A peculiar application of the fractal–fractional derivative in the dynamics of a nonlinear scabies model

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ABSTRACT

In this paper, we provide a generic mathematical framework for scabies transmission mechanisms. The infections involving susceptible, highly contagious people and juvenile scabiei mites are characterized by a framework of ordinary differential equations (DEs). The objective of this study is to examine the evolution of scabies disease employing a revolutionary configuration termed a fractal–fractional (FF) Atangana–Baleanu (AB) operator. Generic dynamical estimates are used to simulate the underlying pace of growth of vulnerable people, clinical outcomes, and also the eradication and propagation rates of contaminated people and immature mites. We study and comprehend our system, focusing on a variety of restrictions on its basic functionalities. The model's outcomes are assessed for positivity and boundedness. The formula includes a fundamental reproducing factor, $\mathcal{R}_0$, that ensures the presence and stability of all relevant states. Furthermore, the FF-AB operator is employed in the scabies model, and its mathematical formulation is presented using a novel process. We analyze the FF framework to construct various fractal and fractional levels and conclude that the FF theory predicts the affected occurrences of scabies illness adequately. The relevance and usefulness of the recently described operator has been demonstrated through simulations of various patterns of fractal and fractional data.

Introduction

Scabiei is an extremely contagious virus engendered by the cryptosporidium worm Schistosoma scabiei, which belongs to the group of dermatitis. Seborrheic dermatitis is detected in roughly 300 million people nationwide each year, making it a major environmental disease issue [1–3]. In reality, disease is frequently disseminated in impoverished settings with overcrowding. Pervasiveness in mainland Australia [4,5], for example, can reach 49 percent, compared to 28 percent and 43 percent in Vanuatu and the Solomon Islands, respectively. According to the Saudi Department of Health, over 1700 contaminated people were identified as having mites in Makkah, Saudi Arabia's northwest area, during the first half of 2018 [6]. Ahmed et al. [7] calculated the prevalence of mite relapse amongst Saudi Arabian individuals in 2019. Intimate epidermis interaction is the most conventional approach to mite dissemination in humans. Additional modes of infection are through the exchanging of objects or the use of contaminated domestic possessions like bedding, apparel, and blankets. Furthermore, genital interaction is a primary route for the overgrowth to propagate [8]. Fuller [9] stated that the downstream complications of scabies infestations, such as pyoderma, streptococcal glomerulonephritis, and subsequent chronic renal impairment and rheumatic fever, affect the epidemiological situation for scabies around the world. In 1977, Mellanby [10,11] established a series of experiments with young male volunteers who had agreed to be exposed to scabies infection. Very recently, author [12] discussed the mechanistic and statistical models of skin disease transmission. Izri and Chosidow [13] determined the efficacy of machine laundering to eradicate head lice. Engelman and Steer [14] contemplated innovative strategies, utilizing ivermectin-based mass drug administration, that appear feasible and highly effective. In practice, there is indeed a ten-week delay between the

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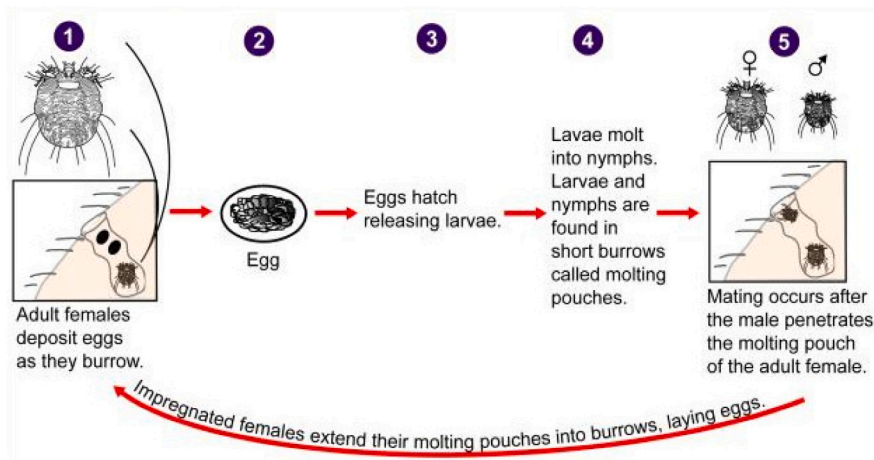
Fig. 1. Reproductive process of scabies mite.

Fig. 2. Reproductive process of scabies mite.

time of its initial contamination and the onset of indications in the affected population. Affected patients experiencing mites can transmit the infection across the neighborhood, even within the undiagnosed phase [4,14].

Scabies mites go through three developmental periods throughout their lives: embryo, juvenile mite, and maturity [3,4]. The female scabiei var hominis mite lays 60–90 eggs in her 30-day lifespan, although less than 10 percent of the eggs result in mature mites. The average patient is infected with 10–15 live adult female mites at any given time [4,15]. Life cycle stages are as follows:

1. Larvae migrate to the skin surface and burrow into the intact stratum corneum to make short burrows, called molting pouches [16–18].

2. Larvae molt into nymphs, which molt once into larger nymphs before becoming adults [16,18].

3. Mating takes place once, and the female is fertile for the rest of her life; the male dies soon after mating [16,17].

4. The female makes a serpentine burrow using proteolytic enzymes to dissolve the stratum corneum of the epidermis, laying eggs in the process; she continues to lengthen her burrow and lay eggs for the rest of her life, surviving 1–2 months [19].

5. Transmission of impregnated females from person-to-person occurs through direct or indirect skin contact. In around two days, immature nymphs evolve into presumably male or female grownups, see (Fig. 1).

Mature fleas devote approximately two fortnights to hunting for a companion. The expectant infestations proceed to produce offspring during genital interaction, extending the colonization phase. Male mites, in particular, disappear eventually after coupling. Female mites can live for months longer than adult mealybugs [3]. The subsequent population of adulthood scabies arises roughly a month after the original outbreak and resumes the invasive species process [4,12,19], see (Fig. 2). In addition to grasping the intricacies of epidemiological models, scholars have developed and constructed numerical simulations for a variety of objectives [20–26]. Researchers have considered significant studies on epidemics based on clarity purposes [27,28]. The researchers of [29], for example, investigated the periodicity of vector-borne clinical studies. Linden et al. [30] take into account infectious transmission and superdiffusion. On the other hand, the estimates reported in [31] disregarded characteristics of the Sarcoptes scabiei's entire lifespan and effectively as an underlying true connection involving larvae and susceptible people. Lydeamore et al. [4] developed a numerical framework to depict the mechanisms of mite infestation, taking into account the mite's entire process and interactions with recipients, and also several modalities of medication and vulnerability to bacterial invasion. The understanding of impaired acquisition strategies is not presented in these representations, which is a restriction.

Furthermore, when employing systems predicated on integer derivatives, it is hard to analyze the interactions involving successive points. Multiple sorts of fractional order derivative frameworks have been

Table 1
Table of specified variables and their descriptions.

Parameters	Explanation	Data estimated [30]
$\mathbf{S(t)}$	Amount of susceptible individuals in time $\mathbf{t}$	
$\mathbf{I_1(t)}$	Infectious people that having living mites and eggs after infection	
$\mathbf{I_2(t)}$	Infectious people that having living mites and eggs after developing symptoms	
$\mathbf{I_3(t)}$	Infected individuals who have only young mites	
$\mathbf{M(t)}$	Number of adults mites at time $\mathbf{t}$	
σ_1	Infection rate for $\mathbf{I_1}$	0.002
σ_2	Infection rate for $\mathbf{I_2}$	0.003
σ_3	Infection rate for $\mathbf{I_3}$	0.005
σ_4	Infection rate for $\mathbf{M}$	0.007
κ	Birth rate of susceptible persons	200
η	Transmission rate of developing symptoms	0.4
$\wp$	Transmission rate of egg hatching	0.4
ϵ	Natural death rate	0.5
ς	Transmission rate for mites mature and become adults	0.4
θ	Natural death rate of the adult mites	0.3

reported in the research to overcome such challenges, see [32–40] and the sources included. Researchers demonstrate significant contemporary scholarly literature on fractional operators in association with a variety of challenges in the scientific domain [41,42]. For example, Rashid et al. [43] examined the analytical solutions of Fisher's equation via the Caputo fractional derivative operator. Qudah et al. [44] expounded the fuzzy solutions of the Cauchy reaction diffusion equation by employing the Atangana–Baleanu fractional derivative operator. Gómez-Aguilar [45] proposes using FF operators to solve Shinriki's oscillator framework. Researcher [46] explores the fractional derivative evaluation of the Vallis system. Solís-Pérez [47] considers the mechanics of vasculature recognition. The physical and mechanical challenges in fractional DEs are adequately achieved in the information supplied earlier. Aside from that, the FF calculus [48] is a novel domain of investigation that has lately been established and has successfully produced scientific and engineering knowledge. Several contemporary fractional derivative numerical simulations were studied in [49,50]. The following are the primary accomplishments of this manuscript: (1) A mite epidemic approach is performed via the FF operator having a Mittag-Leffler kernel. The framework illustrates the connections involving vulnerable, highly contagious people and mature mites in a population. With the aid of the FF approach, we developed a scabies system and tested its characteristics using realistic transmission occurrences. We evaluate FF pattering to factual information from scabies-infected individuals and find many remarkable outcomes. Furthermore, we discussed the research on scabies infection in general, as well as fractional operator approaches. (2) The system's wellposedness is demonstrated by exhibiting the outcome's positivity and boundedness. (3) The presence and durability of disease-free and prevalent equilibrium are determined by the fundamental reproduction rate. (4) Numerical methods are used to highlight the simulated predictions in the system in the FF sense. (5) The infected equilibria are conducted for a robustness evaluation. Finally, the study is summarized, for more information (see, Table 1).

Scabies model transmission

In this part, a five-dimensional framework constructed by AlShamrani et al. [51] to illustrate the behavior of a scabies infestation in disease transmission:

$$\begin{cases} \frac{d\mathbf{S(t)}}{d\mathbf{t}} = \Phi - \epsilon\mathbf{S(t)} - \left(\sigma_1\mathbf{I_1(t)} + \sigma_2\mathbf{I_2(t)} + \sigma_3\mathbf{I_3(t)} + \sigma_4\mathbf{M(t)}\right)\mathbf{S}^q\mathbf{(t)}, \\ \frac{d\mathbf{I_1(t)}}{d\mathbf{t}} = \left(\sigma_1\mathbf{I_1(t)} + \sigma_2\mathbf{I_2(t)} + \sigma_3\mathbf{I_3(t)} + \sigma_4\mathbf{M(t)}\right)\mathbf{S}^q\mathbf{(t)} - (\eta + \epsilon)\mathbf{I_1(t)}, \\ \frac{d\mathbf{I_2(t)}}{d\mathbf{t}} = \eta\mathbf{I_1(t)} - (\wp + \epsilon)\mathbf{I_2(t)}, \\ \frac{d\mathbf{I_3(t)}}{d\mathbf{t}} = \wp\mathbf{I_2(t)} - (\varsigma + \epsilon)\mathbf{I_3(t)}, \\ \frac{d\mathbf{M(t)}}{d\mathbf{t}} = \varsigma\mathbf{I_3(t)} - \theta\mathbf{M(t)}. \end{cases} \tag{1}$$

subject to ICs

$$\mathbf{S}(0) = \mathbf{S_0} \geq 0, \ \mathbf{I_1}(0) = \mathbf{I_{1_0}} \geq 0, \ \mathbf{I_2}(0) = \mathbf{I_{2_0}} \geq 0,$$

$$\mathbf{I_3}(0) = \mathbf{I_{3_0}} \geq 0, \mathbf{M}(0) = \mathbf{M_0} \geq 0. \tag{2}$$

At time $\mathbf{t}$, the group can be classified into five groups: highly vulnerable people $\mathbf{S(t)}$, extremely contagious persons with staying ectoparasites and larvae during the apparently healthy timeframe after virus $\mathbf{I_1(t)}$, contagious persons with having to live ectoparasites and larvae after getting an infection $\mathbf{I_2(t)}$, and contaminated people with only adolescent ectoparasites (i.e. larvae have been fertilized). The infectious disease prevalence constant is referred to as the component $\mathbf{I_3(t)}$. Furthermore, Φ indicates the birth rate constant of highly vulnerable people, η signifies the transmission raye constantly producing indications and $\wp$ refers to the transmission rate constantly for egg hatching, respectively. Additionally, regardless of poor prognosis, ϵ has a spontaneous mortality rate that remains stable. $\mathbf{M(t)}$ represent the proportion of mature mites present at time $\mathbf{t}$. The dissemination speed constant for mites that progress by becoming mature and the spontaneous rate of death constant for mature fleas, respectively, are parameters ς and θ. Furthermore, we refer the researcher to the comprehensive research review article [30] for further knowledge on scabies outbreak simulation.

Framework (14) assumes a conventional bilinear transmission (incidence) rate that is predicated on the idea of collective protest in terms of the amount of vulnerable and pathogenic organisms persons [52,53]. Therefore, this model does not well reflect the heterogeneity interaction of people in a community. Additionally, as the amount of highly contagious persons grows, the dissemination speed may increase in rebuttal than linearly [54,55]. Numerous researchers have subsequently included nonlinear parameterization in their modeling techniques [56], where $q > 0$ are constants.

Mechanisms of the scabies model

To demonstrate that (14) is outbreaks significant, we must explain that the scheme's accompanying model parameters are non-negative for every time $\mathbf{t}$. This is more conveniently addressed by the observation that the scabies system of DEs (14) having non-negative ICs becomes non-negative for all $\mathbf{t} > 0$. Here, we present the lemma as follows:

Lemma 1. *Assume that there be initial data $\mathcal{G}(0) \geq 0$, where*

$$\mathcal{G}(\mathbf{t}) = (\mathbf{S(t)}, \mathbf{I_1(t)}, \mathbf{I_2(t)}, \mathbf{I_3(t)}, \mathbf{M(t)}). \tag{3}$$

Thus the model (14) represents the non-negative solution for every time $\mathbf{t} > 0$. Also, $\lim_{\mathbf{t} \to \infty} \mathcal{N}(\mathbf{t}) \leq \frac{\Phi}{\epsilon}$ having $\mathcal{N}(\mathbf{t}) = \mathbf{S(t)} + \mathbf{I_1(t)} + \mathbf{I_2(t)} + \mathbf{I_3(t)} + \mathbf{M(t)}$.

Proof. Let $\nu = \sup\{\mathbf{t} > 0 : \mathcal{G}(\mathbf{t}) > 0 \in [0, \mathbf{t}]\}$. Therefore, $\nu > 0$. For the first component of the problem (14), we acquire the significant findings

as follows

$$\frac{d\mathbf{S}(\mathbf{t})}{d\mathbf{t}} = \Phi - \epsilon\mathbf{S}(\mathbf{t}) - \big(\sigma_1\mathbf{I}_1(\mathbf{t}) + \sigma_2\mathbf{I}_2(\mathbf{t}) + \sigma_3\mathbf{I}_3(\mathbf{t}) + \sigma_4\mathbf{M}(\mathbf{t})\big)\mathbf{S}^q(\mathbf{t}). \qquad (4)$$

Taking $\lambda = \big(\sigma_1\mathbf{I}_1(\mathbf{t}) + \sigma_2\mathbf{I}_2(\mathbf{t}) + \sigma_3\mathbf{I}_3(\mathbf{t}) + \sigma_4\mathbf{M}(\mathbf{t})\big) = \sum_{t=1}^{3}\sigma_t\mathbf{I}_t$, then (4) reduces to

$$\frac{d\mathbf{S}(\mathbf{t})}{d\mathbf{t}} = \Phi - \epsilon\mathbf{S}(\mathbf{t}) - \lambda\mathbf{S}^q(\mathbf{t}). \qquad (5)$$

(17) can be demonstrated in more context as shown below

$$\frac{d}{d\mathbf{t}}\left(\mathbf{S}(\mathbf{t})\exp\left(\epsilon\mathbf{t} + \int_0^\nu \lambda(\phi)d\phi\right)\right) = \Phi\exp\left(\epsilon\mathbf{t} + \int_0^\nu \lambda(\phi)d\phi\right). \qquad (6)$$

Therefore, we have

$$\mathbf{S}(\mathbf{t})\exp\left(\epsilon\nu + \int_0^\nu \lambda(\phi)d\phi\right) - \mathbf{S}(0) = \Phi\exp\left(\epsilon y_1 + \int_0^\nu \lambda(\wp)d\wp\right)dy_1. \qquad (7)$$

It follows that

$$\mathbf{S}(\mathbf{t}) = \mathbf{S}(0)\exp\left(-\left(\epsilon\nu + \int_0^\nu \lambda(\phi)d\phi\right)\right) + \exp\left(-\left(\epsilon\nu + \int_0^\nu \lambda(\phi)d\phi\right)\right)$$
$$\times \int_0^\nu \Phi\exp\left(\epsilon y_1 + \int_0^\nu \lambda(\wp)d\wp\right)dy_1 > 0. \qquad (8)$$

Therefore, we can obtain $\mathcal{G}(\mathbf{t}) > 0$ for any $\mathbf{t} > 0$ by repeating the previous methods for the leftover components of framework (14). Thus, we conclude that $0 < \mathbf{S}(0) \leq \mathcal{N}(\mathbf{t})$, $0 < \mathbf{I}_1(0) \leq \mathcal{N}(\mathbf{t})$, $0 < \mathbf{I}_2(0) \leq \mathcal{N}(\mathbf{t})$, $0 < \mathbf{I}_3(0) \leq \mathcal{N}(\mathbf{t})$, $0 < \mathbf{M}(0) \leq \mathcal{N}(\mathbf{t})$. After adding the all formulations, lead to

$$\frac{d\mathcal{N}}{d\mathbf{t}} = \Phi - \epsilon\mathcal{N}. \qquad (9)$$

Consequently, we have

$$\lim_{\mathbf{t}\mapsto\infty} \mathcal{N}(\mathbf{t}) \leq \frac{\Phi}{\epsilon}, \qquad (10)$$

which gives the immediate consequence. □

Further, we illustrate the invariant domains for the specified scabies model (14). For this, we propose the feasible domain as follows:

$$\Lambda = \left\{ (\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}) \in \mathbf{R}_+^5 : \mathcal{N}(\mathbf{t}) \leq \frac{\Phi}{\epsilon} \right\}.$$

For the plausibility domain, the accompanying outcomes are reported.

Lemma 2. *The region presented by Λ is positively invariant for the scabies model* (14) *incorporating the non-negative ICs.*

Proof. By means of (9), then the scabies model (14) leads to

$$\frac{d\mathcal{N}}{d\mathbf{t}} = \Phi - \epsilon\mathcal{N},$$

which concludes that $\frac{d\mathcal{N}}{dt} \leq 0$, if $\mathcal{N}(0) \geq \frac{\Phi}{\epsilon}$. Thus $\mathcal{N}(\mathbf{t}) \leq \mathcal{N}(0)\exp(-\epsilon\mathbf{t}) + \frac{\Phi}{\epsilon}(1-\exp(-\epsilon\mathbf{t}))$. Therefore, the domain followed by Λ is positively invariant. Moreover, if $\mathcal{N}(0) > \frac{\Phi}{\epsilon}$, then either the outcomes belongs to Λ in finite time, or we can say that $\mathcal{N}(\mathbf{t})$ approaches to $\frac{\Phi}{\epsilon}$ asymptotically. As a result, all of the responses in $\mathbb{R}_+^5$ are drawn to the boundaries defined by Λ. □

Basic concepts of fractal–fractional calculus

Here, the outcomes of FF calculus, structure analysis in the FF operator of the AB derivative perspective, and system (14) robustness are discussed in this part. Considering the findings in [30], we commence with the fundamentals of FF calculus considering the generalized Mittag-Leffler (GML) kernel.

Definition 1 (*[48]*). Suppose there be a continuous mapping $g_1(\mathbf{t})$ which is fractal differentiable in (a_1, a_2) having order ω, then the FF

derivative of $g_1(\mathbf{t})$ have order α described in the Riemann–Liouville perspective considering GML kernel is presented as follows:

$$^{FFM}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(g_1(\mathbf{t})) = \frac{\mathbf{ABC}(\alpha)}{1-\alpha}\frac{d}{d\mathbf{t}^\omega}\int_0^{\mathbf{t}} E_\alpha\left(-\frac{\alpha}{1-\alpha}(\mathbf{t}-\varrho)^\alpha\right)g_1(\varrho)d\varrho, \qquad (11)$$

where $\alpha > 0$, $\omega \leq 1 \in \mathbb{N}$ and $\mathbf{ABC}(\alpha) = 1 - \alpha + \frac{\alpha}{\Gamma(\alpha)}$.

Definition 2 (*[48]*). Suppose there be a continuous mapping $g_1(\mathbf{t})$ on (a_1, a_2), then the FF integral of $g_1(\mathbf{t})$ having order α with the GML kernel is presented as

$$^{FFM}\mathbf{J}_{0,\mathbf{t}}^{\alpha,\omega}(g_1(\mathbf{t})) = \frac{\alpha\omega}{\mathbf{ABC}(\alpha)}\int_0^{\mathbf{t}} \varrho^{\omega-1}(\mathbf{t}-\varrho)^{\alpha-1}g_1(\varrho)d\varrho + \frac{\omega(1-\alpha)}{\mathbf{ABC}(\alpha)}\mathbf{t}^{\omega-1}g_1(\mathbf{t}). \qquad (12)$$

Fractal–fractional scabies model

In this part, we implement the FF operator on the structure (14) in the AB perspective as follows:

$$\begin{cases} ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{S}(\mathbf{t})) = \Phi - \epsilon\mathbf{S}(\mathbf{t}) - \big(\sigma_1\mathbf{I}_1(\mathbf{t}) + \sigma_2\mathbf{I}_2(\mathbf{t}) + \sigma_3\mathbf{I}_3(\mathbf{t}) \\ \quad + \sigma_4\mathbf{M}(\mathbf{t})\big)\mathbf{S}^q(\mathbf{t}), \quad \mathbf{S}(0) = \mathbf{S}_0 \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_1(\mathbf{t})) = \big(\sigma_1\mathbf{I}_1(\mathbf{t}) + \sigma_2\mathbf{I}_2(\mathbf{t}) + \sigma_3\mathbf{I}_3(\mathbf{t}) \\ \quad + \sigma_4\mathbf{M}(\mathbf{t})\big)\mathbf{S}^q(\mathbf{t}) - (\eta + \epsilon)\mathbf{I}_1(\mathbf{t}), \quad \mathbf{I}_1(0) = \mathbf{I}_{10} \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_2(\mathbf{t})) = \eta\mathbf{I}_1(\mathbf{t}) - (\wp + \epsilon)\mathbf{I}_2(\mathbf{t}), \quad \mathbf{I}_2(0) = \mathbf{I}_{20} \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_3(\mathbf{t})) = \wp\mathbf{I}_2(\mathbf{t}) - (\varsigma + \epsilon)\mathbf{I}_3(\mathbf{t}), \quad \mathbf{I}_3(0) = \mathbf{I}_{30} \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{M}(\mathbf{t})) = \varsigma\mathbf{I}_3(\mathbf{t}) - \theta\mathbf{M}(\mathbf{t}), \quad \mathbf{M}(0) = \mathbf{M}_0 \geq 0, \end{cases} \qquad (13)$$

where α and ω denote the fractional and fractal order, respectively.

Existence and positivity

Next, we investigate the existence and positivity of the scabies model (14). For further investigation, we refer the readers [21,48,49].

Theorem 1. *Suppose there be a unique positive outcome for the model* (14) *that remains in $\mathbb{R}_+^5$.*

Proof. To reveal the preceding outcome to demonstrate that the response of the framework (14) is positive:

$$\begin{cases} ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{S}(\mathbf{t}))\big|_{\mathbf{S}=0} = \Phi \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_1(\mathbf{t}))\big|_{\mathbf{I}_1=0} = 0 \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_2(\mathbf{t}))\big|_{\mathbf{I}_2=0} = \eta\mathbf{I}_1(\mathbf{t}) \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_3(\mathbf{t}))\big|_{\mathbf{I}_2=0} = \wp\mathbf{I}_2(\mathbf{t}) \geq 0, \\ ^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{M}(\mathbf{t}))\big|_{\mathbf{M}=0} = \varsigma\mathbf{I}_3(\mathbf{t}) \geq 0. \end{cases} \qquad (14)$$

This demonstrates that the system result will persist in $\mathbb{R}_+^5$ for all time $\mathbf{t} \geq 0$. Moreover, summing all the components in (14), we have

$$^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathcal{N}(\mathbf{t})) = \Phi - \epsilon\mathcal{N}. \qquad (15)$$

Observe that

$$\limsup_{\mathbf{t}\to\infty} \mathcal{N}(\mathbf{t}) \leq \frac{\Phi}{\epsilon}. \qquad (16)$$

Consequently, the biological viable domain for the system (14) can be described as

$$\Lambda^1 = \left\{ (\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}) \in \mathbf{R}_+^5 : \mathcal{N}(\mathbf{t}) \leq \frac{\Phi}{\epsilon} \right\}.$$

The framework for the scabies system aforementioned (14) in the FF-AB operator is implemented to derive the relevant findings in the subsequent part. □

Stability analysis disease free case

Furthermore, we now illustrate the stability criteria for disease-free cases.

This section investigates the stability findings for the scabies models proposed by the disease-free equilibrium (DFE) $\mathcal{E}_0$. We generate the subsequent formulation by changing the right hand side of the scabies model (14) equal to zero. Then, we have

$$
\begin{cases}
{}^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{S}(\mathbf{t})) = 0, \\
{}^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_1(\mathbf{t})) = 0, \\
{}^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_2(\mathbf{t})) = 0, \\
{}^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{I}_3(\mathbf{t})) = 0, \\
{}^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}(\mathbf{M}(\mathbf{t})) = 0.
\end{cases}
\tag{17}
$$

This yields that

$$
\mathcal{E}_0 = (\mathbf{S}_0, 0, 0, 0, 0) = \left(\frac{\boldsymbol{\Phi}}{\epsilon}, 0, 0, 0, 0\right).
\tag{18}
$$

The fundamental reproducing value $\mathcal{R}_0$, which can be generated using the next generation approach for the scheme (14), can be utilized to examine the robustness of DFE at $\mathcal{E}_0$. Assuming that the contaminated components in the scabies system (14) are $\mathbf{I}_1$, $\mathbf{I}_2$, $\mathbf{I}_3$, $\mathbf{M}$ and that you implement the procedure in [57], the matrices $\mathcal{F}$ and $\mathcal{V}$ are as follows:

$$
\mathcal{F} = \begin{bmatrix} 0 & 0 & \sigma_1 + \sigma_2 + \sigma_3 + \sigma_4 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{bmatrix}
\tag{19}
$$

and

$$
\mathcal{V} = \begin{bmatrix} (\eta + \epsilon) & 0 & 0 \\ -\eta & (\wp + \epsilon) & 0 \\ 0 & \wp & (\varsigma + \epsilon) \end{bmatrix}
\tag{20}
$$

The model's (14) requisite fundamental reproducing factor is derived by the spectral radius of the matrix $\mathcal{R}_0 = \rho(\mathcal{F}\mathcal{V}^{-1})$, provided by

$$
\mathcal{R}_0 = \frac{(\sigma_1(\varsigma + \epsilon)(\epsilon + \wp) + \sigma_2\eta(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_4\eta\varsigma\wp)\mathbf{S}_0^q}{(\eta + \epsilon)(\varsigma + \epsilon)(\epsilon + \wp)}.
\tag{21}
$$

As shown in the accompanying result, the Scabies model presented in (14) is locally asymptotically stable at the disease free equilibrium $\mathcal{E}_0$. According to [58], we address the relevant outcomes.

Theorem 2. *The scabies model presented in* (14) *at the disease free equilibrium* $\mathcal{E}_0$ *is locally asymptotically stable whenever* $\mathcal{R}_0 < 1$ *having assumption* $\left|Arg(\lambda_i)\right| > \frac{\sigma_1\pi}{2}$.

Proof. To show the aforementioned hypothesis, we must first acquire the Jacobian matrix by estimating the parameters (14) at the disease free equilibrium $\mathcal{E}_0$, and then

$$
\mathcal{J}(\mathcal{E}_0) = \begin{bmatrix} -\epsilon & 0 & 0 & 0 & 0 \\ 0 & -\kappa_1 & 0 & 0 & 0 \\ 0 & \eta & -\kappa_2 & 0 & 0 \\ 0 & 0 & \wp & -\kappa_3 & 0 \\ 0 & 0 & 0 & \varsigma & -\theta \end{bmatrix},
\tag{22}
$$

where $\kappa_1 = (\eta + \epsilon)$, $\kappa_2 = (\wp + \epsilon)$, $\kappa_3 = (\varsigma + \epsilon)$. The eigenvalues can be achieved by the following $\lambda^3 + \varpi_1\lambda^2 + \varpi_2\lambda + \varpi_3 = 0$, where $\varpi_1 = \kappa_1 + \kappa_1 + \epsilon$, $\varpi_2 = \kappa_3\epsilon + \kappa_2(\kappa_3 + \epsilon)$, $\varpi_3 = \kappa_1\kappa_2\epsilon(1 - \mathcal{R}_0^2)$.

The indices produced by ϖ_i for $i = 1, 2, 3$ are favorable for clearly for ϖ_i for $i = 1, 2$ whereas ϖ_3 can be positive or negative depending on the number of $\mathcal{R}_0$; in the disease free equilibrium, e.g., the quantity of the fundamental reproduction factor must be smaller than 1, hence the penultimate factor is positive when $\mathcal{R}_0 < 1$. Thus, all the factors involving ϖ_i, $i = 1, 2, 3$ positive, so that, they must fulfill the Routh–Hurtwiz condition [58], this is simple to satisfy, provided the prerequisites are met $\varpi_1\varpi_2 > \varpi_3^2$, where $\varpi_i > 0$, $\forall i = 1, 2, 3$. As a result, the Routh–Hurtwiz requirements guarantee the local asymptotic consistency of the Scabies system (14) at disease free equilibrium $\mathcal{E}_0$. □

Impact of the parameter q

The effect of the quantity q on the fundamental reproductive quantity as well as the persistence of the equilibrium is analyzed in this part. Let us calculate the number of $q^{critical}$ in such a way that

$$
\mathcal{R}_0 = \frac{(\sigma_1(\varsigma + \epsilon)(\epsilon + \wp) + \sigma_2\eta(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_4\eta\varsigma\wp)\mathbf{S}_0^{q^{critical}}}{(\eta + \epsilon)(\varsigma + \epsilon)(\epsilon + \wp)} = 1.
\tag{23}
$$

Therefore, we have

$$
q^{critical} = \ln\left(\frac{(\eta + \epsilon)(\varsigma + \epsilon)(\epsilon + \wp)}{(\sigma_1(\varsigma + \epsilon)(\epsilon + \wp) + \sigma_2\eta(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_4\eta\varsigma\wp)}\right) / \ln(\mathbf{S}_0), \quad \mathbf{S}_0 \neq 1.
\tag{24}
$$

Thus, $\mathcal{R}_0 \leq 1$ whenever $0 < q \leq q^{critical}$ and $\mathcal{R}_0 > 1$ whenever $q > q^{critical}$.

Sensitivity evaluation of the endemic equilibrium

This approach can help anticipate which of the factors in system (14) is more effective at influencing the stable equilibrium outcomes over time $\mathbf{t}$. Determine the endemic equilibrium $\bar{\mathcal{R}} = (\bar{\mathbf{S}}, \bar{\mathbf{I}}_1, \bar{\mathbf{I}}_2, \bar{\mathbf{I}}_3, \bar{\mathbf{M}})$, where factor $q = 1$ is specified in framework (14), then we get $\bar{\mathbf{S}}, \bar{\mathbf{I}}_1, \bar{\mathbf{I}}_2, \bar{\mathbf{I}}_3, \bar{\mathbf{M}}$ (see Box I). From the aforementioned result, we construct the subsequent outcome:

Theorem 3. *The scabies framework presented by* (14) *has:*
(i) If $\mathcal{R}_0 \leq 1$, *then the disease free equilibrium* $\bar{\mathcal{R}}$ *is globally asymptotically stable.*
(ii) If $\mathcal{R}_0 > 1$, *then the endemic equilibrium* $\bar{\mathcal{R}}$ *is globally asymptotically stable.*

Existence and uniqueness of the proposed model

The existence and uniqueness of the scabies framework structure developed by the FF operator are succinctly summarized in (14). So to provide it, we will employ the accompanying procedure to generate the generic Cauchy equation having a FF derivative:

$$
\begin{cases}
{}^{FF}\mathbf{D}_{0,\mathbf{t}}^{\alpha,\omega}\Omega(\mathbf{t}) = \chi(\mathbf{t}, \Omega(\mathbf{t})), \\
\Omega(0) = \Omega_0.
\end{cases}
\tag{25}
$$

From (25), giving the foregoing and pertaining to Definition 1:

$$
\frac{\mathbf{ABC}(\alpha)}{1 - \alpha} \frac{d}{d\mathbf{t}} \int_0^{\mathbf{t}} \chi(\mathbf{s}, \Omega(\mathbf{s})) \bar{E}_\alpha\left(-\frac{\alpha}{1 - \alpha}(\mathbf{t} - \mathbf{s})^\alpha\right) d\mathbf{s} = \omega\mathbf{t}^{\omega-1}\chi(\mathbf{t}, \Omega(\mathbf{t})).
\tag{26}
$$

Considering the implementation of the appropriate integral, the aforementioned outcomes are obtained:

$$
\Omega(\mathbf{t}) = \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega-1}\chi(\mathbf{t}, \Omega(\mathbf{t}))
$$
$$
+ \frac{\omega\alpha}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}(\mathbf{t} - \mathbf{u})^{\alpha-1}\chi(\mathbf{u}, \Omega(\mathbf{u}))\mathbf{u}^{\omega-1}d\mathbf{u} + \Omega(0).
$$

In view of the Picard–Lindelof formulation, yields

$$
\prod_{\delta_1}^{\delta_2} = \mathcal{I}_{\mathbf{r}}(\mathbf{t}_{\mathbf{r}}) \times \overline{\mathbf{A}_0(\Omega_0)},
$$

where $\overline{\mathbf{I}_{\mathbf{s}}(\mathbf{t}_{\mathbf{s}})} = \left[\mathbf{t}_{\mathbf{s}-\zeta_1}, \mathbf{t}_{\mathbf{s}+\zeta_1}\right]$, $\overline{\mathbf{A}_0(\Omega_0)} = \left[\mathbf{t}_0 - \zeta_2, \mathbf{t}_0 + \zeta_2\right]$.

Surmise that

$$
\mathfrak{J} = \sup_{\mathbf{t} \in \prod_{\delta_1}^{\delta_2}} \left\|\chi\right\|.
$$

Furthermore, the norm is applied as follows:

$$
\left\|\chi\right\|_\infty = \sup_{\mathbf{t} \in \prod_{\delta_1}^{\delta_2}} \left\|\chi\right\|,
$$

and surmise the operator

$$
Y\left[\mathbb{C}\left[\mathcal{I}_{\mathbf{s}}(\mathbf{t}_{\mathbf{s}}), \mathbf{A}_{\mathbf{c}}(\mathbf{t}_{\mathbf{s}})\right]\right] \longrightarrow \mathbb{C}\left(\mathcal{I}_{\mathbf{s}}(\mathbf{c}), \mathbf{A}_{\mathbf{c}}(\mathbf{t}_{\mathbf{s}})\right),
$$

$$\bar{\mathbf{S}} = \frac{\theta(\eta + \epsilon)(\varsigma + \epsilon)(\epsilon + \wp)}{\theta[\sigma_2\eta(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_1(\epsilon + \varsigma)(\epsilon + \wp)] + \sigma_4\eta\varsigma\wp},$$

$$\bar{\mathbf{I}}_1 = \frac{\theta[-\epsilon(\eta + \epsilon)(\varsigma + \epsilon)(\epsilon + \wp) + \kappa\{\sigma_2\eta(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_1(\epsilon + \varsigma)(\epsilon + \wp)\}] + \kappa\eta\varsigma\wp\sigma_4}{(\epsilon + \eta)\{\theta[\sigma_2\eta(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_1(\epsilon + \varsigma)(\epsilon + \wp)] + \sigma_4\eta\varsigma\wp\}},$$

$$\bar{\mathbf{I}}_2 = \frac{-\theta\eta\epsilon(\epsilon + \varsigma)}{\theta(\epsilon + \varsigma) + \eta\varsigma\wp\sigma_4 + \theta\wp\sigma_3\eta + \sigma_1(\epsilon + \varsigma)} + \frac{\kappa\eta}{(\epsilon + \eta)(\epsilon + \wp)},$$

$$\bar{\mathbf{I}}_3 = \frac{\eta\wp[\kappa\sigma_4\eta\varsigma\wp + \theta\{\kappa(\eta\sigma_2(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_1(\epsilon + \varsigma)(\epsilon + \wp)\} - \{\epsilon(\epsilon + \eta)(\epsilon + \varsigma)(\epsilon + \wp)\}\}]}{(\epsilon + \eta)(\epsilon + \varsigma)(\epsilon + \wp)\eta\varsigma\wp\sigma_4 + \theta[\eta\sigma_2(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_1(\epsilon + \varsigma)(\epsilon + \wp)]},$$

$$\bar{\mathbf{M}} = -\frac{\eta\wp\varsigma[-\kappa\sigma_4\eta\varsigma\wp + \theta\{-\kappa(\eta\sigma_2(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_1(\epsilon + \varsigma)(\epsilon + \wp)\} + \{\epsilon(\epsilon + \eta)(\epsilon + \varsigma)(\epsilon + \wp)\}\}]}{\theta(\epsilon + \eta)(\epsilon + \varsigma)(\epsilon + \wp)\eta\varsigma\wp\sigma_4 + \theta[\eta\sigma_2(\epsilon + \varsigma) + \sigma_3\eta\wp + \sigma_1(\epsilon + \varsigma)(\epsilon + \wp)]}.$$

Box I.

described by

$$\Upsilon\chi(\mathbf{t}) = \chi_0 + \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\chi(\mathbf{t}, \Omega(\mathbf{t}))$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}(\mathbf{t} - \mathbf{u})^{\alpha - 1}\chi(\mathbf{u}, \Omega(\mathbf{u}))\mathbf{u}^{\omega - 1}d\mathbf{u}.$$

The main intention is to illustrate that the aforesaid operator can transform a completely empty metric space over onto itself. We also aim to demonstrate that it has the potential to identify contractions. First and foremost, we show that

$$\left\|\Upsilon\Omega(\mathbf{t}) - \Omega_0\right\| \leq \mathbf{c},$$

$$\left\|\Upsilon\Omega(\mathbf{t}) - \Omega_0\right\| \leq \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\left\|\chi(\mathbf{t}, \Omega(\mathbf{t}))\right\|_\infty$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}(\mathbf{t} - \mathbf{u})^{\alpha - 1}\left\|\chi(\mathbf{u}, \Omega(\mathbf{u}))\right\|\mathbf{u}^{\omega - 1}d\mathbf{u}$$
$$\leq \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\chi + \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\chi\int_0^{\mathbf{t}}(\mathbf{t} - \mathbf{u})^{\alpha - 1}\mathbf{u}^{\omega - 1}d\mathbf{u}.$$

plugging $\mathbf{u} = \mathbf{tx}$, then gives the subsequent

$$\left\|\Upsilon\Omega(\mathbf{t}) - \Omega_0\right\| \leq \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\chi + \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\chi\mathbf{t}^{\alpha + \omega - 1}B_1(\omega, \alpha).$$

Therefore, we have

$$\left\|\Upsilon\Omega(\mathbf{t}) - \Omega_0\right\| \leq \mathbf{c} \mapsto \chi < \frac{\mathbf{c}B_1(\omega, \alpha)}{\frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1} + \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\mathbf{t}^{\alpha + \omega - 1}}.$$

Surmising that $\Omega_1, \Omega_2 \in \mathbb{C}[\mathcal{I}_\mathbf{s}(\mathbf{t}_\mathbf{s}), \mathbf{A}_\mathbf{c}(\mathbf{t}_\mathbf{s})]$. To obtain the following result, employ the Banach fixed point theorem:

$$\left\|\Upsilon\Omega_1 - \Upsilon\Omega_2\right\| \leq \mathcal{L}_\Omega\left\|\Omega_1 - \Omega_2\right\|_\infty,$$

where $\mathcal{L}_\Omega < 1$.

$$\left\|\Upsilon\Omega_1 - \Upsilon\Omega_2\right\| \leq \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\left\|\chi(\mathbf{t}, \Omega_1) - \chi(\mathbf{t}, \Omega_2)\right\|$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}(\mathbf{t} - \mathbf{u})^{\alpha - 1}\mathbf{u}^{\omega - 1}\left\|\chi(\mathbf{t}, \mathbf{u}_1) - \chi(\mathbf{t}, s_2)\right\|d\mathbf{u}.$$

Using the fact of contraction mapping χ, gives

$$\left\|\Upsilon\Omega_1 - \Upsilon\Omega_2\right\| \leq \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\mathcal{L}_\Omega\left\|\Omega_1 - \Omega_2\right\|_\infty$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\mathcal{L}_\Omega\left\|\Omega_1 - \Omega_2\right\|_\infty\int_0^{\mathbf{t}}(\mathbf{t} - \mathbf{u})^{\alpha - 1}\mathbf{u}^{\omega - 1}d\mathbf{u}$$
$$\leq \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\mathcal{L}_\Omega\left\|\Omega_1 - \Omega_2\right\|_\infty$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\mathcal{L}_\Omega\left\|\Omega_1 - \Omega_2\right\|_\infty\mathbf{t}^{\alpha + \omega - 3}B_1(\omega, \alpha).$$

As a result, we have

$$\left\|\Upsilon\Omega_1 - \Upsilon\Omega_2\right\| \leq \left(\frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{t}^{\omega - 1}\mathcal{L}_\Omega + \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\mathcal{L}_\Omega\mathbf{t}^{\alpha + \omega - 3}B_1(\omega, \alpha)\right)$$
$$\times\left\|\Omega_1 - \Omega_2\right\|_\infty$$

$$< \left(\frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{a}^{\omega - 1}\mathcal{L}_\Omega + \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\mathcal{L}_\Omega\mathbf{a}^{\alpha + \omega - 3}B_1(\omega, \alpha)\right)$$
$$\times\left\|\Omega_1 - \Omega_2\right\|_\infty.$$

Consequently, assuming the essential supposition is valid

$$\mathcal{L}_\Omega < \frac{1 - \alpha}{\mathbf{ABC}(\alpha)}\omega\mathbf{a}^{\omega - 1}\mathcal{L}_\Omega + \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\mathcal{L}_\Omega\mathbf{a}^{\alpha + \omega - 3}B_1(\omega, \alpha).$$

The contraction criterion is then satisfied, i.e.

$$\left\|\Upsilon\Omega_1 - \Upsilon\Omega_2\right\| \leq \left\|\Omega_1 - \Omega_2\right\|_\infty.$$

Ultimately, this shows that the system (14) has a unique solution.

Numerical approach for nonlinear scabies fractal–fractional AB derivative model

The aim of this assignment is to offer a staged process methodology for addressing the scabies infection framework (14), employing the FF operator in the AB perspective. Transforming the framework (14) into the FF-AB derivative pattern:

$$^{ABR}\mathbf{D}_{0,\mathbf{t}}^\alpha(\mathbf{S}(\mathbf{t})) = \omega\mathbf{t}^{\omega - 1}f_1(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t}),$$
$$^{ABR}\mathbf{D}_{0,\mathbf{t}}^\alpha(\mathbf{I}_1(\mathbf{t})) = \omega\mathbf{t}^{\omega - 1}f_2(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t}),$$
$$^{ABR}\mathbf{D}_{0,\mathbf{t}}^\alpha(\mathbf{I}_2(\mathbf{t})) = \omega\mathbf{t}^{\omega - 1}f_3(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t}),$$
$$^{ABR}\mathbf{D}_{0,\mathbf{t}}^\alpha(\mathbf{I}_3(\mathbf{t})) = \omega\mathbf{t}^{\omega - 1}f_4(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t}),$$
$$^{ABR}\mathbf{D}_{0,\mathbf{t}}^\alpha(\mathbf{M}(\mathbf{t})) = \omega\mathbf{t}^{\omega - 1}f_5(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t}). \tag{27}$$

The following findings were achieved by using the AB fractional integral operator:

$$\mathbf{S}(\mathbf{t}) = \mathbf{S}(0) + \frac{\omega\mathbf{t}^{\omega - 1}(1 - \alpha)}{\mathbf{ABC}(\alpha)}f_1(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t})$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}\xi^{\omega - 1}(\mathbf{t} - \xi)^{\alpha - 1}f_1(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi)d\xi,$$

$$\mathbf{I}_1(\mathbf{t}) = \mathbf{I}_1(0) + \frac{\omega\mathbf{t}^{\omega - 1}(1 - \alpha)}{\mathbf{ABC}(\alpha)}f_2(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t})$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}\xi^{\omega - 1}(\mathbf{t} - \xi)^{\alpha - 1}f_2(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi)d\xi,$$

$$\mathbf{I}_2(\mathbf{t}) = \mathbf{I}_2(0) + \frac{\omega\mathbf{t}^{\omega - 1}(1 - \alpha)}{\mathbf{ABC}(\alpha)}f_3(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t})$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}\xi^{\omega - 1}(\mathbf{t} - \xi)^{\alpha - 1}f_3(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi)d\xi,$$

$$\mathbf{I}_3(\mathbf{t}) = \mathbf{I}_3(0) + \frac{\omega\mathbf{t}^{\omega - 1}(1 - \alpha)}{\mathbf{ABC}(\alpha)}f_4(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t})$$
$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)}\int_0^{\mathbf{t}}\xi^{\omega - 1}(\mathbf{t} - \xi)^{\alpha - 1}f_4(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi)d\xi,$$

$$\mathbf{M(t)} = \mathbf{M}(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_5(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \mathbf{t})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \int_0^{\mathbf{t}} \xi^{\omega-1}(\mathbf{t}-\xi)^{\alpha-1} f_5(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi. \quad (28)$$

At $\mathbf{t}_{n_1+1}$, we acquire the accompanying.

$$\mathbf{S}^{n_1+1}(\mathbf{t}) = \mathbf{S}(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_1(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \int_0^{\mathbf{t}_{n_1+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_1(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{I}_1^{n_1+1}(\mathbf{t}) = \mathbf{I}_1(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_2(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \int_0^{\mathbf{t}_{n_1+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_2(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{I}_2^{n_1+1}(\mathbf{t}) = \mathbf{I}_2(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_3(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \int_0^{\mathbf{t}_{n_1+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_3(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{I}_3^{n_1+1}(\mathbf{t}) = \mathbf{I}_3(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_4(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \int_0^{\mathbf{t}_{n_1+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_4(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{M}^{n_1+1}(\mathbf{t}) = \mathbf{M}(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_5(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \int_0^{\mathbf{t}_{n_1+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_5(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi. \quad (29)$$

(29) is restructured considerably, with the corresponding findings:

$$\mathbf{S}^{n_1+1}(\mathbf{t}) = \mathbf{S}(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_1(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \sum_{j=0}^{n_1} \in \mathbf{t}_{\mathbf{t}_j}^{\mathbf{t}_{j+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_1(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{I}_1^{n_1+1}(\mathbf{t}) = \mathbf{I}_1(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_2(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \sum_{j=0}^{n_1} \in \mathbf{t}_{\mathbf{t}_j}^{\mathbf{t}_{j+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_2(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{I}_2^{n_1+1}(\mathbf{t}) = \mathbf{I}_2(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_3(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \sum_{j=0}^{n_1} \in \mathbf{t}_{\mathbf{t}_j}^{\mathbf{t}_{j+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_3(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{I}_3^{n_1+1}(\mathbf{t}) = \mathbf{I}_3(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_4(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \sum_{j=0}^{n_1} \in \mathbf{t}_{\mathbf{t}_j}^{\mathbf{t}_{j+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_4(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi,$$

$$\mathbf{M}^{n_1+1}(\mathbf{t}) = \mathbf{M}(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_5(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{\alpha\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha)} \sum_{j=0}^{n_1} \in \mathbf{t}_{\mathbf{t}_j}^{\mathbf{t}_{j+1}} \xi^{\omega-1}(\mathbf{t}_{n_1+1}-\xi)^{\alpha-1} f_5(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi) d\xi. \quad (30)$$

Furthermore, employing $\xi^{\alpha-1} f_\iota(\mathbf{S}, \mathbf{I}_1, \mathbf{I}_2, \mathbf{I}_3, \mathbf{M}, \xi)$ *for* $\iota = 1, 2, \ldots, 5$ to represent the formulations in (30), in the specified interval $[\mathbf{t}_j, \mathbf{t}_{j+1}]$, the appropriate numerical technique is developed as

$$\mathbf{S}^{n_1+1}(\mathbf{t}) = \mathbf{S}(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_1(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{(\Delta \mathbf{t})^{\alpha}\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha+2)} \sum_{j=0}^{n_1} \left\{ \left\{ \mathbf{t}_j^{\alpha-1} f_1(\mathbf{S}^j, \mathbf{I}_1^j, \mathbf{I}_2^j, \mathbf{I}_3^j, \mathbf{M}^j, \mathbf{t}_j) \right\} \right.$$

$$\times \left((n_1+1-j)^{\alpha}(n_1-j+2\alpha) - (n_1-j)^{\alpha}(n_1-j+2+2\alpha) \right)$$

$$- \left\{ \mathbf{t}_{j-1}^{\omega-1} f_1(\mathbf{S}^{j-1}, \mathbf{I}_1^{j-1}, \mathbf{I}_2^{j-1}, \mathbf{I}_3^{j-1}, \mathbf{M}^{j-1}, \mathbf{t}_{j-1}) \right\}$$

$$\left. \times \left((n_1-j+1)^{\alpha+1} - (n_1-j)^{\alpha}(n_1-j+1+\alpha) \right) \right\},$$

$$\mathbf{I}_1^{n_1+1}(\mathbf{t}) = \mathbf{I}_1(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_2(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{(\Delta \mathbf{t})^{\alpha}\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha+2)} \sum_{j=0}^{n_1} \left\{ \left\{ \mathbf{t}_j^{\alpha-1} f_2(\mathbf{S}^j, \mathbf{I}_1^j, \mathbf{I}_2^j, \mathbf{I}_3^j, \mathbf{M}^j, \mathbf{t}_j) \right\} \right.$$

$$\times \left((n_1+1-j)^{\alpha}(n_1-j+2\alpha) - (n_1-j)^{\alpha}(n_1-j+2+2\alpha) \right)$$

$$- \left\{ \mathbf{t}_{j-1}^{\omega-1} f_2(\mathbf{S}^{j-1}, \mathbf{I}_1^{j-1}, \mathbf{I}_2^{j-1}, \mathbf{I}_3^{j-1}, \mathbf{M}^{j-1}, \mathbf{t}_{j-1}) \right\}$$

$$\left. \times \left((n_1-j+1)^{\alpha+1} - (n_1-j)^{\alpha}(n_1-j+1+\alpha) \right) \right\},$$

$$\mathbf{I}_2^{n_1+1}(\mathbf{t}) = \mathbf{I}_2(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_3(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{(\Delta \mathbf{t})^{\alpha}\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha+2)} \sum_{j=0}^{n_1} \left\{ \left\{ \mathbf{t}_j^{\alpha-1} f_3(\mathbf{S}^j, \mathbf{I}_1^j, \mathbf{I}_2^j, \mathbf{I}_3^j, \mathbf{M}^j, \mathbf{t}_j) \right\} \right.$$

$$\times \left((n_1+1-j)^{\alpha}(n_1-j+2\alpha) - (n_1-j)^{\alpha}(n_1-j+2+2\alpha) \right)$$

$$- \left\{ \mathbf{t}_{j-1}^{\omega-1} f_3(\mathbf{S}^{j-1}, \mathbf{I}_1^{j-1}, \mathbf{I}_2^{j-1}, \mathbf{I}_3^{j-1}, \mathbf{M}^{j-1}, \mathbf{t}_{j-1}) \right\}$$

$$\left. \times \left((n_1-j+1)^{\alpha+1} - (n_1-j)^{\alpha}(n_1-j+1+\alpha) \right) \right\},$$

$$\mathbf{I}_3^{n_1+1}(\mathbf{t}) = \mathbf{I}_3(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_4(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{(\Delta \mathbf{t})^{\alpha}\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha+2)} \sum_{j=0}^{n_1} \left\{ \left\{ \mathbf{t}_j^{\alpha-1} f_4(\mathbf{S}^j, \mathbf{I}_1^j, \mathbf{I}_2^j, \mathbf{I}_3^j, \mathbf{M}^j, \mathbf{t}_j) \right\} \right.$$

$$\times \left((n_1+1-j)^{\alpha}(n_1-j+2\alpha) - (n_1-j)^{\alpha}(n_1-j+2+2\alpha) \right)$$

$$- \left\{ \mathbf{t}_{j-1}^{\omega-1} f_4(\mathbf{S}^{j-1}, \mathbf{I}_1^{j-1}, \mathbf{I}_2^{j-1}, \mathbf{I}_3^{j-1}, \mathbf{M}^{j-1}, \mathbf{t}_{j-1}) \right\}$$

$$\left. \times \left((n_1-j+1)^{\alpha+1} - (n_1-j)^{\alpha}(n_1-j+1+\alpha) \right) \right\}, \quad (31)$$

$$\mathbf{M}^{n_1+1}(\mathbf{t}) = \mathbf{M}(0) + \frac{\omega \mathbf{t}^{\omega-1}(1-\alpha)}{\mathbf{ABC}(\alpha)} f_5(\mathbf{S}^{n_1}, \mathbf{I}_1^{n_1}, \mathbf{I}_2^{n_1}, \mathbf{I}_3^{n_1}, \mathbf{M}^{n_1}, \mathbf{t}_{n_1})$$

$$+ \frac{(\Delta \mathbf{t})^{\alpha}\omega}{\mathbf{ABC}(\alpha)\Gamma(\alpha+2)} \sum_{j=0}^{n_1} \left\{ \left\{ \mathbf{t}_j^{\alpha-1} f_5(\mathbf{S}^j, \mathbf{I}_1^j, \mathbf{I}_2^j, \mathbf{I}_3^j, \mathbf{M}^j, \mathbf{t}_j) \right\} \right.$$

$$\times \left((n_1+1-j)^{\alpha}(n_1-j+2\alpha) - (n_1-j)^{\alpha}(n_1-j+2+2\alpha) \right)$$

$$- \left\{ \mathbf{t}_{j-1}^{\omega-1} f_5(\mathbf{S}^{j-1}, \mathbf{I}_1^{j-1}, \mathbf{I}_2^{j-1}, \mathbf{I}_3^{j-1}, \mathbf{M}^{j-1}, \mathbf{t}_{j-1}) \right\}$$

$$\left. \times \left((n_1-j+1)^{\alpha+1} - (n_1-j)^{\alpha}(n_1-j+1+\alpha) \right) \right\}.$$

Numerical results and discussion

Here, we shall simulate the results in this article to make sure the mathematical argument is correct. Moreover, we investigate a scabies model (14) and associated approximate formulations. While Al-Shamrani et al. [51] integer-order simulation results show similarities
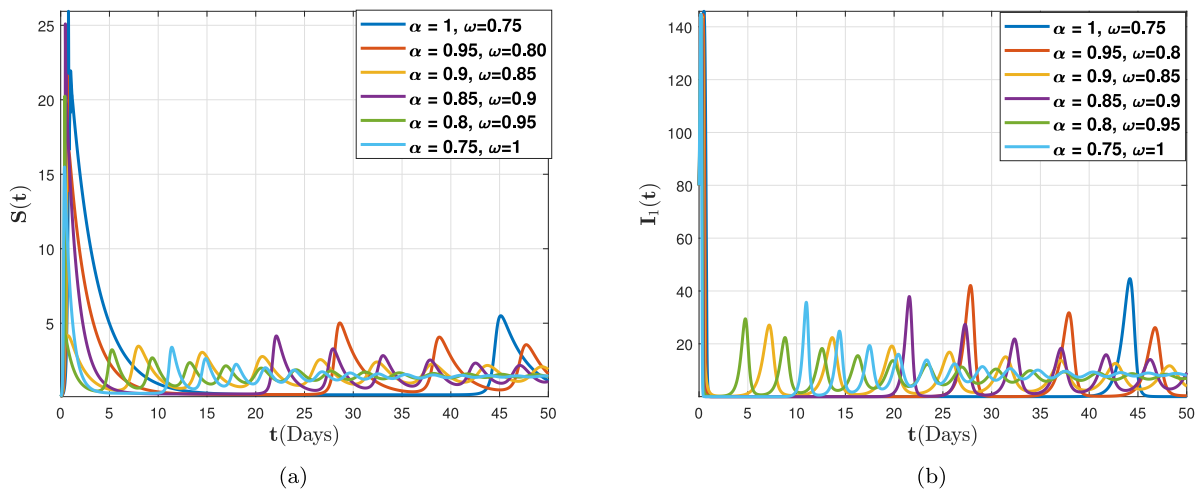
Fig. 3. (a) Susceptible individuals $S(t)$ (b) During the asymptomatic phase after infestation, the growth of infected patients with surviving mites and larvae $I_1(t)$ for system (14) for various fractional-order when ICs $(350, 10, 10, 5, 5)$.
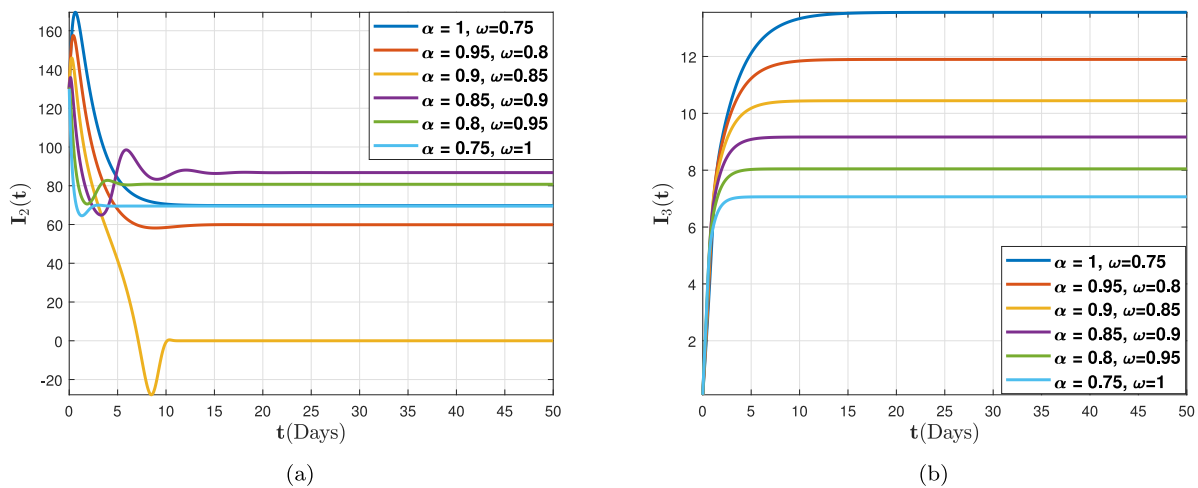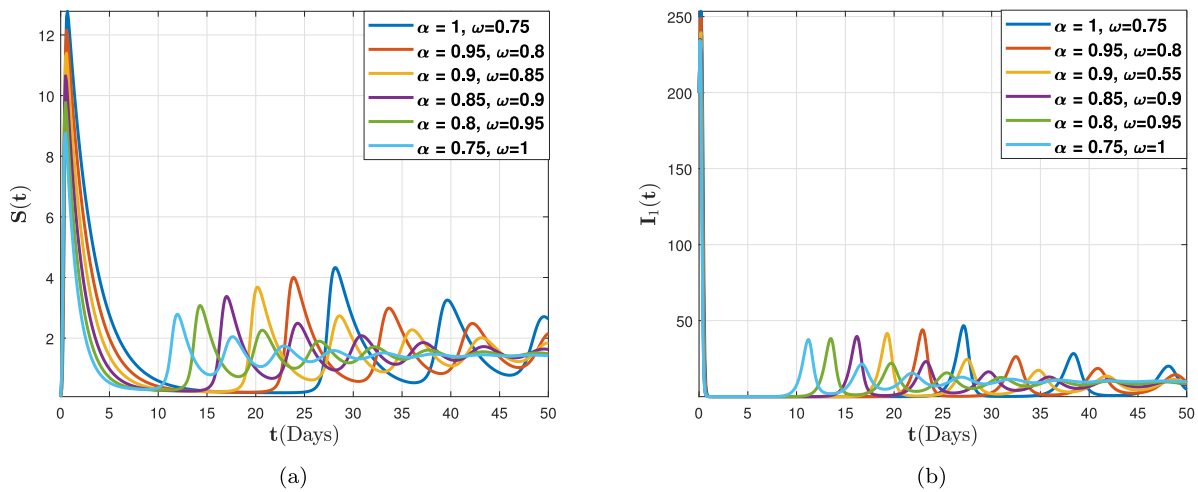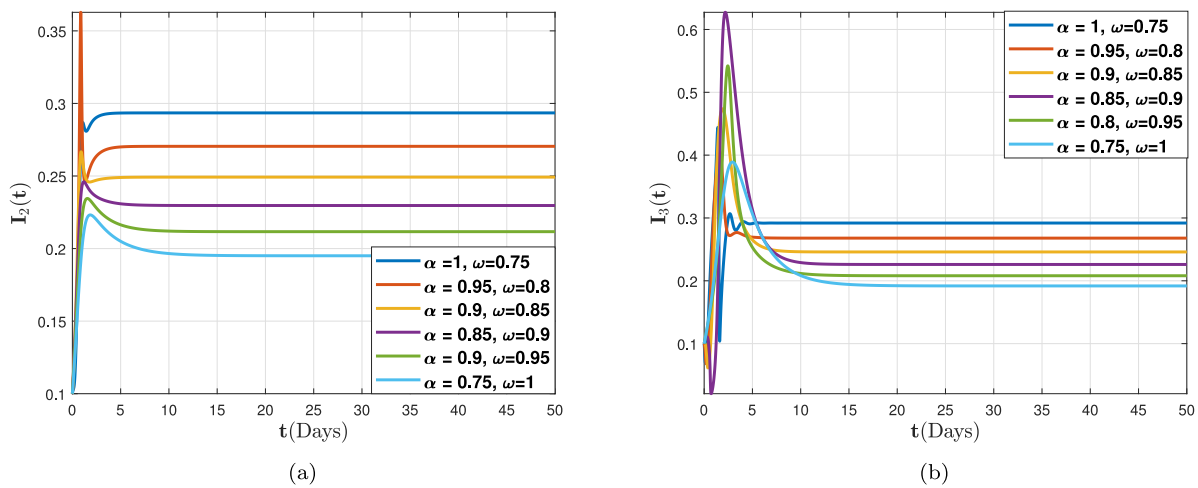

Fig. 4. (a) After getting an infection for system (14), the development of infected persons with active mites and larvae $I_2(t)$ (b) Modeling the growth of contaminated people using only young mites $I_3(t)$ for system (14) for various fractional-order when ICs $(350, 10, 10, 5, 5)$.

and discrepancies between the two modeling techniques, we furnish a comprehensive study of the complexities, including the configurations of the FF-AB operator, demonstrating structures whereby mites relate to scabies perseverance and revealing impressive discrepancies for the model.

In the case of susceptibility treated with a non-ovicidal medication, Figs. 3(a)–(b) show that the treatment period must be around thirty days, a figure that is directly connected to the mite's entire lifespan with the ICs $(350, 10, 10, 5, 5)$. A potentially feasible regimen during the asymptotic phase after infestation, covered by a thin dosage provided periodically, has also been studied. This concludes that switching from a two-dose to a three-dose regimen results in a significant boost in elimination potential in a surprisingly limited period of time. Figures Fig. 4(a)–(b) discovered a monotonous relationship between requirements and relatively brief effectiveness when we evaluated the relevant factors with larvicidal medication. If the problem regarding larvicidal medications is one of conformity, then the fact that Fig. 5 complying and non-compliant individuals are not divided is an apparent flaw in this concept. As a consequence, the efficacy of antifertility medication is often exaggerated, as, on the whole, patients are inclined to accept successive proposals of medication.
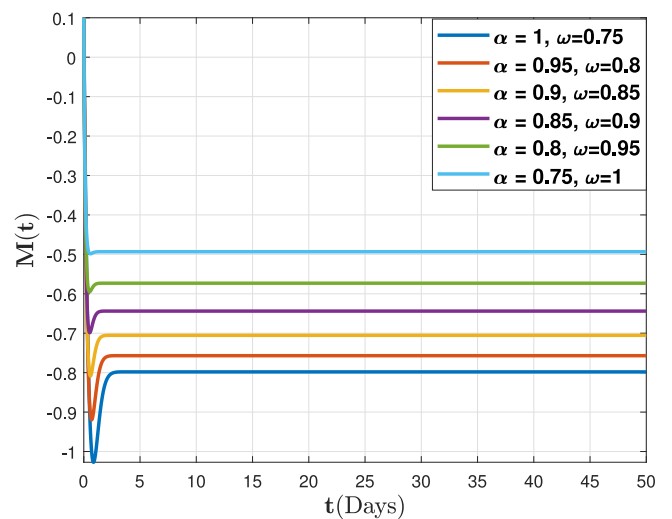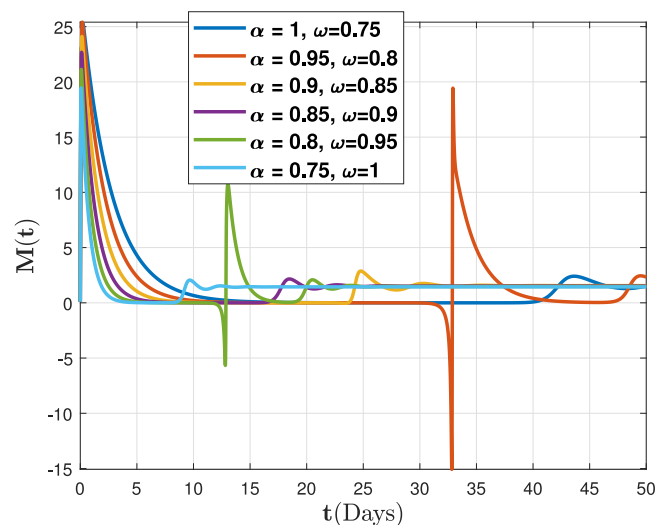

Fig. 5. Adult mites have evolved a mechanism of transmission for system (14) for various fractional-order when ICs $(350, 10, 10, 5, 5)$.
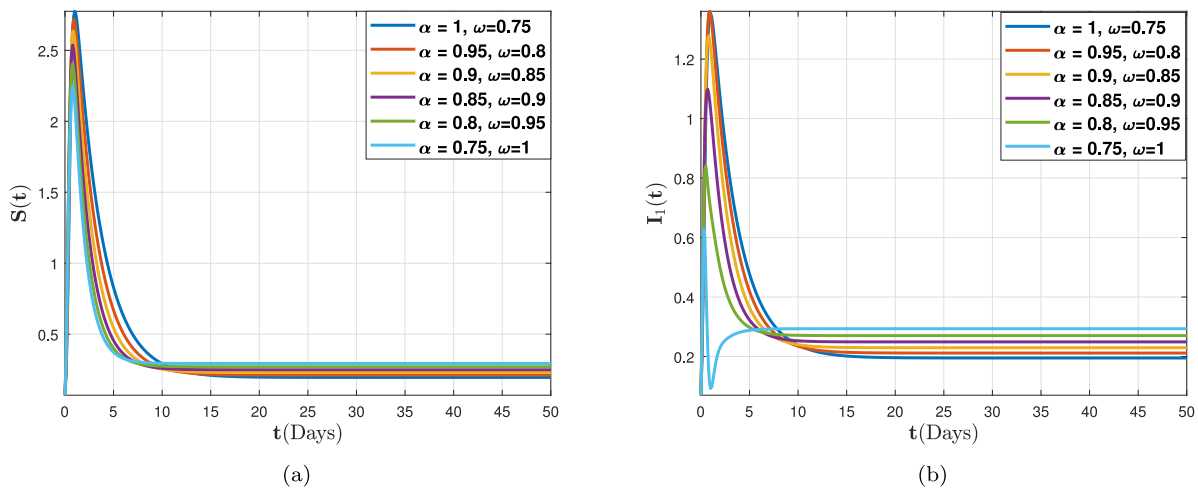
Fig. 6. (a) Susceptible individuals $S(t)$ (b) During the asymptomatic phase after infestation, the growth of infected patients with surviving mites and larvae $I_1(t)$ for system (14) for various fractional-order when ICs $(300, 30, 20, 10, 10)$.

Fig. 7. (a) After getting an infection for system (14), the development of infected persons with active mites and larvae $I_2(t)$ (b) Modeling the growth of contaminated people using only young mites $I_3(t)$ for system (14) for various fractional-order when ICs $(300, 30, 20, 10, 10)$..

Figs. 6(a)–(b) shows that the vulnerable persons $S(t)$ achieve their regular range, whereas $I_1(t)$, $I_2(t)$, $I_3(t)$ and $M(t)$ drop significantly as time passes when ICs is $(300, 30, 30, 10, 10)$. However, Fig. 7 a two-dose regimen might be properly supported for ten years, the chance of dropping dead is even less than 90 percent. On the other hand, Fig. 8 significantly increases the possibility of elimination, with 90 percent of simulated indicating elimination after four years. This indicates that the infection is no longer present.

Figs. 9–10 represents the variability in infection penetration effects the outcome when ICs are $(250, 60, 30, 15, 15)$, as seen in Fig. 11. In our approach, accessibility and compatibility are integrated simultaneously. Unexpectedly, the smallest fraction of affected patients is now monotonically falling between the extremities.

Figs. 12–13 state that when the community of contaminated humans and mature mites accumulates, the spontaneous mortality rates of infected patients and mature mites grow asymptotic. The competence of extremely contagious juveniles and mature mites is dealt with by ICs $(300, 100, 50, 20, 30)$ which is the proportion of overall pathogenic to eventual eradication. Thus, Fig. 14 denotes that the extremely contagious persons' and grownup mites' efficiency is nonincreasing in relation to their respective density.

Fig. 8. Adult mites have evolved a mechanism of transmission for system (14) for various fractional-order when ICs $(300, 30, 20, 10, 10)$.

Fig. 9. (a) Susceptible individuals $S(t)$ (b) During the asymptomatic phase after infestation, the growth of infected patients with surviving mites and larvae $I_1(t)$ for system (14) for various fractional-order when ICs $(250, 60, 30, 15, 15)$.
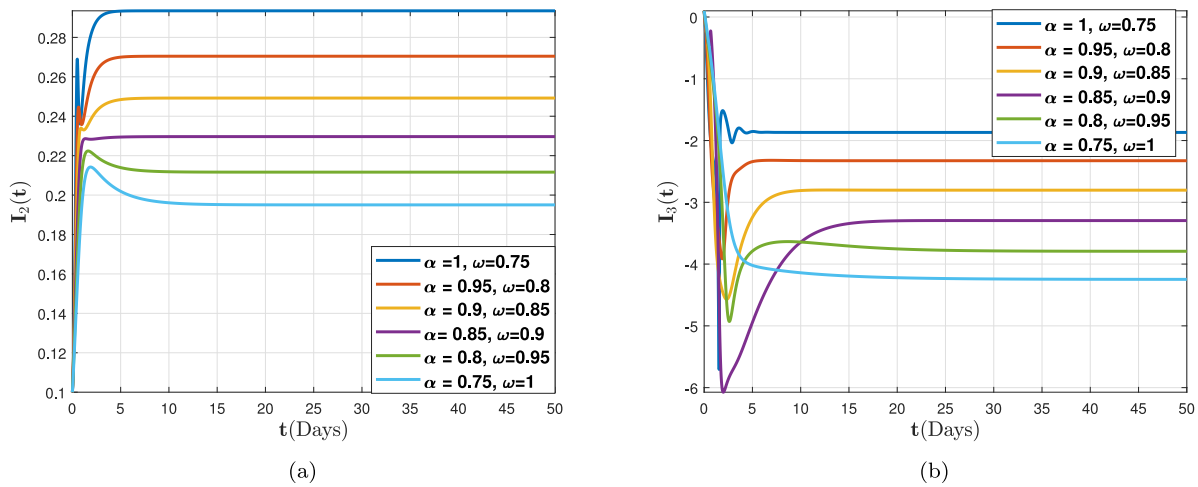

Fig. 10. (a) After getting an infection for system (14), the development of infected persons with active mites and larvae $I_2(t)$ (b) Modeling the growth of contaminated people using only young mites $I_3(t)$ for system (14) for various fractional-order when ICs $(250, 60, 30, 15, 15)$.

Finally, the numerical simulation performed by FF operator considering multiple fractional orders and fractal dimensions leads to a revolutionary reduction in the susceptible population and has a rise in infestation, including mature mites.

Conclusion

In the context of a revolutionary technique described as FF-AB, we developed an analytical simulation of a scabies model with treatment. We generated the structure in the FF version using different concepts. Moreover, we proceeded by describing the system and demonstrating the underlying analytical findings. Using the fundamental reproductive factor, we determined the system's durability and discovered that whenever $\mathcal{R}_0 < 1$, the system is locally asymptotically stable in the illness equilibria. Then, leveraging the novel designed FF-AB operator, we developed the scabies framework and proposed an innovative modeling method for its implementation. We approximated the component values and outfitted the system based on the scabies description in the scenario where $\alpha = \omega$. The fractal–fractional simulation is performed against evidence containing various fractal and fractional order parameters, and it was discovered that altering simultaneous fractal and
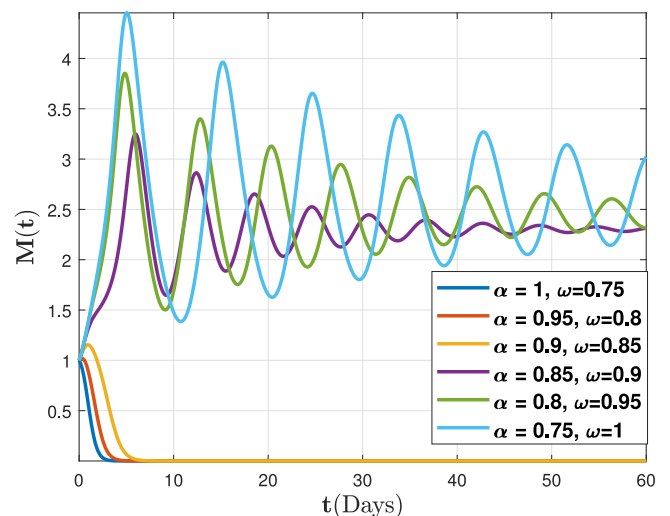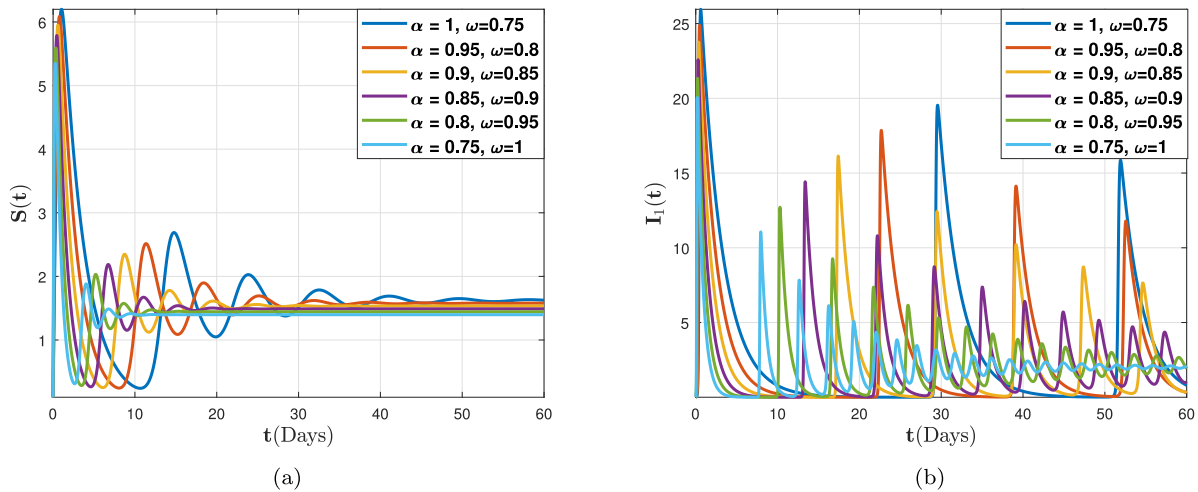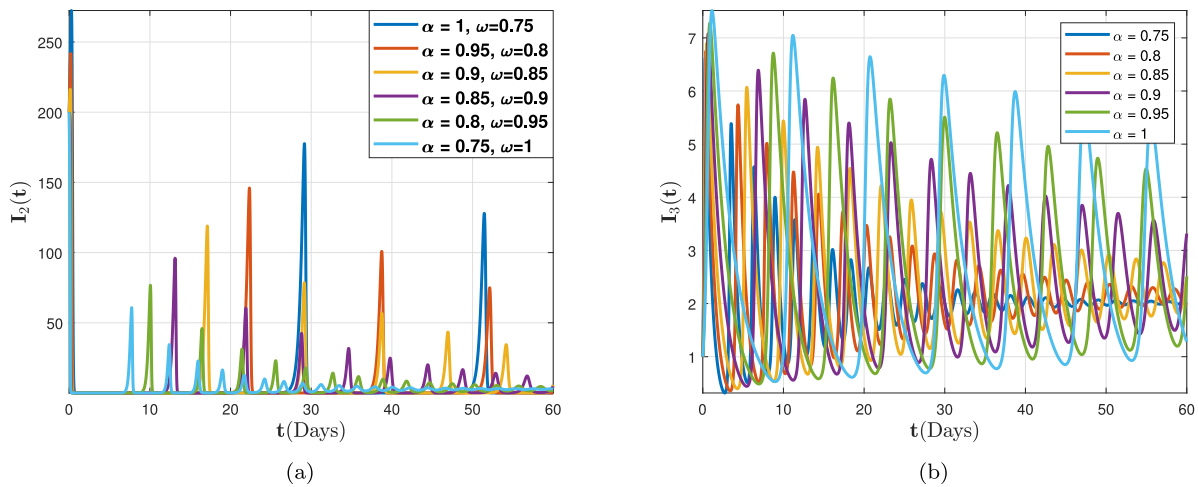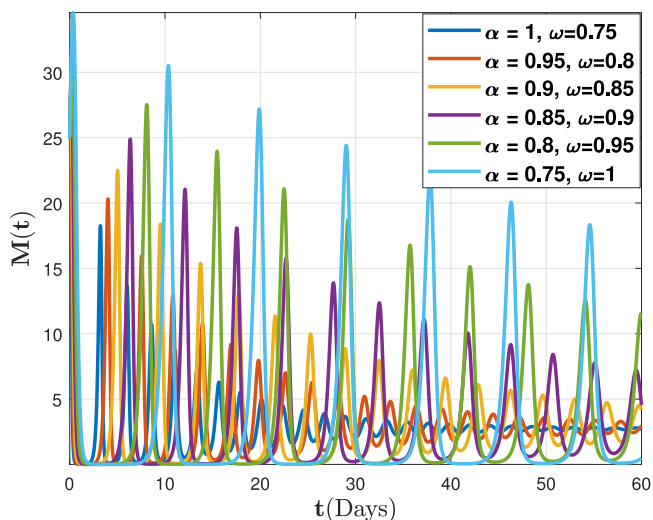

Fig. 11. Adult mites have evolved a mechanism of transmission for system (14) for various fractional-order when ICs $(250, 60, 30, 15, 15)$.

Fig. 12. (a) Susceptible individuals $S(t)$ (b) During the asymptomatic phase after infestation, the growth of infected patients with surviving mites and larvae $I_1(t)$ for system (14) for various fractional-order when ICs $(300, 100, 50, 20, 30)$.

Fig. 13. (a) After getting an infection for system (14), the development of infected persons with active mites and larvae $I_2(t)$ (b) Modeling the growth of contaminated people using only young mites $I_3(t)$ for system (14) for various fractional-order when ICs $(300, 100, 50, 20, 30)$.

Fig. 14. Adult mites have evolved a mechanism of transmission for system (14) for various fractional-order when ICs $(300, 100, 50, 20, 30)$.

fractional phases produces satisfactory adaptation to the actual figures. The system computation is then performed utilizing the anticipated and adjusted attributes, yielding varied illustrated results from multiple eventualities. We established that manipulating both α and ω and modifying them correspondingly generates significant reductions in the infectious sectors of viruses and individuals, whereas a massive rise arises for the susceptible person, using varied configurations of the levels of FF ordering characteristics. This approach can also be expanded in the future by focusing on various FF formulations using various mathematical approaches.

CRediT authorship contribution statement

Saima Rashid: Conceptualization, Methodology, Investigation, Resources, Data curation, Writing – original draft, Supervision, Project administration, Funding acquisition. **Bushra Kanwal:** Conceptualization, Methodology, Investigation, Resources, Data curation, Writing – original draft, Supervision, Project administration, Funding acquisition. **Fahd Jarad:** Conceptualization, Methodology, Investigation, Resources, Data curation, Writing – original draft, Supervision, Project administration, Funding acquisition. **S.K. Elagan:** Conceptualization,

Methodology, Resources, Data curation, Writing – original draft, Supervision, project adminstration, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data availability

No data were used to support this study.

References

[1] Gilmore SJ. Control strategies for endemic childhood scabies. PLoS ONE 2011;6(1):e15990.

[2] Karthikean K. Treatment of scabies: newer perspectives. Postgrad Med J 2005;81(951):7–11.

[3] Elmaraghy MA, Meghawry AME. Inflammatory allergic immune response in scabies pyoderma. J Am Sci 2011;7(8):577–82.

[4] Lydeamore MJ, Campbell PT, Regan DG, Tong SYC, Andrews RM, Steer AC, et al. A biological model of scabies infection dynamics and treatment informs mass drug administration strategies to increase the likelihood of elimination. Math Biosci 2019;309:163–73.

[5] Kearns T, Clucas D, Connors C, Currie BJ, Carapetis JR, Andrews RM. Clinic attendances during the first 12 months of life for aboriginal children in five remote communities of northern australia. PLoS ONE 2013;8(3):1–5.

[6] Ahmed AE, Jradi H, AlBuraikan DA, ALMuqil BI, Albaijan MA, Al-Shehri AM, et al. Rate and factors for scabies recurrence in children in Saudi Arabia: A retrospective study. BMC Pediatr 2019;19(1):187.

[7] Ahmed AE, Shehri AMAl, Al-Jahdali H, Jradi H, ALMuqil BI, AlBuraikan DA, et al. Recurrence rate of scabies in patients 14 years or older in Saudi Arabia. Saudi Med J 2019;40(12):1267–71.

[8] Kinyanjui T, Middleton J, Guttel S, Cassell J, Ross J, House T. Scabies in residential care homes: Modelling, inference and interventions for well-connected population sub-units. PLOS Comput Biol 2018;14(3):e1006046.

[9] Fuller LC. Epidemiology of scabies. Curr Opin Infect Dis 2013;26(2):123–6.

[10] Mellanby K. Scabies in 1976. R Soc Health J 1977;97(1):32–6.

[11] Mellanby K. Transmission of scabies. Br Med J 1941;2(4211):405–6.

[12] Lydeamore MJ. Mechanistic and statistical models of skin disease transmission. Bull Aust Math Soc 2020;101(1):174.

[13] Izri A, Chosidow O. Efficacy of machine laundering to eradicate head lice: rec- ommendations to decontaminate washable clothes, linens, and fomites. Clin Infect Dis 2006;42(2):e9–10.

[14] Engelman D, Steer AC. Control strategies for scabies. Tropical Med Infectious Disease 2018;3(3):98.

[15] University of sydney. scabies. http://medent.usyd.edu.au/fact/scabies.html.

[16] Arlian-Moher LG, Vyszenski DL. Life cycle of sarcoptes scabiei var. canis. J Par-Asitol 1988;74(3):427–30.

[17] Currie BJ, McCarthy JS. Permethrin and ivermectin for scabies. N Engl J Med 2010;362(8):717–25.

[18] Walton SF, Holt DC, Currie BJ, Kemp DJ. Scabies: new future for a neglected disease. Adv Parasitol 2004;57:309–76.

[19] Mellanby K. The development of symptoms, parasitic infection and immunity in human scabies. Parasitology 1944;35(4):197–206.

[20] Agusto FB, Khan MA. Optimal control strategies for dengue transmission in Pakistan. Math Biosci 2018;305:102–21.

[21] Qureshi S, Atangana A. Mathematical analysis of dengue fever outbreak by novel fractional operators with field data. Physica A 2019;526:121127.

[22] Shen W-Y, Chu Y-M, Rahman M, Mahariq I, Zeb A. Mathematical analysis of HBV and HCV co-infection model under nonsingular fractional order derivative. Results Phys 2021;28:104582.

[23] Rashid S, Sultana S, Karaca Y. Khalid A, Chu Y-M. Some further extensions considering discrete proportional fractional operators. Fractals 2022;30(1):2240026.

[24] Zhao T-H, Castillo O, Jahanshahi H, Yusuf A, Alassafi MO, Alsaadi FE, et al. A fuzzy-based strategy to suppress the novel coronavirus (2019-NCOV) massive outbreak. Appl Comput Math 2021;20(1):160–76.

[25] Jin F, Qian Z-S, Chu Y-M, Rahman MU. On nonlinear evolution model for drinking behaviour under Caputo–fabrizio derivative. J Appl Anal Comput 2022;12(2):790–806.

[26] Iqbal SA, Hafez MG, Chu Y-M, Park C. Dynamical analysis of nonauthonomous RLC circuit with the absence and presence of Atangana-Baleanu fractional derivative. J Appl Anal Comput 2022;12(2):770–89.

[27] Khan MA, Gómez-Aguilar F. Tuberculosis model with relapse via fractional conformable derivative with power law. Math Methods Appl Sci 2019;42(2).

[28] Shah K, Jarad F, Abdeljawad T. On a nonlinear fractional order model of dengue fever disease under Caputo–fabrizio derivative. Alexandria Eng J 2020;59(4):2305–13.

[29] Rocha F, Mateus L, Skwara U, Aguiar M, Stollenwerk N. Understanding dengue fever dynamics: A study of seasonality in vector-borne disease models. Int J Comput Math 2016;93(8):1405–22.

[30] van der Linden N, van Gool K, Gardner K, Dickinson H, Agostino J, Regan DG, et al. A systematic review of scabies transmission models and data to evaluate the cost-effectiveness of scabies interventions. PLoS Negl Trop Dis 2019;13(3):e0007182.

[31] Kinyanjui T, Middleton J, Guttel S, Cassell J, Ross J, House T. Scabies in residential care homes: Modelling, inference and interventions for well-connected population sub-units. PLOS Comput Biol 2018;14(3):e1006046.

[32] Caputo M, Fabrizio M. A new definition of fractional derivative without singular kernel. Prog Fract Differ Appl 2015;73:1–13.

[33] Atangana A, Baleanu D. New fractional derivatives with non-local and non-singular kernel. Theory Appl Heat Transfer Model 2022. arXiv 2016:1602.03408.

[34] Scherer R, Kalla SL, Tang Y, Huanget J. The grünwald–letnikov method for fractional differential equations. Comput Math Appl 2011;62:902–17.

[35] Li C, Qian D, Chen YQ. On Riemann–Liouville and Caputo derivatives. Discret Dyn Nat Soc 2011;2011:562494.

[36] Chen S-B, Rajaee F, pour AYousef, Alcaraz R, Chu Y-M, Gómez-Aguilar JF, et al. Antiretroviral therapy of HIV infection using a novel optimal type-2 fuzzy control strategy. Alex Eng J 2021;60(1):1545–55.

[37] Hoan LUC, Akinlar MA, Inc M, Gómez-Aguilar JF, Chu Y-M, Almohsen B. A new fractional-order compartmental disease model. Alex Eng J 2020;59(5):3187–96.

[38] Pandey P, Chu Y-M, Gómez-Aguilar JF, Jahanshahi H, Aly AA. A novel fractional mathematical model of COVID-19 epidemic considering quarantine and latent time. Results Phys 2021;26:104286.

[39] Shen Z-H, Chu Y-M, Khan MA, Muhammad S, Al-Hartomy OA, Higazy M. Mathematical modeling and optimal control of the COVID-19 dynamics. Results Phys 2021;31:105028.

[40] Chu Y-M, Ali A, Khan MA, Islam S, Ullah S. Dynamics of fractional order COVID-19 model with a case study of Saudi Arabia. Results Phys 2021;21:103787.

[41] Saad KM, Gómez-Aguilar JF. Analysis of reaction– diffusion system via a new fractional derivative with nonsingular kernel. Physica A 2018;509:703–16.

[42] Atangana A, Mekkaoui T. Trinition the complex number with two imaginary parts: Fractal, chaos and fractional calculus. Chaos Solit Fract 2019;128:366–81.

[43] Rashid S, Hammouch Z, Aydi H, Ahmad AG, Alsharif AM. Novel computations of the time-fractional Fisher's model via generalized fractional integral operators by means of the elzaki transform. Fractal Fract. 2021;5(3):94.

[44] Alqudah MA, Ashraf R, Rashid S, Singh J, Hammouch Z, Abdeljawad T. Novel numerical investigations of fuzzy Cauchy reaction–diffusion models via generalized fuzzy fractional derivative operators. Fractal Fract 2021;5(4):151.

[45] Gómez-Aguilar JF. Chaos and multiple attractors in a fractal– fractional shinriki's oscillator model. Physica A 2020;539:122918.

[46] Gómez-Aguilar JF. Multiple attractors and periodicity on the vallis model for el Niño/la Niña-southern oscillation model. J Atmos Solar Terr Phys 2020;197:105172.

[47] Solís-Pérez JE, Gómez-Aguilar JF, Escobar-Jiménez RF, Reyes-Reyes J. Blood vessel detection based on fractional hessian matrix with non-singular Mittag–Leffler gaussian kernel. Biomed Sig Proc Contr 2019;54:101584.

[48] Atangana A. Fractal-fractional differentiation and integration: Connecting fractal calculus and fractional calculus to predict complex system. Chaos Solit Fract 2017;102:396–406.

[49] Wang W, Khan MA. Analysis and numerical simulation of fractional model of bank data with fractal-fractional atangana-baleanu derivative. J Comput Appl Math 2019;369:112646.

[50] Li Z, Liu Z, Khan MA. Fractional investigation of bank data with fractal-fractional Caputo derivative. Chaos, Solit Fract 2019;131:109528.

[51] Al-Shamrani NH, Elaiw AM, Batarfia H, Hobiny AD, Dutta H. Global stability analysis of a general nonlinear scabies dynamics model. Chaos Solit Fract 2020;138:110133.

[52] Korobeinikov A. Lyapunov functions and global stability for SIR and SIRS epidemiological models with non-linear transmission. Bull Math Biol 2006;68(3):615–26.

[53] Hethcote HW. The mathematics of infectious diseases. SIAM Rev 2000;42(4):599–653.

[54] Anderson RM, May RM. Infectious diseases in humans: Dynamics and control. Oxford: Oxford University Press; 1992.

[55] Capasso V, Serio G. A generalisation of the kermack-mckendrick deterministic epidemic model. Math Biosci 1978;42(1–2):43–61.

[56] Brown GC, Hasibuan R. Conidial discharge and transmission efficiency of neozygites floridana, an entomopathogenic fungus infecting two-spotted spider mites under laboratory conditions. J Invertebr Pathol 1995;65(1):10–6.

[57] Van Den Driessche P, Watmough J. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. Math Biosci 2002;180(1–2):29–48.

[58] Ahmed E, El-Sayed AMA, El-Saka HAA. On some routh–hurwitz conditions for fractional order differential equations and their applications in lorenz, rössler, chua and chen systems. Phys Lett A 2006;358(1):1–4.