Sigma J Eng Nat Sci, Vol. 43, No. 6, pp. 1930–1942, December, 2025

Sigma Journal of Engineering and Natural Sciences
Web page info: https://sigma.yildiz.edu.tr
DOI: 10.14744/sigma.2025.1926

Research Article

Numerical investigation of the mathematical model including the possibility of reinfection in infectious diseases

Mehmet KOCABIYIK[1,*]

[1]*Department of Mathematics, Burdur Mehmet Akif Ersoy University, Burdur, 15200, Türkiye*

ARTICLE INFO

Article history
Received: 24 July 2024
Revised: 09 September 2024
Accepted: 06 November 2024

Keywords:
Discretization; Infectious Diseases; Numerical Analysis; Stability

ABSTRACT

This study examines the challenges of reinfection in infectious disease modeling, emphasizing the risk that previously infected individuals may lose temporary immunity and become susceptible again. Accurate modeling of reinfection dynamics is vital for understanding disease spread and assessing intervention strategies. To address this, an adapted Susceptible, Infected, Treated, Recovered, Susceptible model is being developed. The adapted model differs from traditional models by incorporating distributed order differential equations, as opposed to standard ordinary or fractional models, which allows for a more flexible representation of disease dynamics. In addition, the adapted version also includes the possibility of reinfection. This improves the ability to interpret the situation of individuals who have re-infected. This approach improves both the interpretability and the computational efficiency of the model. The equilibrium points of the modified system are derived, and stability analyses are performed. The nonstandard finite difference method is used to eliminate instabilities. Numerical simulations are performed to evaluate the performance of the model. The data here is taken from sources derived from real world scenarios. Comparisons are then made based on this data. The results show that the improved model fits experimental data and previous studies quite well, giving a faster and more accurate picture of how diseases change over time. The models showed that adding reinfection can significantly change the expected paths of outbreaks and the effects of interventions. The novelty of this work lies in the incorporation of reinfection dynamics into a distributed order framework, hence augmenting the model's practical applicability compared to previous conventional models. This advance provides faster and more accurate insights into the behaviour of infectious diseases and represents a significant step forward in the field.

Cite this article as: Kocabıyık M. Numerical investigation of the mathematical model including the possibility of reinfection in infectious diseases. Sigma J Eng Nat Sci 2025;43(6):1930–1942.

INTRODUCTION

Mathematical modeling of infectious diseases has received significant attention in recent years. These models are necessary for figuring out how diseases spread, how well public health measures work, and when outbreaks may happen in the future. The convergence of infectious disease

---

***Corresponding author.**
*E-mail address: mkocabiyik@mehmetakif.edu.tr

This paper was recommended for publication in revised form by Editor-in-Chief Ahmet Selim Dalkilic

Published by Yıldız Technical University Press, İstanbul, Turkey

epidemiology and mathematical modeling establishes a comprehensive framework for examining the dissemination and regulation of infectious illnesses.

Mathematical models are important tools in epidemiology because they give us information that we can't get from real-world data alone. For example, models can show how different ways of stopping disease spread, such vaccination or social separation, would work. They can also be used to figure out important epidemiological numbers, including the basic reproduction number [1].

The field of infectious disease modeling has evolved significantly. This evolution has been greatly affected by improvements in computer power and the availability of data. For example, agent-based models show how people in a population interact with each other, showing the differences in behavior and contact patterns [2]. These models have been extremely useful in studying illnesses with complex transmission dynamics, such as influenza and HIV. Network-based models are likewise becoming more common. These make it evident how people are grouped together in space and time. This strategy helps us learn more about how diseases spread across various contact networks, such as schools, workplaces, and homes [3].

These models use real-world data about how people interact with each other to make disease transmission more realistic. Then, they help make focused treatments easier to plan. Recent research has shown how important mathematical models are for dealing with public health emergencies. During the COVID-19 pandemic, for instance, models were used to anticipate how the virus would spread, help make policy decisions, and make sure that healthcare resources were used in the best way possible [4]. These models have enabled governments and health organizations to act quickly and effectively. Consequently, the epidemic's impact has diminished.

Mathematical modeling of infectious illnesses is still a field that is always changing and growing. Novel problems are coming up, such as novel germs and the growing complexity of global health systems. Consequently, the use of models in directing public health interventions is growing progressively essential. It is vital to continuously improving modeling methods in order to overcome these difficulties. Innovations are more likely to improve health outcomes around the world when they are coupled with work from other fields.

A major focus in this area is the development and refinement of epidemic models such as SIR (Susceptible-Infectious-Recovered), SEIR (Susceptible-Exposed-Infectious-Recovered), and STIR (Susceptible-Treated-Infectious-Recovered). Kermack and McKendrick [5] were the first to come up with the SIR model. Researchers that have worked on these models since then have looked into how different types of immunity, latency periods, and transient immunity affect the spread of disease. Differential equations and other mathematical methods have been used to explain how quickly each portion of the model changes. This gives us useful knowledge about how public health measures might work and how infectious illnesses usually act in communities. This continuous research is essential for formulating more effective strategies to manage and prevent the dissemination of infectious illnesses [6].

Çilli et al. employed SI and SIS models to forecast the dissemination of epidemic diseases, including TB, malaria, HIV/AIDS, CCHF, and measles across various nations. Their research demonstrates that these models accurately predict the exact number of infected individuals, showing notable precision and responsiveness to real data trends [7]. Uçar et al. focus on obtaining numerical approximate solutions for the nonlinear modified Burgers' equation (MBE) using modified cubic B-spline differential quadrature methods. The results show that this approach is an effective numerical scheme for solving the MBE, supported by a stability analysis [8].

Karakoç et al. present a finite element scheme for numerically solving the Gilson-Pickering equation using septic B-spline functions. They perform a Von-Neumann stability analysis and demonstrate the reliability of their method by examining the behaviour of single solitons, with the results illustrated by tables and graphs showing the effectiveness of the method [9]. Özgür and Demir explain a mathematical framework to investigate the stability of a neural field model involving two neuron populations with small delays. The primary aim of this analysis is to provide a unifying framework that illustrates the effects of small delays in the context of the Routh-Hurwitz criterion [10].

Elsonbaty et al. present a discrete fractional SITRS (Susceptible, Infectious, Treated, Recovered, Susceptible) model to simulate the COVID-19 pandemic. This study includes the potential for reinfection due to the loss of temporary immunity. They also talked about the wrong idea that the infection rates are the same for people who are normally susceptible and those who are more likely to get sick. Because of what they did, they were able to fit their models to the experimental data [11].

In this study, the SITRS model is investigated using distributed order differential equations. This adjustment makes it possible to better show how the disease changes over time by showing how the rates of infection and recovery fluctuate. The model may take into consideration how complicated and diverse the development of a disease is by using distributed order differential equations. So, it is a more realistic and flexible way to model how infectious diseases like COVID-19 spread and how to stop them.

There is a substantial amount of literature in this field. Some of the studies conducted in this area can be summarised as follows. Ayoub et al. found that the test-negative design was highly effective in predicting the protective efficacy of prior COVID-19 infection against reinfection, as shown by %97.0 and %85.5 protection estimates against Alpha and Beta variants, respectively, in Qatar. The method remains robust to potential misclassification and changing infection levels in the population [12].

Shah and colleagues developed a three-compartment model to examine the reinfection of viral infections, including COVID-19. The model includes a hybrid fractal-fractional equation system that includes key theoretical aspects such as stability analysis and sensitivity of the basic reproduction number. Lagrange interpolation polynomials were used to run numerical simulations that tested the model by comparing real data to simulated data [13].

Schuh et al. propose a mathematical model that encapsulates both the acute and chronic dynamics of post-acute SARS-CoV-2 infection. The model also looks at reinfection situations using attributes that are distinct to each variety. This gives us information that can be used in both clinical and public health settings [14]. Salman and Mohd employed a reaction-diffusion system to simulate the transmission dynamics of COVID-19, incorporating both SEIRS-type kinetics and the spatial distribution of individuals. The research assessed the effects of reinfection, spatial dissemination, and control strategies utilizing optimal control theory and numerical simulations. Thus, the effectiveness of vaccination and treatment in preventing future outbreaks was highlighted. [15].

The literature review shows that the reinfection situation is important in infectious diseases. However, it is noteworthy that the studies focus on a single equation type in the light of these studies. This type of equation is usually an ordinary or fractional differential equation. In other words, expressing the solutions in a single style is quite important even in the numerical analysis section, but it also reveals the necessity of using ordinary and fractional type equation systems in some models. For this reason, this study was planned considering the deficiency in this section of the literature. By adapting the equation system used to distributed order differential equations, comments can be made about both ordinary and fractional order differential equations. This is because distributed order differential equations involve the analysis of other types of equations due to the function they contain.

Although the first contact of infectious diseases is important, reinfection is more important. This is because if the immune system encounters the disease twice, it can have a vital effect. It is therefore essential to be able to interpret the reinfection situation quickly and accurately in different situations or conditions and to develop the ability to act accordingly. The aim of our study is to link the dynamics to a single equation system and to determine numerical simulations under different equation systems or conditions by changing several functions.

In similar studies or models addressing reinfection dynamics, it is observed that different equation systems are developed for each unique effect of the disease, which can lead to delays in interpreting results. These delays can result in wasted time and, ultimately, deaths due to the lack of available resources. The first barrier that distributed order differential equations aim to overcome is precisely this issue. By employing a density function, the equations are designed to adapt to various external influences without needing to change the system itself. In the current model, which incorporates reinfected individuals, this approach has been tested for the first time, and the comparison of results has demonstrated its suitability.

Distributed order differential equations were introduced and developed by Caputo [16]. They include a system of differential equations that integrates Caputo, Riemann-Liouville, and Grünwald-Letnikov fractional derivatives. The most important feature is the density function it contains. This function allows the system to be transformed into either an ordinary or a fractional differential equation. This ability allows a better understanding of different systems of equations and the behaviour of the system under different external influences. Numerous studies have applied distributed order differential equations to diverse areas, including the modeling of dielectric induction and diffusion [17], numerical solutions [18,19], reaction diffusion equations [20], and the SVIR model analysis [21,22].

The numerical analysis in this study is performed using the Nonstandard Finite Difference (NSFD) method. This approach helps get rid of instabilities by letting you choose the denominator function. Introduced by Mickens, the NSFD method has been shown to be applicable to various differential equation systems [23,24], with further details available in relevant literature [25-30].

Computational advances have made it possible to achieve faster results while minimizing errors. This model's modern method is better at getting rid of instability than other methods, which makes it easier to spot associated developments. As a result, the temporal gaps that could cause problems in systems have been cut down, making forecasts more reliable and accurate.

This work is new since it uses distributed order differential equations to create a new version of the SITRS model. This advancement facilitates a more thorough and precise representation of disease dynamics, including the complexities of re-infection and the variability of immunity.

This study significantly impacts infectious disease modeling by the application of modern mathematical methodologies. The better model makes it easier to forecast how diseases will spread. It also makes public health programs work better. It also highlights its relevance and specificity in advancing our understanding of infectious diseases. The results address gaps in current research. That is, it highlights the importance of developing epidemiological models to improve public health outcomes.

There are five sections to this study. The second section provides the necessary background for the reformulation and discretization of the system. The third section presents the discretization of the adapted distributed order system. The fourth section carries out a numerical investigation using equilibrium points under different conditions. The fifth section presents the results and discussion.

DEFINITIONS AND FUNDAMENTALS

Caputo's development of distributed order differential equations represents a significant advance in the field of fractional calculus [16]. This method builds on classical fractional differential equations by letting differential equations be spread out over an interval. Distributed order differential equations are mathematical equations that use several fractional derivatives instead of whole values. This makes it easier to model complicated systems, including how infectious illnesses spread. These explanations are meant to help people understand the ideas better and how they apply to actual life. In this situation, it also has a density function. A density function is a math tool that shows how a certain amount is spread out throughout a population or area. In infectious disease models, it shows how people with different traits (such immunity or susceptibility) are distributed out over the community, which helps us understand how diseases spread in more detail. This flexibility makes it possible to simulate complicated systems more accurately, because distinct processes may change at different speeds.

One advantage of Caputo's distributed order differential equations is that they can handle real-world circumstances better. Applications span a wide range of fields, including physics, engineering, and biology. These equations provide a more detailed understanding of processes related to memory and hereditary influences.

Definition 2.1: The Caputo derivative of order α for a function $f(t)$ is defined as:

$$D^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_{\alpha}^{t} \frac{f^{(n)}(\tau)}{(t-\tau)^{\alpha-n+1}} d\tau, \qquad (1)$$

where, $n \in \mathbb{N}^{+}$, $n - 1 < \alpha \leq n$, Γ is the Gamma function and $f(n)$ is the n - th derivative of $f(t)$ [31]. The Caputo derivative, denoted as $D^{\alpha}f(t)$, is commonly used in fractional calculus to describe processes that involve memory or non-local behavior.

Definition 2.2: The Riemann-Liouville fractional derivative of order α for a function $f(t)$ is defined as:

$$D^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \frac{d^{n}}{dt^{n}} \int_{\alpha}^{t} \frac{f(\tau)}{(t-\tau)^{\alpha-n+1}} d\tau, \qquad (2)$$

where, $n \in \mathbb{N}^{+}$, $n - 1 < \alpha \leq n$, Γ is the Gamma function [31]. The Riemann-Liouville derivative, denoted as $D^{\alpha}f(t)$, is a conventional technique in fractional calculus, mostly utilized to model systems with memory; nonetheless, it encounters difficulties in handling beginning conditions due to its requirement for non-integer initial values.

Definition 2.3: The Grünwald-Letnikov fractional derivative is defined as:

$$D^{\alpha} f(t) = \lim_{h \to 0} \frac{1}{h^{\alpha}} \sum_{k=0}^{\alpha} (-1)^{k} \binom{\alpha}{k} f(t - kh), \qquad (3)$$

where h is a small step size and $\binom{\alpha}{k}$ is the generalized binomial coefficient [31]. The Grünwald-Letnikov derivative, denoted as $D^{\alpha}f(t)$, provides a discrete approximation of fractional derivatives, making it useful for numerical implementations and simulations of systems with fractional dynamics.

Definition 2.4: The Distributed order derivative of a function $f(t)$ can be expressed as:

$$D_{t}^{k(\alpha)}(f(t), k(\alpha), \alpha) = \int_{0}^{1} k(\alpha)\, D^{\alpha} f(t)\, d\alpha, \qquad (4)$$

where $k(\alpha)$ is a density function that defines the distribution of the orders, $D^{\alpha}f(t)$ is a fractional derivative of order α. The distributed order derivative, $D^{k(\alpha)}(f(t), k(\alpha), \alpha)$, introduces a more generalized concept. Instead of applying a single fractional order, it incorporates a range of fractional derivatives, weighted by a function $k(\alpha)$, and integrates over all possible orders. This allows the model to capture a broader spectrum of dynamic behaviors without needing to adjust the system for different conditions. $D^{\alpha}f(t)$ fractional derivative it can be chosen as Caputo, Riemann-Liouville or Grünwald-Letnikov fractional derivative [16,32].

In distributed order differential equations, the Caputo derivative is better, especially for situations with beginning conditions. It makes it easier to understand how to deal with these situationsCaputo employs integer order derivatives to characterize initial conditions, distinguishing it from the Riemann-Liouville derivative. These are more familiar and make more sense in a physical way. This makes it easier to add real-world beginning data to the model. Caputo's technique makes sure that beginning conditions fit together naturally in dispersed order systems with more than one fractional order. This avoids complications in interpretation. As a result, it is more suitable for applications like infectious disease dynamics, where initial states are critical for accurate simulations.

The NSFD method represents a significant innovation in the numerical analysis for differential equations [23]. This method was developed to overcome the stability problems found in traditional finite difference approaches. The approach uses carefully chosen denominator functions to make the numbers more stable. The main advantage of the NSFD method is its ability to increase the stability and accuracy in numerical solutions of differential equations. This benefit is especially helpful when working with difficult issues or equations that oscillate quickly. It can be used in many fields of science and engineering, including as fluid dynamics, reaction-diffusion systems, and epidemiological modeling. It makes it easier to solve complex dynamic systems. People often utilize numerical approaches like Runge-Kutta, Adams, and Theta to study how populations interact with each other. But the magnitude of the time step has a big effect on how accurate and stable they are. In contrast, the NSFD method guarantees positive discrete solutions for positive initial conditions. On the other hand, the structured NSFD scheme may introduce a slight delay in travelling wave propagation when large step sizes are used [33,34].

Definition 2.5: The general form of a nonstandard finite difference scheme for a differential equation $\frac{dy}{dt} = f(y,t)$ can be written as:

$$\frac{y_{n+1}-y_n}{\varphi(h)} = f(y_n, t_n), \tag{5}$$

where y_n is the approximation of $y(t)$ at the discrete time step t_n and h is the time step size. $\varphi(h)$ is designed to fit the behavior of a continuous system. It is usually a function chosen to ensure that certain properties are preserved. In the literature it is referred to as the denominator function [23].

THE DISCRETIZATION OF ADAPTED ENDEMIC MODEL

In this section, we present a new SITRS model that includes two groups of susceptible individuals $S_1(t)$ and $S_2(t)$, an infected individual $I(t)$, a treatment individual $T(t)$, and a recovered individual $R(t)$. The improved model builds on the traditional SITR model by allowing for the possibility that recovered individuals may lose temporary immunity and become re-susceptible to the virus. In addition, the original model assumes that infection rates are the same for both the general susceptible population and for elderly or seriously ill individuals. This model does not have this limitation [11]. In addition, the system is transformed into a distributed order model with different arrangement. The updated SITRS model is presented below:

$$\begin{aligned}
&\mathrm{D_t^{k(\alpha)}S_1 = G - b\, I(t)S_1(t) - dS_1(t) + lR(t)},\\
&\mathrm{D_t^{k(\alpha)}S_2 = G - b\, I(t)S_2(t) - dS_2(t) + lR(t)},\\
&\mathrm{D_t^{k(\alpha)}I = (-\tau - d + c\,)I(t) + bI(t)\big(S_1(t) + S_2(t)\big) - T(t)},\\
&\mathrm{D_t^{k(\alpha)}T = \gamma I(t) - wT(t)},\\
&\mathrm{D_t^{k(\alpha)}R = \tau I(t) - (d + l)\; R(t)}.
\end{aligned} \tag{6}$$

In this definition, the $D^\alpha f(t)$ derivative is chosen as a Caputo type fractional derivative. The reason for this selection is to choose the most suitable fractional derivative that can be used with the initial data we have.

The parameters in this system are given in Table 1, where G is the natural growth rate of susceptible individuals; b is the infection rate of contact between susceptible and infected individuals; l is the rate of loss of immunity of recovered individuals; d is the mortality rate; c is the rate of dry cough, cold and fever of infected individuals; γ is the cure rate of infected individuals; τ is the recovery rate of treated individuals; and w is the treatment control parameter of treated individuals. The constants listed in Table 1, are derived from empirical studies where these parameters have been measured and analyzed in real world settings. These values have been chosen from existing literature that explicitly deals with these variables to make sure that the comparisons are correct and useful. By basing these constants on real-world data, we make the model stronger and more believable, which makes it more like how diseases really work. If the defined discretization is applied to the system of equations, the discrete state of the system becomes as follows:

$$\begin{aligned}
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=0}^{n+1} u_i^{\alpha_j} S_{n+1-i}^1 = G - b\, I_n S_{n+1}^1 - dS_{n+1}^1 + lR_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=0}^{n+1} u_i^{\alpha_j} S_{n+1-i}^2 = G - b\, I_n S_{n+1}^2 - dS_{n+1}^2 + lR_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=0}^{n+1} u_i^{\alpha_j} I_{n+1-i} = (-\tau - d + c)I_{n+1} + bI_{n+1}(S_n^1 + S_n^2) - T_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=0}^{n+1} u_i^{\alpha_j} T_{n+1-i} = \gamma I_n - wT_{n+1},\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=0}^{n+1} u_i^{\alpha_j} R_{n+1-i} = \tau I_n - (d + l)R_{n+1},
\end{aligned} \tag{7}$$

where v, $j = 1,2,3,4,5$, $0 < \alpha_j < 1$, $u_0^{\alpha_j} = (\varphi_v(h))^{-\alpha_j}$, and denominator functions are chosen as:

$$\begin{aligned}
&\varphi_1(h) = \frac{1-e^{dh}}{d}, \varphi_2(h) = \frac{1-e^{dh}}{d}, \varphi_3(h) = \frac{1-e^{(\tau+d-c\,)h}}{\tau + d - c},\\
&\varphi_4(h) = \frac{1-e^{wh}}{w},\ \ \varphi_5(h) = \frac{1-e^{(d+l)h}}{d+l}.
\end{aligned} \tag{8}$$

By expanding the terms on the left and rearranging the system of equations,

$$\begin{aligned}
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\left(u_0^{\alpha_j} S_{n+1}^1 + \sum_{i=1}^{n+1} u_i^{\alpha_j} S_{n+1-i}^1\right) = G - b\, I_n S_{n+1}^1 - dS_{n+1}^1 + lR_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\left(u_0^{\alpha_j} S_{n+1}^2 + \sum_{i=1}^{n+1} u_i^{\alpha_j} S_{n+1-i}^2\right) = G - b\, I_n S_{n+1}^2 - dS_{n+1}^2 + lR_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\left(u_0^{\alpha_j} I_{n+1} + \sum_{i=1}^{n+1} u_i^{\alpha_j} I_{n+1-i}\right) = (-\tau - d + c\,)I_{n+1} + bI_{n+1}(S_n^1 + S_n^2) - T_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\left(u_0^{\alpha_j} T_{n+1} + \sum_{i=1}^{n+1} u_i^{\alpha_j} T_{n+1-i}\right) = \gamma I_n - wT_{n+1},\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\left(u_0^{\alpha_j} R_{n+1} + \sum_{i=1}^{n+1} u_i^{\alpha_j} R_{n+1-i}\right) = \tau I_n - (d + l)R_{n+1}.
\end{aligned} \tag{9}$$

Table 1. Description of the constants for the distributed order model

Symbol	Description	Symbol	Description
G	*Natural grow rate*	c	*Dry cough, cold and fever rate*
b	*Infection rate*	γ	*Treatment rate*
l	*Loss of temporary immunity rate*	w	*Treatment control parameter*
d	*Death rate*	τ	*Recovering rate*

Hence

$$\begin{aligned}
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_1(h)^{-\alpha_j}S^1_{n+1}+\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S^1_{n+1-i}=G-b\,I_nS^1_{n+1}-dS^1_{n+1}+lR_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_2(h)^{-\alpha_j}S^2_{n+1}+\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S^2_{n+1-i}=G-b\,I_nS^2_{n+1}-dS^2_{n+1}+lR_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_3(h)^{-\alpha_j}I_{n+1}+\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}I_{n+1-i}=(-\tau-d+c)I_{n+1}+bI_{n+1}(S^1_n+S^2_n)-T_n,\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_4(h)^{-\alpha_j}T_{n+1}+\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}T_{n+1-i}=\gamma I_n-wT_{n+1},\\
&\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_5(h)^{-\alpha_j}R_{n+1}+\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}R_{n+1-i}=\tau I_n-(d+l)R_{n+1}.
\end{aligned}\tag{10}$$

The last expressions can be simplified as follows

$$\begin{aligned}
&S^1_{n+1}\left(\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_1(h)^{-\alpha_j}+b\,I_n+d\right)=G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S^1_{n+1-i},\\
&S^2_{n+1}\left(\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_2(h)^{-\alpha_j}+b\,I_n+d\right)=G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S^2_{n+1-i},\\
&I_{n+1}\left(\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_3(h)^{-\alpha_j}+\tau+d-c-b(S^1_n+S^2_n)\right)=-T_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}I_{n+1-i},\\
&T_{n+1}\left(\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_4(h)^{-\alpha_j}+w\right)=\gamma I_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}T_{n+1-i},\\
&R_{n+1}\left(\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_5(h)^{-\alpha_j}+d+l\right)=\tau I_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}R_{n+1-i}.
\end{aligned}\tag{11}$$

with $\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\varphi_i(h)^{-\alpha_j}=K_i, for\ i,j=1,2,3,4,5,$ and finally

$$\begin{aligned}
S^1_{n+1}&=\frac{G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S^1_{n+1-i}}{K_1+b\,I_n+d},\\
S^2_{n+1}&=\frac{G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S^2_{n+1-i}}{K_2+b\,I_n+d},\\
I_{n+1}&=\frac{-T_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}I_{n+1-i}}{K_3+\tau+d-c\ -b(S^1_n+S^2_n)},\\
T_{n+1}&=\frac{\gamma I_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}T_{n+1-i}}{K_4+w},\\
R_{n+1}&=\frac{\tau I_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}R_{n+1-i}}{K_5+d+l}.
\end{aligned}\tag{12}$$

Thus, the final state of the discretized system is found as Equation 12.

NUMERICAL INVESTIGATION OF DISCRETIZED SYSTEM

In this section, equilibrium point analysis and numerical simulations have been performed. Constant values are important for analysis and simulations. The values have been chosen as shown in Table 2 and evaluations are made accordingly [11].

Equilibrium point analysis is very important for this model since it helps us understand how the system will behave throughout time. We can find out if these points are stable by finding and studying equilibrium points. This tells us if the disease will stay the same, get worse, or die out in a certain group of people. This method allows researchers and public health officials to predict what might happen if an infectious disease emerges and identify the best ways to stop it. For example, an unstable balance point indicates that a small change, such as an increase in infection rates, could cause infections to return. A stable point means the disease is likely to remain under control.

The equilibrium points chosen for this model are meant to show how things work in the actual world, especially during epidemics of infectious diseases like COVID-19. One of the equilibrium points, for instance, would be when the population reaches herd immunity, which is important for figuring out what has to happen to stop the virus from spreading.

The identified equilibrium points can also help identify constraints for intervention strategies. If the study indicates that a particular region has a low incidence of infection, this information can help public health officials establish regulations to maintain low transmission levels. By comparing equilibrium points with existing data from COVID-19 outbreaks, such as hospitalization rates and infection trends, we can improve our understanding of the mechanisms of the disease. This interpretation strengthens the theoretical framework of the model and highlights its practical importance in addressing public health challenges.

Firstly, finding the equilibrium point is a crucial requirement in discretized systems. This requires the solution of Equation 13.

Table 2. Initial conditions and the constants' values for equilibrium point analysis

Initial Conditions	**Values**	**Parameter Conditions 1**	**Values**	**Parameter Conditions 2**	**Values**
$S_1(0)$	0.65	G	0.3	G	0.3
$S_2(0)$	0.15	b	0.35	b	0.3
I(0)	0.75	l	0.3	l	0.3
T(0)	0.35	d	0.25	d	0.2
R(0)	0.1	c	0.01	c	0.01
		γ	0.2	γ	0.03
		w	0.1	w	0.1
		τ	0.1	τ	0.15

$$S_n^1=\frac{G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S_n^1}{K_1+b\,I_n+d},$$

$$S_n^2=\frac{G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S_n^2}{K_2+b\,I_n+d},$$

$$I_n=\frac{-T_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}I_n}{K_3+\tau+d-c\,-b(S_n^1+S_n^2)}, \tag{13}$$

$$T_n=\frac{\gamma I_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}T_n}{K_4+w},$$

$$R_n=\frac{\tau I_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}R_n}{K_5+d+l}.$$

If the system expressed by Equation 13 is solved using the Maple software package, the equilibrium points can be determined. However, due to the lengthy calculations required for the second equilibrium point, it is expressed later using parameters.

$$Eq_1=\left(\frac{G}{K_1+d+\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}},\frac{G}{K_2+d+\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}},0{,}0{,}0\right) \tag{14}$$

$$Eq_2=(eq_{21},eq_{22},eq_{23},eq_{24},eq_{25}) \tag{15}$$

Another requirement for the analysis of equilibrium points is the Jacobian matrix. Therefore, the Jacobian matrix corresponding to the discretized Equation 12 is determined as follows:

$$J=\begin{pmatrix} s_{11} & 0 & s_{13} & 0 & s_{15}\\ 0 & s_{22} & s_{23} & 0 & s_{25}\\ s_{31} & s_{32} & s_{33} & s_{34} & 0\\ 0 & 0 & s_{43} & s_{44} & 0\\ 0 & 0 & s_{53} & 0 & s_{55}\end{pmatrix}$$

$$s_{11}=\frac{-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}}{K_1+b\,I_n+d},s_{13}=\frac{-b\,(G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S_n^1)}{(K_1+b\,I_n+d)^2},$$

$$s_{15}=\frac{l\,R_n}{K_1+b\,I_n+d},s_{22}=\frac{-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}}{K_2+b\,I_n+d},$$

$$s_{23}=\frac{-b\,(G+l\,R_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}S_n^2)}{(K_2+b\,I_n+d)^2},s_{25}=\frac{l}{K_2+b\,I_n+d},$$

$$s_{31}=\frac{b\,(-T_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}I_n)}{(K_3+\tau+d-c\,-b(S_n^1+S_n^2))^2},s_{32}=\frac{b\,(-T_n-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}I_n)}{(K_3+\tau+d-c\,-b(S_n^1+S_n^2))^2},$$

$$s_{33}=\frac{-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}}{K_3+\tau+d-c\,-b(S_n^1+S_n^2)},s_{34}=\frac{-1}{K_3+\tau+d-c\,-b(S_n^1+S_n^2)},$$

$$s_{43}=\frac{\gamma I_n}{K_4+w},\ s_{44}=\frac{-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}}{K_4+w},s_{53}=\frac{\tau}{K_5+d+l},$$

$$s_{55}=\frac{-\sum_{j=1}^{A}\frac{k(\alpha_j)}{A}\sum_{i=1}^{n+1}u_i^{\alpha_j}}{K_5+d+l}.$$

In order to perform a stability analysis using the Jacobian matrix and eigenvalues, it is necessary to use some criteria. The following definition is given.

Definition 4.1: According to the Matignon criterion, let J denote the Jacobian matrix obtained by the classical method, and let λ represent the set of eigenvalues of this Jacobian matrix. The equilibrium point is considered to be locally asymptotically stable if the following condition is satisfied [35]:

$$|arg(\lambda(J))|>\frac{\alpha\pi}{2}.$$

The choice of the Matignon criterion for stability analysis is particularly suitable for this model due to the inclusion of density functions in distributed order differential equations, which exhibit fractional-order behavior. This criterion effectively addresses the complexities associated with fractional derivatives, enhancing the understanding of stability.

The Matignon criterion is crucial for determining the stability of fractional differential equations. This makes it easier to see how stable solutions are over time, even while fractional derivatives don't behave like they should. On the other hand, the Matignon criterion can handle the problems that come up in fractional systems, where memory effects are quite important.

This method is very important for making sure that systems with distributed order behavior work correctly. By looking at stability areas, it helps researchers uncover important things that determine how well a system works. Employing the Matignon criterion, researchers can ascertain the long-term outcomes and the stability of various equilibrium points. This ultimately leads to enhanced strategies for disease management and treatment.

Analysis of stability for the first initial condition

According to the first initial conditions, the equilibrium points E_1 and E_2 are as follows:

$$E_1 = (0.2383316093, 0.2383316093, 0, 0, 0),$$
$$E_2 = (3.821405429, 3.821405429, -6.053820498, -1.091269786, 0.3887500955),$$

where $k(\alpha) = \Gamma(2 - \alpha)$. Substituting the first equilibrium point into the Jacobian matrix yields the following characteristic equation:

$$P(\lambda) = (0.008008005674 + \lambda)^2\ (0.0009463035997 + 0.1465278380\ \lambda + 0.2360336746\ \lambda^2 + \lambda^3)$$

After solving the characteristic equations, it is evident that

$$\lambda_1 = -0.008008005674,\ \lambda_2 = -0.008008005674,$$
$$\lambda_3 = -0.006463069302,\ \lambda_4 = -0.008570149079 + 0.3825488356i,\ \lambda_5 = -0.008570149079 - 0.3825488356i.$$

If the Matignon criterion is checked with the eigenvalues, the result is $|\arg(\lambda_g)| = \pi > \alpha\pi/2$, *for* $g = 1,2,3$, with the order satisfying $0 < \alpha < 2$ On the other hand, $|\arg(\lambda_4)| = |\arg(\lambda_5)| = 0.32158245\pi > \alpha\pi/2$ is satisfying $\alpha < 0.64316490$.

Our conclusion is the equilibrium point is E_1 locally asymptotically stable if the order satisfies the condition $0 < \alpha < 0.64316490$. Equilibrium point E_1 represents the disease-free equilibrium, which is significant because it indicates a state where the disease does not persist in the population. It is important to understand how stable is E_1, since it can help us figure out if therapies are working to stop the spread of the disease. If the eigenvalues meet the stability criterion for E_1, it means that the disease can be gotten rid of in some situations. So, looking at this equilibrium point not only shows how important it is to the model, but it also shows how useful it is for public health initiatives that want to manage disease.

Similarly, by substituting the second equilibrium point into the Jacobian matrix gives the characteristic equation:

$$P(\lambda)=\lambda^5 + 0.1680779743\,\lambda^4 + 37.44603747\,\lambda^3 + 1.926168284\,\lambda^2 - 0.2164013283\,\lambda - 0.002077367950$$

After solving the characteristic equations, it can be seen that

$$\lambda_1 = -0.05830984123 + 6.119020776i,$$
$$\lambda_2 = -0.05830984123 - 6.119020776i,$$
$$\lambda_3 = -0.009004270986,\ \lambda_4 = -0.1025395746,$$
$$\lambda_5 = 0.06008555375.$$

If the Matignon criterion is checked on the basis of the eigenvalues obtained $|\arg(\lambda_3)| = |\arg(\lambda_4)| = \pi > \alpha\pi/2$, with the order satisfying $0 < \alpha < 2$, and $|\arg(\lambda_1)| = |\arg(\lambda_2)| = 0.3204179\,\pi > \alpha\pi/2$ is satisfying $\alpha < 6408358$. On the other hand, since it is $|\arg(\lambda_5)| = 0 < \alpha\pi/2$, it was seen that the second eigenvalue does not satisfy Matignon criterion. So, our conclusion is that E_2 is not locally asymptotically stable under these conditions.

Stability analysis for the second condition

The equilibrium point E_2 according to the second condition is

$$E_2 = (2.292233390, 2.292233390, -3.756823905, -0.1015815158, -0.3738128142)$$

The characteristic equation is obtained by substituting the second equilibrium point under the second set of conditions:

$$P(\lambda) = \lambda^5 + 0.003221828833\,\lambda^4 + 0.00005278835547\,\lambda^3 + 0.8488407333\ 10^{-7}\lambda^2 + 0.4624925316\ 10^{-10}\,\lambda + 0.8237844801\ 10^{-14}$$

After solution of characteristic equations, it is seen that

$$\lambda_1 = -0.0007656339572 + 0.006977097614i,$$
$$\lambda_2 = -0.0007656339572 - 0.006977097614i,$$
$$\lambda_3 = -0.0004046569892,\ \lambda_4 = -0.0006299162648,$$
$$\lambda_5 = -0.0006559876645.$$

Using the eigenvalues obtained to check the Matignon criterion, $|\arg(\lambda_3)| = |\arg(\lambda_4)| = |\arg(\lambda_5)| = \pi > \alpha\pi/2$, with the order satisfying $0 < \alpha < 2$. On the other hand, $|\arg(\lambda_1)| = |\arg(\lambda_2)| = 0.3291035\,\pi > \alpha\pi/2$ is satisfying $\alpha < 0.6582070$. E_2 is locally asymptotically stable if the order satisfies the condition $0 < \alpha < 0.6582070$.

An examination of the numerical simulations after the equilibrium point analysis has the following results.

Figure 1 shows that with the density function chosen according to condition 1, the results are quite consistent with the literature. At the same time, it has been observed that there is an approach to the equilibrium point with this shape. When examining the literature studies in this area, it has been shown through simulation that the equilibrium point of the ordinary order differential equation can be reached by selecting the equilibrium point [11,36].

Figure 2 examines the behaviour of susceptible and treated individuals in time-varying situations. As can be seen here, the dynamic analysis of the system can be interpreted quickly by changing the density function. In other words, the aim of the study is achieved. It can sometimes take a long time to understand the effects of external influences on infectious diseases. Writing the density function based on these external effects can minimize this effect. Looking more closely at Figure 2, the rate of increase of susceptible and treated individuals can be expressed as a function of time and external effects. In this way it has been seen that the density function can be more effective in determining the dynamics by choosing the threshold value of α.

Figure 3 shows that the system works under changing conditions and analysis can be made according to the equilibrium points. Here it is shown that changing external effects can be slower in reaching the equilibrium point. It is not necessary to set up another system of equations for this, it is shown that only the change in the equilibrium point is sufficient.

Figure 4 shows the effect of changing the density function for sensitive individuals. It has been found that the analysis is faster if the external effects are defined within the density function. As can be seen in the figure, it has been observed that the variability increases with the change. In other words, it has been shown that when the re-infection rate increases, the effect of the disease can be observed again with this system of equations.

In Figure 5, the variability between infected, treated and recovered individuals is obtained by selecting the density function. By changing the density function, for example, the number of individuals recovering over time can be determined. However, this system of equations can also be used to determine the acceleration or deceleration of the increase in newly infected individuals, as shown in the other simulation in the figure. The same dynamics can be determined for infected and treated individuals. The simulations in this section were carried out using the MATLAB package programme.

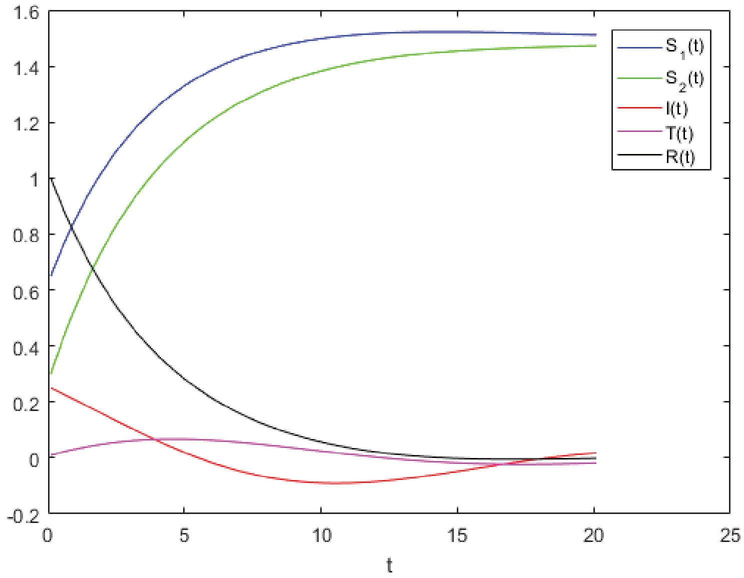

Figure 1. Time series of population variables in the SITRS model with conditions 1 for $k(\alpha) = \Gamma(\alpha - 0.5)$.

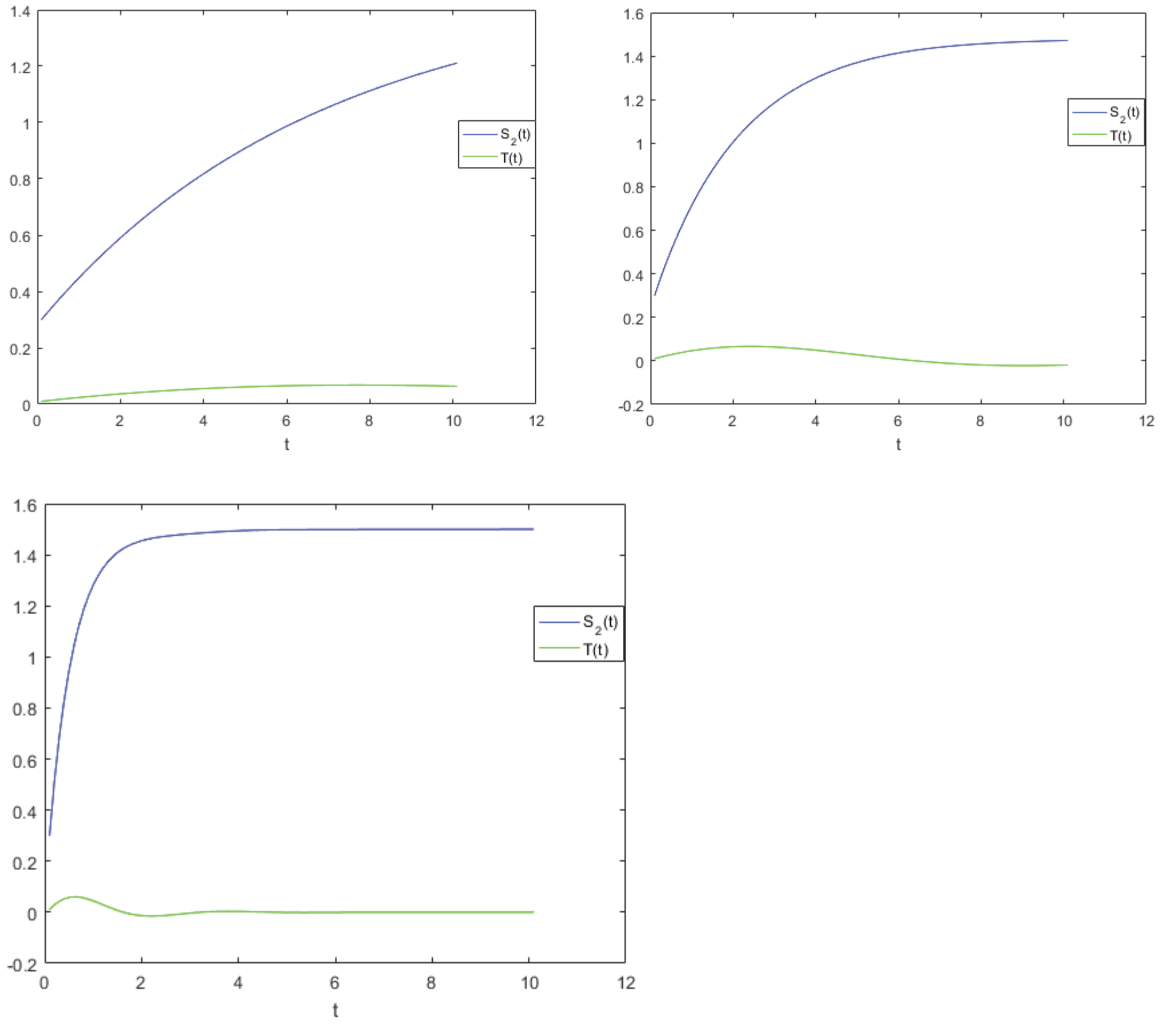

Figure 2. Estimation of Susceptible and Treatment individuals in infectious diseases using the proposed distributed SITRS model (Conditions 1 for $k_1(\alpha) = \Gamma(\alpha - 0.7)$, $k_2(\alpha) = \Gamma(\alpha + 0.7)$ and $k_3(\alpha) = \Gamma(2\alpha + 0.3)$).

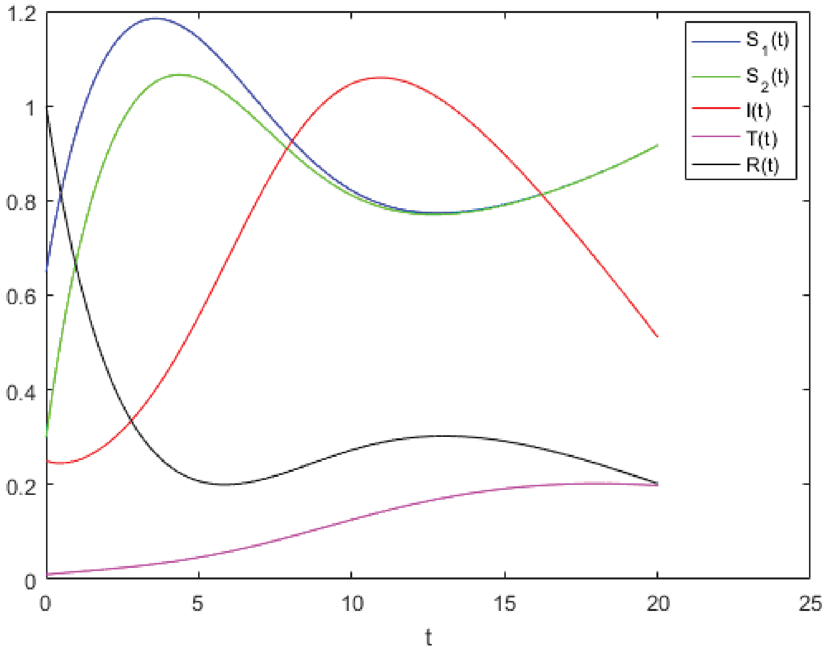

Figure 3. Time series of population variables in the SITRS model with conditions 2 for $k(\alpha) = \Gamma(\alpha - 0.1)$.

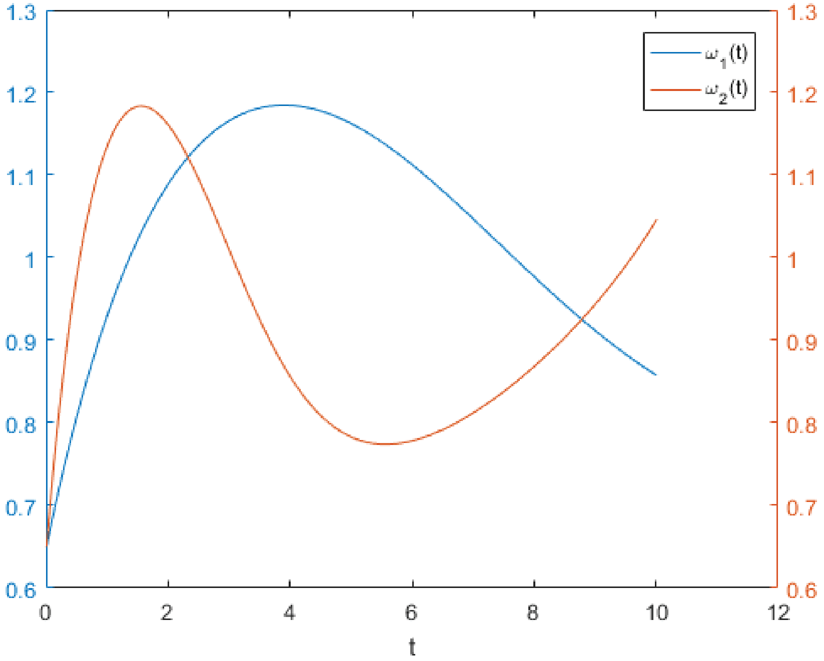

Figure 4. Prediction of behavior of susceptible individuals with varying density function [Conditions 2 for $w_1(\alpha) = \Gamma(\alpha - 0.2)$ and $w_2(\alpha) = \Gamma(\alpha - 0.8)$] .

As stated in our paper, the ability to interpret the behavior of the system largely depends on the variation in the density function. From the figures, it is clear that changes in the infection rare and recovery rates are appropriately reflected in our model equations. Moreover, the use of distributed-order modeling is precisely intended to account for such variations. In other words, changes in these factors can be accommodated through adjustments to the density function, which is dependent on the conditions. This approach allows us to select the density function based on these rates and avoid unnecessary time loss.

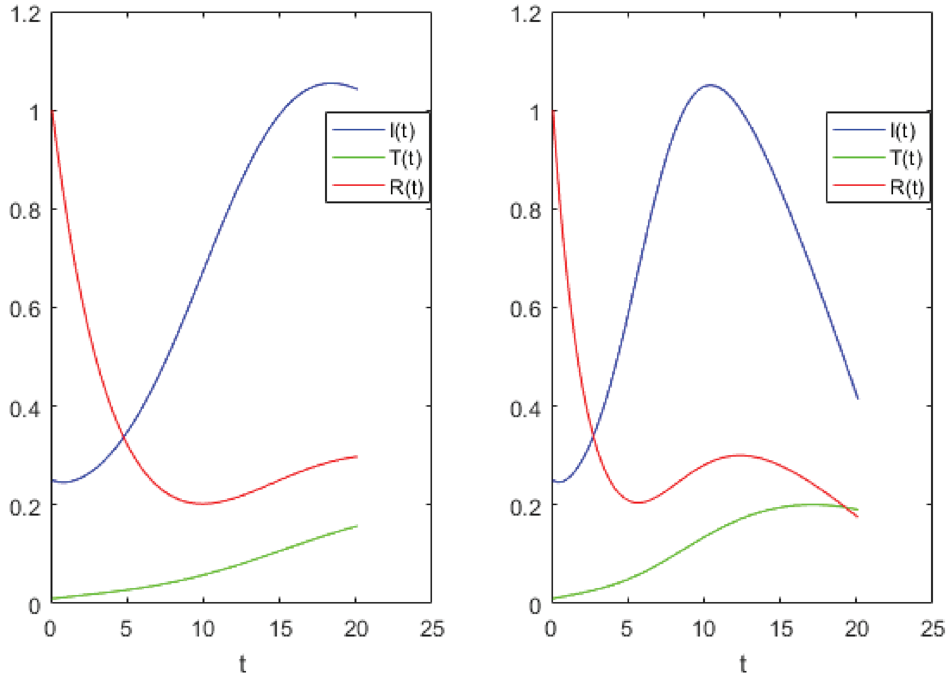

Figure 5. Numerical analysis of Infected, Treatment and Recovered individuals with conditions 2 for $k_1(\alpha) = \Gamma(\alpha - 0.5)$ and $k_2(\alpha) = \Gamma(\alpha)$.

For comparison with clinical data, the study by Elsonbaty et. al. [11] was considered. Data from this study and from COVID-NET were compared with our findings [37]. The data in question were collected in 14 US states and represent a population of approximately 32 million people living in these states. Firstly, the comparisons showed that the rate of increase of infection in the given area was similar to Figure 1. Therefore, the modified SITRS model was found to be an appropriate model. Again, when the same data were examined, it was found that the graph shown in Figure 4 best predicted the first or second wave phase of the disease. Finally, it was found that the rate of increase of infected individuals in Figure 5, expressed under the wave, was again consistent with the COVID-NET data [11,37].

CONCLUSION

In this study, the mathematical Susceptible, Infected, Treated, Recovered, Susceptible model, which is crucial for determining the dynamics of endemic models, was examined. The modified version of the model is described using distributed order differential equations. This approach allows for greater flexibility in the interpretation of different types of equation systems through the use of density functions. Equilibrium points, which are critical for the analysis of the disease, have been examined. Stability analyses of the equilibrium points were carried out and the results were found to be consistent with the data. This shows that distributed order equations are suitable for such systems. The results are compared with sources using real world data. The results are highly consistent, indicating the potential for more rapid and effective monitoring of infectious disease dynamics. Future work will focus on improving density functions and analyzing their impact across multiple scenarios, including changing population dynamics and intervention strategies. This article fills a research gap by examining the use of distributed order differential equations in public health models. It demonstrates the importance of having rapid methods for preventing and managing diseases. Our goal is to use these equations more frequently to facilitate modeling of infectious diseases and other basic science domains. This will enable us to create frameworks that are more robust and versatile.

AUTHORSHIP CONTRIBUTIONS

Authors equally contributed to this work.

DATA AVAILABILITY STATEMENT

The authors confirm that the data that supports the findings of this study are available within the article. Raw data that support the finding of this study are available from the corresponding author, upon reasonable request.

CONFLICT OF INTEREST

The author declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

ETHICS

There are no ethical issues with the publication of this manuscript.

STATEMENT ON THE USE OF ARTIFICIAL INTELLIGENCE

Artificial intelligence was not used in the preparation of the article.

REFERENCES

[1] Anderson RM, May RM. Infectious diseases of humans: dynamics and control. Oxford: Oxford University Press; 1991. [CrossRef]

[2] Keeling MJ, Eames KT. Networks and epidemic models. J R Soc Interface 2005;2:295–307. [CrossRef]

[3] House T, Keeling MJ. Household structure and infectious disease transmission. Epidemiol Infect 2009;137:654–661. [CrossRef]

[4] Ferguson NM, Laydon D, Nedjati-Gilani G, Imai N, Ainslie K, Baguelin M, et al. Impact of non-pharmaceutical interventions (NPIs) to reduce COVID-19 mortality and healthcare demand. Imperial College COVID-19 Response Team 2020;20:77482.

[5] Kermack WO, McKendrick AG. A contribution to the mathematical theory of epidemics. Proc R Soc Lond A 1927;115:700–721. [CrossRef]

[6] Brauer F. Mathematical epidemiology: past, present, and future. Infect Dis Model 2017;2:113–127. [CrossRef]

[7] Çilli A, Ergen K, Akat E. Some mathematical models and applications used in epidemics. Sigma J Eng Nat Sci 2019;37:17–23.

[8] Uçar Y, Yağmurlu M, Başhan A. Numerical solutions and stability analysis of modified Burgers equation via modified cubic B-spline differential quadrature methods. Sigma J Eng Nat Sci 2019;37:129–142.

[9] Karakoç SBG, Bhowmik SK, Omrani K, Sucu DY. A numerical treatment for the Gilson-Pickering equation using collocation method with error estimation. Sigma J Eng Nat Sci 2023;41:875–884. [CrossRef]

[10] Özgür B, Demir A. The stability analysis of a neural field model with small delay. Sigma J Eng Nat Sci 2024;42:900–904. [CrossRef]

[11] Elsonbaty AMR, Sabir Z, Ramaswamy R, Adel W. Dynamical analysis of a novel discrete fractional SITRS model for COVID-19. Fractals 2021;29:2140035. [CrossRef]

[12] Ayoub HH, Tomy M, Chemaitelly H, Altarawneh HN, Coyle P, Tang P, et al. Estimating protection afforded by prior infection in preventing reinfection: applying the test-negative study design. Am J Epidemiol 2024;193:883–897. [CrossRef]

[13] Shah K, Sarwar M, Abdeljawad T. A comprehensive mathematical analysis of fractal-fractional order nonlinear re-infection model. Alexandria Eng J 2024;103:353–365. [CrossRef]

[14] Schuh L, Markov PV, Veliov VM, Stilianakis NI. A mathematical model for the within-host (re)infection dynamics of SARS-CoV-2. Math Biosci 2024;371:109178. [CrossRef]

[15] Salman A, Mohd MH. Sustainable COVID-19 control measures: a mathematical modelling study. J Sustain Sci Manag 2024;19:25–35. [CrossRef]

[16] Caputo M. Mean fractional-order-derivatives differential equations and filters. Ann Univ Ferrara Sci Mat 1995;41:73–83. [CrossRef]

[17] Caputo M. Distributed order differential equations modelling dielectric induction and diffusion. Fract Calc Appl Anal 2001;4:421–442.

[18] Diethelm K, Ford NJ. Numerical analysis for distributed-order differential equations. J Comput Appl Math 2009;225:96–104. [CrossRef]

[19] Katsikadelis JT. Numerical solution of distributed order fractional differential equations. J Comput Phys 2014;259:11–22. [CrossRef]

[20] Morgado ML, Rebelo M. Numerical approximation of distributed order reaction-diffusion equations. J Comput Appl Math 2015;275:216–227. [CrossRef]

[21] Kocabıyık M, Ongun MY, Çetinkaya İT. Numerical analysis of distributed order SVIR model by nonstandard finite difference method. J BAUN Inst Sci Technol 2021;23:577–591. [CrossRef]

[22] Kocabiyik M, Ongun MY. Construction a distributed order smoking model and its nonstandard finite difference discretization. AIMS Math 2022;7:4636–4654. [CrossRef]

[23] Mickens RE. Nonstandard finite difference models of differential equations. Singapore: World Scientific; 1994. [CrossRef]

[24] Mickens RE. Nonstandard finite difference schemes for differential equations. J Differ Equ Appl 2002;8:823–847. [CrossRef]

[25] Ongun MY, Arslan D, Garrappa R. Nonstandard finite difference schemes for a fractional-order Brusselator system. Adv Differ Equ 2013;2013:1–13. [CrossRef]

[26] Çetinkaya İT, Kocabıyık M, Ongun MY. Stability analysis of discretized model of glucose–insulin homeostasis. Celal Bayar Univ J Sci 2021;17:369–377. [CrossRef]

[27] Kocabıyık M. Nonstandard discretization and stability analysis of a novel type malaria-Ross model. J Inst Sci Technol 2022;12:1023–1033. [CrossRef]

[28] Melkamu B, Mebrate B. A fractional model for the dynamics of smoking tobacco using Caputo–Fabrizio derivative. J Appl Math 2022;2022:2009910. [CrossRef]

[29] Çetinkaya İT. An application of nonstandard finite difference method to a model describing diabetes mellitus and its complications. J New Theory 2023;45:105–119. [CrossRef]

[30] Öztürk Z, Bilgil H, Sorgun S. A new application of fractional glucose-insulin model and numerical solutions. Sigma J Eng Nat Sci 2024;42:407–413. [CrossRef]

[31] Podlubny I. Fractional differential equations: an introduction to fractional derivatives, fractional differential equations, to methods of their solution and some of their applications. San Diego: Academic Press; 1999.

[32] Ding W, Patnaik S, Sidhardh S, Semperlotti F. Applications of distributed-order fractional operators: a review. Entropy 2021;23:110. [CrossRef]

[33] Liu L, Clemence DP, Mickens RE. A nonstandard finite difference scheme for contaminant transport with kinetic Langmuir sorption. Numer Methods Partial Differ Equ 2011;27:767–785. [CrossRef]

[34] Kocabiyik M, Ongun MY. Distributed order hantavirus model and its nonstandard discretizations and stability analysis. Math Methods Appl Sci 2024;1–17. [CrossRef]

[35] Matignon D. Stability results for fractional differential equations with applications to control processing. In: Computational engineering in systems applications. 1996;2:963–968.

[36] Widayati R, Muntoha RH, Ma'rifah U, Yulianti N. Analisa kestabilan bebas penyakit model SITRS pada penyebaran DIARE. Diophantine J Math Its Appl 2024;3:52–63. [CrossRef]

[37] Centers for Disease Control and Prevention. COVID-NET interactive database. Available at: https://www.cdc.gov/covid/php/covid-net/index.html Accessed on Nov 05, 2025.