the disease-free state variables (S_1, A_1, R_1, S_2) and rewrite the full system (1) in the following form:

$$\begin{aligned}\frac{dX}{dt} &= P(X,Y), \\ \frac{dY}{dt} &= G(X,Y), \quad \text{with } G(X,0) = 0,\end{aligned} \tag{6}$$

where

$$X = (S_1, A_1, R_1, S_2) \in \mathbb{R}^4_+ \quad \text{and} \quad Y = (I_1, I_2) \in \mathbb{R}^2_+.$$

We use Castillo-Chavez's method to determine the DFE's global stability. (P_0). Specifically, we define:

$$\widehat{G}(X,Y) = A_3|_{P_0}Y - G(X,Y), \tag{7}$$

where $G(X,Y)$ is given by

$$G(X,Y) = \begin{bmatrix} \frac{\beta_1 S_1 I_2}{N_1} - (\mu_1 + \gamma) I_1 \\ \frac{\beta_2 S_2 I_1}{N_1} - \mu_2 I_2 \end{bmatrix}.$$

Here $A_3|_{P_0}$ is the Jacobian matrix $G(X,Y)$ evaluated at the DFE P_0. The Jacobian $A_3|_{P_0}$ is expressed as:

$$A_3|_{P_0} = \begin{bmatrix} -(\mu_1 + \gamma) & \beta_1 \\ \frac{\beta_2 N_2}{N_1} & -\mu_2 \end{bmatrix}.$$

Substituting $G(X,Y)$ and $A_3|_{P_0}$ into equation (7), we obtain:

$$\widehat{G}(X,Y) = \begin{bmatrix} \frac{\beta_1}{N_1} I_2 (N_1 - S_1) \\ \frac{\beta_2}{N_1} I_1 (N_2 - S_2) \end{bmatrix}.$$

Consequently, $\widehat{G}(X,Y) \geq 0, (X,Y) \in \Omega$. Therefore, P_0 is globally asymptotically stable when $\mathcal{R}_0 < 1$.

Theorem 3. *The endemic equilibrium point P^* of system* (1) *is globally asymptotically stable.*

Proof. First, we normalize state variables:

$$s_1 = \frac{S_1}{N_1}, \quad i_1 = \frac{I_1}{N_1}, \quad a_1 = \frac{A_1}{N_1}, \quad r_1 = \frac{R_1}{N_1},$$
$$s_2 = \frac{S_2}{N_2}, \quad i_2 = \frac{I_2}{N_2}.$$

Under this normalization, the total human and mosquito populations satisfy:

$$s_1 + i_1 + a_1 + r_1 = 1, \quad s_2 + i_2 = 1.$$

Furthermore, the endemic equilibrium P^* corresponds to the equilibrium point $(s_1^*, i_1^*, a_1^*, r_1^*, s_2^*, i_2^*)$. To prove global stability, we construct the following Lyapunov function:

$$V(s_1, i_1, a_1, r_1, s_2, i_2) = \frac{1}{2}\Big[(s_1 - s_1^*)^2 + (i_1 - i_1^*)^2 + (a_1 - a_1^*)^2 + (r_1 - r_1^*)^2 + (s_2 - s_2^*)^2 + (i_2 - i_2^*)^2\Big].$$

The derivative of V in (1) is:

$$\frac{dV}{dt} = \Big[(s_1 - s_1^*)\frac{ds_1}{dt} + (i_1 - i_1^*)\frac{di_1}{dt} + (a_1 - a_1^*)\frac{da_1}{dt} + (r_1 - r_1^*)\frac{dr_1}{dt} + (s_2 - s_2^*)\frac{ds_2}{dt} + (i_2 - i_2^*)\frac{di_2}{dt}\Big].$$

Since all normalized states are in $[0,1]$, we obtain

$$\begin{aligned}\frac{dV}{dt} &= \Big[(s_1 - s_1^*)\frac{ds_1}{dt} + (i_1 - i_1^*)\frac{di_1}{dt} + (a_1 - a_1^*)\frac{da_1}{dt} + (r_1 - r_1^*)\frac{dr_1}{dt} \\ &\quad + (s_2 - s_2^*)\frac{ds_2}{dt} + (i_2 - i_2^*)\frac{di_2}{dt}\Big] \\ &\leq \frac{d}{dt}\left(s_1 + i_1 + a_1 + r_1 + s_2 + i_2\right) = 0.\end{aligned}$$

Thus $\frac{dV}{dt} \leq 0$. Furthermore, it $\frac{dV}{dt} = 0$ occurs only at P^*. By LaSalle's Invariance Principle, all trajectories asymptotically approach. P^*. Thus, the endemic equilibrium is globally asymptotically stable.

4. Non-integer calculus

Research of differential operators of fractional orders dates back to 1695 when L'Hospital asked Leibniz the meaning of $\frac{d^u y}{dx^u}$ for $u = 1/2$ [29]. This led to the investigation of fractional calculus, and mathematicians like Euler, Lagrange, and Fourier took an interest in it [30].

The fractional calculus was later used in various fields such as electrical spectroscopy, wave propagation, quantum mechanics, fluid mechanics, and epidemiology [31–34]. Fractal derivatives and hybrid fractal-fractional models were also studied [35–37].

In this paper, the effects of noninteger-order derivatives on the dengue fever epidemic model are considered.

4.1. Fractional operators

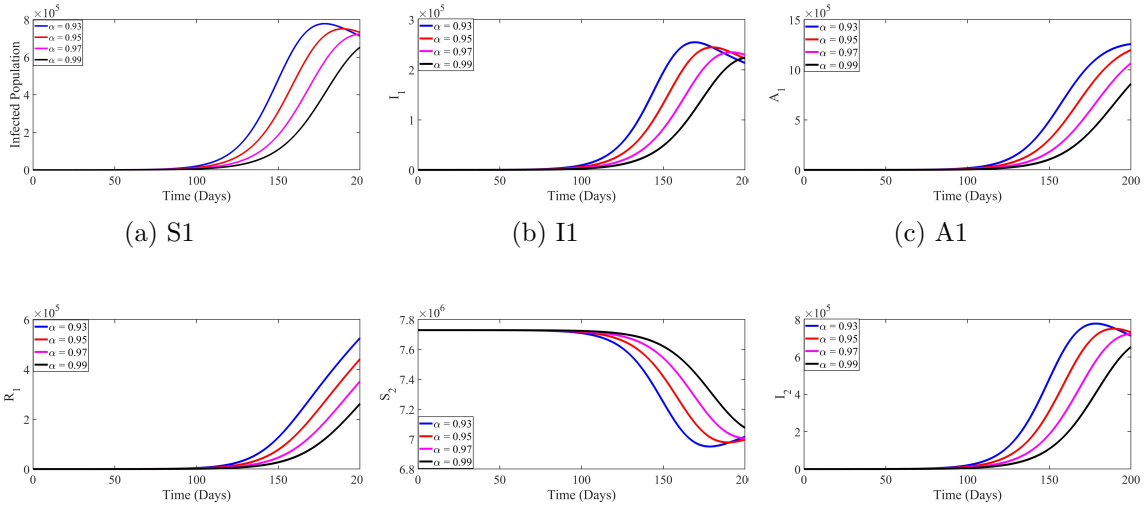
Here, we introduce fundamental concepts of fractional calculus [38–41], which will be used to develop our analysis. Due to the non-linearity of the epidemiological equations, obtaining analytical solutions is usually very difficult or, in most cases, impossible. To overcome this challenge,we present the system 1 under the Caputo operator:

$$\begin{aligned}{}^{C}D_t^{\alpha} S_1 &= \mu_1 N_1 - \beta_1 \frac{S_1 I_2}{N_1} - \mu_1 S_1, \\ {}^{C}D_t^{\alpha} I_1 &= \frac{\beta_1 I_2}{N_1} S_1 + \delta_1 A_1 - (\mu_1 + \gamma) I_1, \\ {}^{C}D_t^{\alpha} A_1 &= p\gamma I_1 - (\mu_1 + \delta_1) A_1, \\ {}^{C}D_t^{\alpha} R_1 &= (1-p)\gamma I_1 - \mu_1 R_1, \\ {}^{C}D_t^{\alpha} S_2 &= \mu_2 N_2 - \beta_2 \frac{S_2 I_1}{N_1} - \mu_2 S_2, \\ {}^{C}D_t^{\alpha} I_2 &= \beta_2 \frac{S_2 I_1}{N_1} - \mu_2 I_2.\end{aligned} \tag{8}$$

Now, we present a numerical method for solving fractional differential equations, referred to as the fractional Euler method. The method is based on the following formula:

$$y_{k+1} = y_0 + \frac{h^\alpha}{\Gamma(\alpha+1)} \sum_{j=0}^{k} \left[(k+1-j)^\alpha - (k-j)^\alpha\right] f(t_j, y_j), \tag{9}$$

When $\alpha = 1$, Equation 9 reduces to the Euler method for ODEs. This scheme is used for solving fractional differential equations with $h = 0.01$ in simulations. Convergence is ensured if $f(t, y)$ satisfies the Lipschitz condition, guaranteeing uniqueness and stability of the solution.



(a) S1 (b) I1 (c) A1

(d) R1 (e) S2 (f) I2

Figure 2: Fractional-order dynamics of the compartmental variables $S_1(t)$, $I_1(t)$, $A_1(t)$, $R_1(t)$, $S_2(t)$, and $I_2(t)$ under varying (α).

The figure illustrates the fractional-order dynamics of model () compartments under varying fractional-order parameters ($\alpha = 0.90, 0.91, 0.92, 0.94$). Fractional-order models effectively capture memory effects and the long-term dependence of disease progression, making them superior to classical integer-order models in representing dengue transmission dynamics.

The (S_1) progressively diminishes over time as a result of infection transmission. Lower values of α lead to an accelerated decline, indicating that fractional derivatives affect the rate of disease transmission by integrating memory effects.

The (I_1) demonstrates an exponential-like growth, where lower α values result in a greater peak and a more gradual decay. This behaviour illustrates the non-local effects and varied characteristics of dengue transmission, as fractional models consider changes in infection duration.

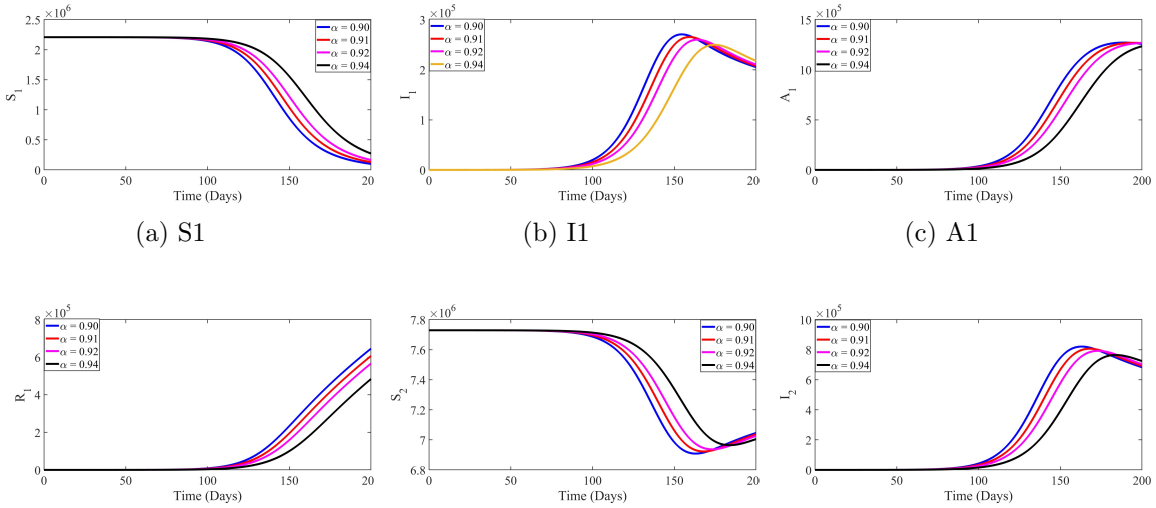
The (A_1) exhibits a comparable growth trajectory to I_1, albeit with diminished intensity. The varying α values affect the trajectory, suggesting that fractional-order models more effectively represent the delay in disease progression and concealed carriers.

4.2. Fractal operator

In this section, we present the concept of fractal derivatives and their significance in dengue fever spread modeling. Traditional differential operators fail to capture the complexities in disease dynamics in heterogeneous environments. Unlike fractional derivatives, which are typically defined in terms of convolution integrals, the fractal derivative is local, thus being computationally efficient and structurally distinct [42–44]. It is formulated in a non-Euclidean fractal metric space [45, 46], making it possible to describe the irregularity and self-similarity of real phenomena. The novelty of this mathematical strategy provides an improved description of dengue fever transmission, particularly in situations where spatial heterogeneity is of the utmost importance. The application of fractal derivatives to epidemiological modeling provides an improved understanding of the transmission of the disease and increases predictive capacity. We present system 1 with a fractal operator:

$$\begin{aligned}
\frac{dS_1}{dt} &= \alpha t^{\alpha-1}\mu_1 N_1 - \beta_1 \frac{S_1 I_2}{N_1} - \mu_1 S_1,\\
\frac{dI_1}{dt} &= \alpha t^{\alpha-1}\frac{\beta_1 I_2}{N_1} S_1 + \delta_1 A_1 - (\mu_1+\gamma) I_1,\\
\frac{dA_1}{dt} &= \alpha t^{\alpha-1} p\gamma I_1 - (\mu_1+\delta_1) A_1,\\
\frac{dR_1}{dt} &= \alpha t^{\alpha-1}(1-p)\gamma I_1 - \mu_1 R_1.\\
\frac{dS_2}{dt} &= \alpha t^{\alpha-1}\mu_2 N_2 - \beta_2 \frac{S_2 I_1}{N_1} - \mu_2 S_2,\\
\frac{dI_2}{dt} &= \alpha t^{\alpha-1}\beta_2 \frac{S_2 I_1}{N_1} - \mu_2 I_2.
\end{aligned} \tag{10}$$

Now, we apply the 4th-order Runge-Kutta method to solve the fractal system and perform simulations using RK4.



(a) S1 (b) I1 (c) A1

(d) R1 (e) S2 (f) I2

Figure 3: Fractal dynamics of the compartmental variables $S_1(t)$, $I_1(t)$, $A_1(t)$, $R_1(t)$, $S_2(t)$, and $I_2(t)$ under varying fractal dimensions (τ).

Figure 2 shows how the model compartments (system 1) changed over time when the fractal values changed ($\alpha = 0.90, 0.91, 0.92, 0.94$).

The human susceptible population (S_1) slowly decreases over time as more people become infected. The rate of decline changes with lower values of *alpha*, where lower values of fractal order accelerate the rate of decline. The asymmetric pattern shows the importance of fractal derivatives in modeling complex disease spread patterns in the real world.

The number of infected people (I_1) grows at an exponential rate over time, reaching a peak before starting to fall. The quantity I_1 increases exponentially over time, attaining a maximum before declining thereafter. Lower α values result in a higher peak and a slower decay, suggesting that fractal dynamics contribute to disease persistence by capturing delayed recovery and spatially heterogeneous transmission.

Over time, the asymptomatic human population (A_1) follows a growth trend similar to I_1 but at a lower intensity.

With time, the recovered population (R_1) gradually increases. The recovery trajectory depends on α, where lower values correspond to faster accumulation of immunity, likely due to shorter infectious periods and delayed waning immunity, as captured by fractal operators. Based on this behavior, it seems that fractal models are a better way to show immune responses, especially when there is partial immunity and infections keep happening.

The susceptible vector population (S_2) declines over time, similar to S_1. However, its trajectory changes, suggesting that reduced sensitivity or resistance to reinfection influences secondary exposure risks.

The infected vector population (I_2) exhibits a multiwave pattern, suggesting delayed

secondary infections or reinfections. The fractal-order operator modifies the transmission dynamics, incorporating erratic disease spread and persistent outbreaks. This means that fractal models are a better way to show the chances of getting dengue again and secondary waves of the disease, which are very important for understanding how long-term epidemics work.

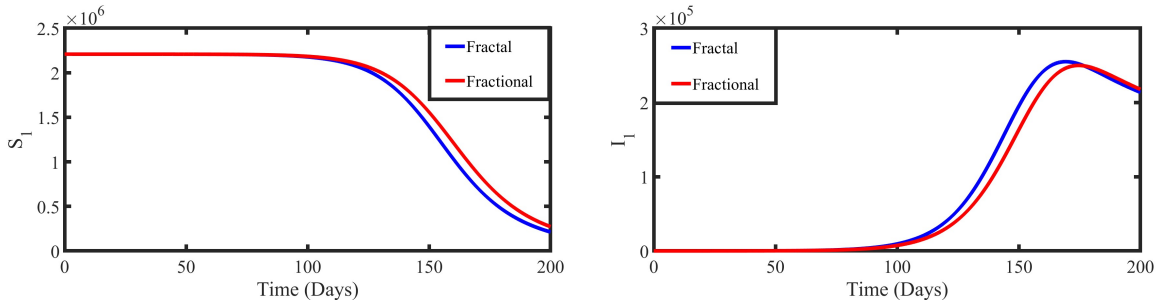
5. Discussion

In this section, we conduct a comparative analysis of three different dengue models: classical, fractal, and fractional models. The predictions obtained from these models are compared with accurate data for the 2023 dengue epidemic in Gedaref State, Eastern Sudan. The classical model is used as a baseline, whereas the fractal and fractional models offer more complex and general frameworks that take into account memory effects in the spread of epidemics.

Compared to the classical model, the fractal and fractional models generally exhibit superior predictive abilities, with the fractional model offering the best fit to actual data.

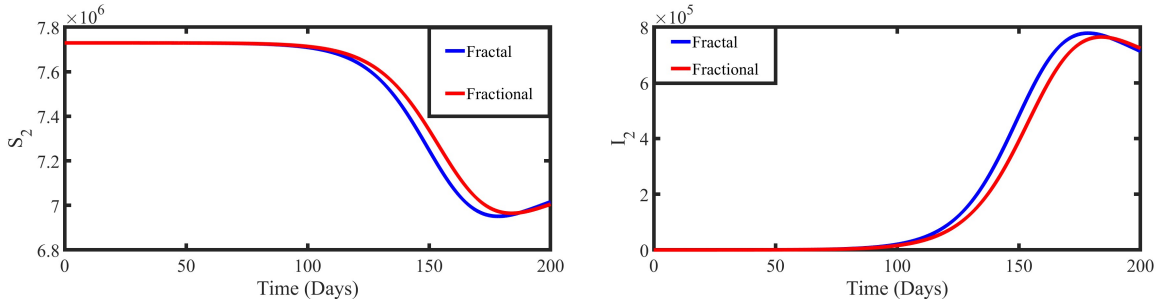
Figures 4, 5, and 6 provide more detailed insights into the evolution of the susceptible (S_1, S_2) and infected (I_1, I_2) populations (humans and mosquitoes) over a period of 200 days. Figure 4, The left panel (a) illustrates the decline in the susceptible human population (S_1), while the right panel (b) depicts the corresponding increase in the infected population (I_1). The results indicate that individuals become infected more rapidly in the fractional and fractal models compared to the classical model. After day 100, we observe a notable reduction in susceptibility, followed by a slow decline in infection due to recovery. Figure 5,This figure illustrates the dynamics of the susceptible (S_2) and infected (I_2) mosquito populations. The fractional and fractal models predict a more significant reduction in the susceptible mosquito population and a rapid increase in the infected population. This finding confirms the ability of these models to capture realistic disease progression patterns. Figure 6,This figure compares A_1 and R_1, which represent asymptomatic infections and recovered individuals. Both quantities increase over time, with the fractional model predicting a slightly faster growth rate.

The results show that the fractal and fractional models better show how complicated and nonlinear dengue transmission is. These models are better than the classical model because they give a more accurate picture of how the disease spreads between susceptible and infected groups and make predicting long-term epidemics easier.



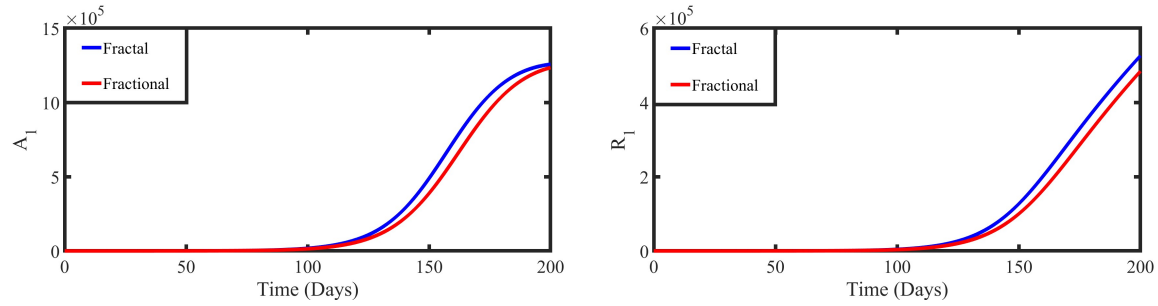
(a) S_1 (b) I_1

Figure 4: Comparison of fractal and fractional dynamics for $S_1(t)$ and $I_1(t)$ over time.



(a) S_2 (b) I_2

Figure 5: Comparison of fractal and fractional dynamics for S2(t) and I2(t) over time..



(a) A_1 (b) R_1

Figure 6: Comparison of fractal and fractional dynamics for $A_1(t)$ and $R_1(t)$ over time

Figure 7 illustrates the temporal evolution of the infected human population as predicted by the three models, using precise dengue data. The curves reveal marked discrepancies in each model's ability to accurately depict the epidemic's trajectory.

The fractional model (blue line) captures non-linear and varied patterns of disease

transmission. It closely matches the data in later epidemic stages, indicating that fractional derivatives effectively capture long-memory effects and fluctuations in disease transmission. The fractal model (red line) captures disease dynamics moderately and lies between the classical and fractional models. By incorporating memory-dependent transmission effects, this model leads to smoother and more gradual disease progression compared to the classical model. The classical model (black line) significantly underestimates the actual cases and exhibits slower infection progression. The results indicate that this model fails to capture the long-term dynamics of the epidemic, especially when secondary waves and delayed transmission effects are considered.

The comparative study emphasizes the inadequacy of the classical model in the correct forecasting of dengue fever outbreaks since it does not take into account the memory effects, spatial heterogeneity, and time-delay transmission dynamics. However, the fractal and fractional models accurately outline the disease transmission process, the best among them being the fractional model, to actual epidemiological data. With the inclusion of long-memory effects and nonlinear transmission patterns, predictive power is introduced by the fractional model, and thus it can be a useful tool for epidemic prediction as well as public health intervention planning. The fractal model also introduces robustness to disease modeling by the inclusion of spatial heterogeneity, which is applicable in describing heterogeneous transmission patterns. Such sophisticated models provide more flexibility and reliability for improved epidemic control interventions and policy planning effectiveness in provinces such as Gedaref.

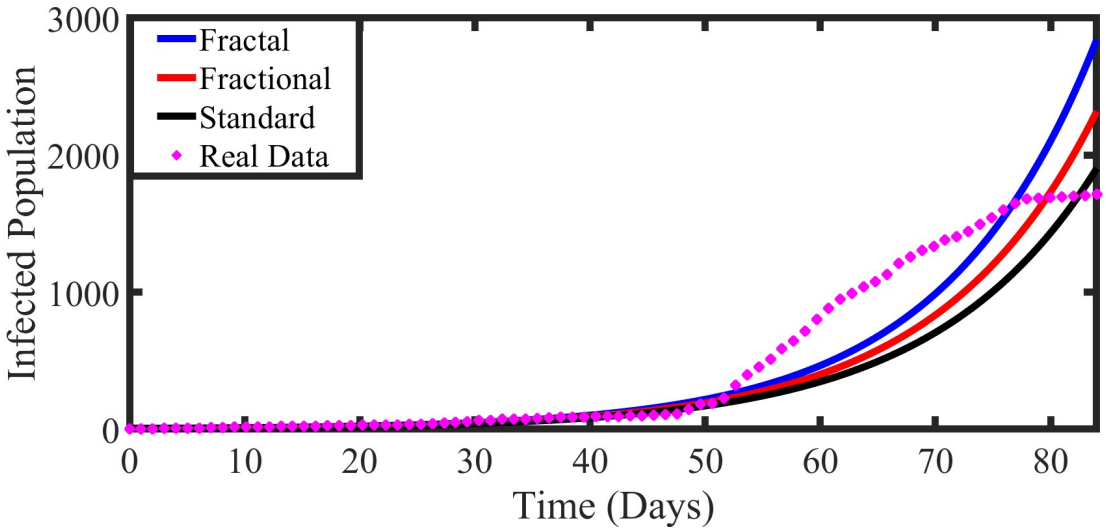


Figure 7: Comparison between fractal, fractional, and classical models against actual data for $I_1(t)$ over time.

6. Conclusion

In this article, we have modeled the spread of dengue by three different SIAR-SI epidemic models based on classical, fractional (Caputo), and fractal (Hausdorff) derivatives. To solve these models numerically, we employed the classical Euler method to solve the standard model, the fractional Euler method to solve the fractional model, and the fourth-order Runge-Kutta method to solve the fractal model. We also examined the stability of both the disease-free and endemic equilibrium states, which reveals the conditions under which the disease will become extinct or persistent. The model was calibrated using real data from Gedaref, Sudan, and parameters were estimated using the Markov Chain Monte Carlo method. The basic reproduction number for 2023 was estimated as $mathcal R_0 = 3.27$, confirming the endemicity of the disease. Among the three models, the classical model best fits real data. The fractional model, in the form of Caputo derivatives, could include memory effects and hence give a more realistic representation of disease spread. The fractal model, in the form of Hausdorff derivatives, offered a new insight into complex transmission dynamics. For further research, some directions of extension are available. The extension of the fractal model into time-evolving parameters will make it even more realistic in its ability to cope with real situations. The integration of stochastic influence into fractional and fractal models will provide a better representation of the diffusion of uncertainty-causing diseases. In future work, we aim to solve new fractional models [47–49] and compare with other numerical methods [50–56].

Conflicts of Interest: The authors declare that they have no conflict of interest.

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