**Research Article****Deterministic and fractional-order modeling of influenza transmission with control law**

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ABSTRACT

Standard integer-order models struggle to capture the complex dynamics of influenza spread. These steps are affected by memories and treatments. The purpose of this study is to develop and evaluate deterministic and fractional-order model of influenza spread to improve control assessment and epidemiological accuracy. The main goal is to examine how memory-based dynamics affect disease progression, stability traits, and the effectiveness of control methods. A detailed study is conducted on a deterministic compartmental influenza model to determine whether it is positive, limited, in equilibrium, and to determine the basic reproduction number. The model is then expanded into a framework that employs a difference operator similar to Caputo's and allows for fractional-order discrete-time to account for memory effects. We do local and global stability assessments, examine the sensitivity of the reproduction number, and employ forward bifurcation analysis and Lyapunov exponents to investigate nonlinear dynamics. A feedback treatment control approach was created to help decrease the effects of epidemics. Using real influenza data to test numerical models, we find that fractional-order dynamics provide a clearer picture of how the virus spreads, revealing more complex patterns such as chaotic oscillations and delayed responses. One approach to show this is to give a more in-depth look at how transmission patterns work. The results indicate that fractional-order modeling provides superior forecasts and supports more effective influenza management strategies than traditional deterministic approaches.

Introduction

Memory effects play a critical role in the transmission of epidemics, but conventional integer-order formulations fail to take them into consideration, despite the importance of compartmental models in the field of epidemiology. This study presents a discrete-time Suspected-Infected-Treated-Recovered (SITR) model of fractional order that utilizes a difference operator akin to Caputo's. The authors of this study were inspired by the work of (Haneche and Hamaizia, 2024). As noted in (Yu et al., 2024; Naik et al., 2020), treatment and

memory effects enable the simulation of complex dynamics such as bifurcations and chaos, thereby making the simulation more biologically realistic. The model is based on discrete fractional calculus theory (Atici and Eloe, 2009; Anastassiou, 2010), which considers positivity, boundedness, stability of the equilibrium, and the basic reproduction number (R_0). The model is validated with empirical influenza data from Saudi Arabia and analyzed for chaotic dynamics by Lyapunov exponents.

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Furthermore, the smoking dynamics of the fractional order are examined utilizing the Caputo-Fabrizio operators (Abaid et al., 2022). Fragmental calculus has recently been shown to be an effective method for defining intricate dynamical systems. Fractional-order epidemiological methods based on generalized Hattaf derivatives have been used to model the COVID-19 epidemic in Saudi Arabia. The results of these models demonstrate the effectiveness of prophylactic interventions and indicate that smaller fractional orders reduce disease transmission (Ahmad et al., 2024). Fractional models for COVID-19, TB, monkeypox, and hepatitis B provide superior stability, data fitting, and control methodologies compared to classical models (Albalawi et al., 2023; Aliasghari et al., 2022; Alsubaie et al., 2024; Anjam et al., 2022; Anjam et al., 2024; Ashurov and Umarov, 2020). It can use numerical methods like fractional Euler, Adams-Bashforth, and nonstandard schemes to check these dynamics. Additionally, modeling cancer, chaotic systems, fluid dynamics, and diabetes using fractional-order methodologies has demonstrated a significant dependence on memory effects and derivative order (Asma et al., 2022; Azeem et al., 2023). These studies indicate that fractional-order modeling is adaptable and precise across various fields, including biology, physics, and epidemiology.

Fractional-order models have been widely used to enhance understanding of complex biological and physical processes. A Caputo fractional SEIR-SEI model (Baba et al., 2023) makes it easier to fit Zika virus data and figure out parameters. This is due to memory effects. Combining fractional calculus with artificial neural networks can make it easier to find and detect cancer (Badshah and Akbar, 2021). Compared with conventional models, fractional models offer improved stability analysis and control strategies for a wide range of infectious diseases, including dengue fever, HFMD, and others (Basti et al., 2021; Batiha et al., 2022; Butt et al., 2023; Chouhan et al., 2024). Fractional operators are used to model phenomena in practical mathematics, including diffusion, time delays, and memory-

dependent processes. Analytical and inverse methods are used to make sure that models of real systems are as accurate as possible (Cui et al., 2022; Ding and Wang, 2022; Elbadri et al., 2023). Many different fields of science and technology have found success with fractional-order models. Scientists studying cancer can learn more about disease control by using fractional models that combine stem cells and chemotherapy. In addition, there is abundant evidence that these solutions are positive, present, and distinctive (Georgiev, 2023). As engineering tools, fractional controllers outperform conventional controllers at capturing complex behaviors, thereby improving the performance of electric vehicle induction motors and air-bearing dynamics (Hasan et al., 2023; Idrees et al., 2023).

The ever-changing dynamics of air bearings allow us to do this. Fractional adaptive control frameworks not only guarantee system stability, but they also reduce computational effort (Ilten, 2023). Compared to integer-order models, fractional models offer greater realism, stability, and data-fitting performance for TB, influenza, and juvenile diseases in epidemiology. In addition, there is a plethora of effective dynamics and control methods in fractional hyper chaotic systems (Iqbal et al., 2023; Karaca, 2023; Khennaoui et al., 2023; Liu et al., 2022). The work uses fractional calculus to compare and contrast the time evolution of models with integer-order parameters with those with fractional-order parameters, simulating the formation of vortices around a rotating rigid body. Analytical solutions demonstrate that anomalous diffusion influences the initial vortice dynamics, and fractional models eventually become classical across different time regimes (Maayah et al., 2022). The paper develops a model for TB propagation using fractional-order delay differentials. This model accounts for the effects of therapy, re-infection, and latent periods simultaneously. Computer simulations, parameter recovery, and qualitative studies have all demonstrated the model's use in predicting disease spread and guiding control actions (Meetei et al., 2024). Even when working with actual, noisy data,

this holds. The purpose of this study is to use a fractal-fractional mathematical model to investigate diagnostic and therapeutic options for lung cancer, including IL-2 cytokine and anti-PD-L1 inhibitors, in people with suppressed immune responses. We present the concepts of stability, positivity, and boundedness, and use Mittag-Leffler kernel simulations to demonstrate how an improved immune response might prevent disease transmission in various fractal dimensions (Mohandoss et al., 2024). To better comprehend the intricate fractal dynamics of tumor growth and immune response, this research will present a breast cancer model based on the Caputo-Fabrizio fractal-fractional derivative. In addition to giving information about tumor proliferation, the model provides a framework for evaluating and developing more effective treatment regimens (Mu et al., 2022). The study uses the Caputo-Fabrizio fractional differential operator to create a model of hepatitis B virus (HBV) transmission that accounts for immunization and asymptomatic carriers. In addition to confirming existence, uniqueness, positivity, and boundedness using numerical simulations with the Mittag-Leffler function, these results show that the fractional-order model better captures actual disease dynamics than the integer-order model (Prasad et al., 2022). The work presents a new corona virus model (2019-nCov). To reflect practical dynamics, the model incorporates a new parameter and piecewise-hybrid fractional-order derivatives. This study examines positivity, boundedness, and existence by comparing and contrasting real data from Spain and Wuhan to assess the model's accuracy (Raezah et al., 2023). The work develops a fractional-order virus epidemic model that includes immunization and asymptomatic infection using the Atangana-Baleanu Caputo operator. Analysis of stability, existence, and sensitivity all attest to the model's ability to capture complicated disease processes using fractional calculus (Rosa and Ndaïrou, 2024). Least squares parameter estimates and computational simulations support this research.

The technique is subsequently utilized in a respiratory syncytial virus (RSV) infection model. The fde12 solver, the fractional Euler method, and the PECE method, all created by Garrapa, are used to obtain numerical solutions. Also, open-source versions are available so that more people can use those (Rosa and Torres, 2023). The research presents a fractional-order NTIE breast cancer model that integrates immune-chemotherapeutic effects. Stochastic Levenberg–Marquardt back propagation neural networks (LMBNNs) are used to solve the model. Numerical results, tested against reference solutions (Sabir et al., 2022, show that the model is accurate, stable, and effective at capturing tumor-immune dynamics during treatment. The study uses nonlinear integro-differential equations to extend a tumor growth model to the fractional-order domain. This enables the model to encompass the dynamics of both cancer stem cells and non-stem cancer cells. Analytical considerations and numerical simulations using fixed-point theory have shown that fractional dynamics affect tumor growth and support the idea that tumor growth is paradoxical (Sene, 2020). The study extends a fractional-order Ebola model by applying Caputo derivatives. A fractional optimal control problem is also formulated, using vaccination as the control mechanism.

A variable-order fractional model that alternates between fractional and classical derivatives is the most effective approach for managing the disease's dynamics (Subramanian et al., 2024). This is evidenced by data fitting and cost-effectiveness analysis. This work presents a fractional-order COVID-19 model utilizing Caputo derivatives to assess the impacts of immunization. The Banach contraction principle is employed to establish the existence and uniqueness of the model. The model's predictive skills and the effect of fractional-order fluctuations on disease dynamics have been demonstrated through numerical simulations tested against real data from Thailand (Sun et al., 2023). The study integrates the Hadamard and Caputo frameworks by examining a modified fractional-order SIRD model for COVID-19. The model is

founded on the Caputo–Katugampola derivative. The application of Schauder's and Banach's fixed-point theorems facilitates the analysis of stability, the existence, and uniqueness of solutions. Furthermore, vaccine effects are incorporated into the susceptible population (Sweilam et al., 2022). The objective of this study is to analyze the stability of a fractional-order SEIR model for malaria in Khyber Pakhtunkhwa, Pakistan, and identify the conditions for epidemic or endemic progression. The next-generation methodology is used to estimate the basic reproduction number, and the Routh-Hurwitz criterion is used to assess the method's stability, with numerical simulations supporting the results (Sweilam et al., 2024). This study examines the stability of a fractional-order Mathieu equation under forced excitation by modeling a pantograph–catenary system to enhance comprehension of its behavior. The findings of both analytical and numerical simulations (Tang et al., 2022) show that forced excitation affect the expressions of periodic solutions. However, the damping coefficient and fractional-order parameters have a big effect on stability borders and resonance behavior.

The research introduces a fractional SEQIR epidemic model that integrates saturated incidence and vaccination. It also sets stability standards and the best ways to govern, based on fractional differential equations. Both analytical and numerical evaluations show that a low level of background noise helps maintain stochastic stability, while optimal control can successfully address the persistence of sickness (Tarasov and Tarasova, 2020). The work used the reduced differential transform method (RDTM) to address two fractional-order cancer tumor models that incorporate chemotherapy effects and variable mortality rates. Analytical and graphical data (Turkyilmazoglu and Alofi, 2024) show that the method effectively demonstrates how chemotherapy affects tumor shrinkage and immune system strengthening. This study uses the Caputo fractional derivative and a multistep Laplace-optimal decomposition method to address a fractional COVID-19 problem. The suggested method is very

accurate, efficient, and effective at capturing disease dynamics across a range of fractional orders, as demonstrated by numerical comparisons with the Runge-Kutta method (Ullah et al., 2023). The paper builds a fractional-order leprosy model using the Caputo derivative. When the fundamental reproduction number (R_0) is applied, the model exhibits positive features and stability. The development of a non-standard finite-difference scheme is complete, and simulations have validated the model's analytical stability and the reliability of the numerical method (Vieira et al., 2023). Using the Caputo-Fabrizio derivative, we introduce and investigate the existence, uniqueness, positivity, boundedness, and stability of an SEIAPHR fractional COVID-19 model. The Adams-Bashforth approach's numerical simulations, which focus on quarantine measures, a suggested optimum control problem, showcase the model's efficacy in lowering disease transmission (Yan et al., 2022). The study uses fixed-point theory to generate a nonlinear HIV transmission model that integrates fractal-fractional Caputo derivatives. It also shows the existence, uniqueness, and Ulam-Hyers stability. Using the fractional Adams–Bashforth method, numerical simulations show that stability trends lead to disease-free equilibrium. These patterns underscore the importance of transmission control techniques to enhance recovery outcomes (Yavuz et al., 2023). The research employs the Caputo–Fabrizio fractional-derivative framework to examine a deterministic Zika virus model that incorporates both vector and sexual transmission dynamics. The analysis's results show that the model has solutions and that they are unique and symmetrical. This helps us understand better how the model's transmission and control techniques work (Yavuz et al., 2022; Cermák et al., 2015).

To develop a fractional-order TB model, an Atangana-Baleanu-Caputo derivative is utilized, and nonlinear techniques are applied to ascertain solutions within the stability framework. The fractional Adams-Bashforth method (Rahman et al., 2021) is used to get numerical values for a few

orders. This is where the fractional Caputo TB model is created. Stability, reproduction numbers, memory effects, and sensitivity are among the factors considered. You can also get plans for alternative diets and therapies. The study indicated that the transmission rate increased with each new reproduction. Coordinated efforts appear to mitigate the dissemination of the disease, while the lack of cures seems to exacerbate its prevalence (Mumbu et al., 2025). Because tuberculosis is still a fatal and contagious disease that affects the lungs and other important organs, it is very important to have strong prevention and treatment plans. A fractional-order model based on modified Laguerre polynomials is used to improve the solution of nonlinear algebraic systems and to examine dynamics (Avazzadeh et al., 2023). A novel fractional tuberculosis model incorporating vaccination, reactivation, and reinfection uses the next-generation matrix method to assess transmission dynamics and estimate reproduction numbers. Researchers discovered that immunization reduces the incidence of infections; however, reinfection and reactivation increase it (Nandi et al., 2024). The novel fractional multistrain TB model with time-delay memory also uses the Grunwald-Letnikov derivative and new parameters. By integrating stability analysis with an innovative finite-difference approach, we can demonstrate that fractional-delay differential equations admit viable numerical solutions (Sweilam et al., 2017). Tuberculosis is still a deadly, contagious illness, even after millions of cases and deaths. Delays in getting medical attention and making diagnoses make the situation worse. According to an analysis using the fractional-order model and the Adams-Bashforth technique (Agbata et al., 2025), therapy accelerates recovery and reduces transmission. A fractional stochastic model employing the Atangana-Baleanu-Caputo derivative and Brownian motion analysis is utilized to examine the dynamics and memory effects of drug-resistant tuberculosis. Studies have shown that combining therapies is more effective than taking each one alone. Also, raising the noise level

might worsen the disruptions and speed up the process of eliminating the condition (Jiang et al., 2025). The dynamics of COVID-19 and tuberculosis are analyzed by incorporating probability density functions and white noise into a fractional co-infection model. The findings demonstrate that random disturbances affect stability, despite the deterministic-probabilistic method improving both knowledge and control strategies (Rashid et al., 2024). The fractal fractional tuberculosis model examines the airborne transmission of the disease by categorizing the population into susceptible, drug-sensitive, multidrug-resistant, isolated, and recovered groups. Using the Adams-Bashforth-Moulton technique, you can find numerical solutions, equilibrium, the reproduction number, and mathematical features (Khan et al., 2024). To examine transmission kinetics and reproduction rates, the fractional Caputo tuberculosis model incorporates the influences of vaccination, reinfection, and memory. On the other hand, the best control techniques aim to reduce illness while simultaneously advocating for cost-effective interventions (Rashid et al., 2022).

The scientists developed a Caputo fractional-order model for HIV-HCV co-infection, analyzed equilibrium points and reproduction numbers, demonstrated stability, and showed that memory effects affect disease dynamics, as evidenced by numerical simulations (Naik et al., 2024). The study the existence, uniqueness, and stability of a fractional HIV-HCV co-infection model with conventional and modified ABC derivatives over two time periods, and we simulate the disease dynamics using numerical data (Saleem et al., 2025). The fuzzy HIV-HCV co-infection model can account for uncertainty, compute fuzzy basic reproduction numbers and fuzzy equilibria, and use the NSFD technique with convergence to yield accurate, realistic numerical simulations (Dayan et al., 2023). In this study, we provide a model for the dynamics of HIV and HCV co-infection, including treatment, awareness, vertical transmission, and prevention. Studies indicate HIV

exacerbates HCV progression and impairs efforts to transmit and control the disease (Abiodun et al., 2022).

Fractional calculus and optimal control models of cholera indicate that medication and vaccines are effective in reducing the risk of infection. The model describes the dynamics of transmission and guarantees positivity and boundedness, all of which are needed for effective public health intervention (Gbodogbe, 2025). Getting vaccinated early is an excellent way to reduce disease and death across the whole population. Proposed numerical technique. Development of a Caputo fractional cholera model and kernels comparison with Yemeni data. The results prove this (Loyinmi et al., 2021). To demonstrate cholera's resistance to antibiotics, two different drugs are used. The results support the need for more effective therapies and suggest that antibiotic medication and second-line therapy can lower the number of cases of multidrug-resistant cholera (Mushanyu et al., 2024). The researchers have considered stability, early detection, treatment with and without medication, and statistical significance (R_0) using a fractional SEIBR cholera model. The simulations have shown that control measures and increased immune system function can successfully reduce the spread of disease (Ahmad et al. 2024).

Motivated by the real-world complexity of disease, classical influenza models are widely used but fail to capture memory and delayed effects. The article shows that flu dynamics are nonlinear, parameter-sensitive, and change over time in response to therapy. These properties can only be captured well enough by integer-order models. As a result, more and more people are using fractional-order formulations. These formulations naturally incorporate memory effects, making epidemiological models more realistic. Despite the wealth of literature on fractional epidemic models, specific questions remain unanswered. In particular, deterministic and fractional-order frameworks are not usually combined in a single influenza model. This is not something that

happens very often. Also, treatment dynamics and control methods are not frequently included in discrete-time fractional models. Finally, in the context of influenza transmission, the effects of fractional order on bifurcation behavior, stability transitions, chaotic dynamics, and related phenomena remain poorly understood. Most of the time, the current models are based on assumptions that lack validation against real data.

Despite extensive research on fractional-order epidemic models, significant gaps remain in the modeling of influenza. There are instances when current models do not integrate all key characteristics, memory effects, treatment dynamics, bifurcation analysis, chaotic behavior and control strategies into a coherent whole. Moreover, few studies have validated fractional discrete-time influenza models with real epidemiological data. In this work, we propose a deterministic fractional-order SITS influenza model with treatment and control supported by stability analysis and real-data validation. The main aim of this study is to address the deficiencies pointed out.

The present study makes a novel contribution by introducing a deterministic, fractional-order discrete-time influenza model. This model integrates treatment and recovery processes with a difference operator akin to Caputo's. This study also includes a complete analysis of positivity and boundedness, a clear derivation and sensitivity analysis of the basic reproduction number, an evaluation of chaotic dynamics using Lyapunov exponents, and the creation of a nonlinear feedback control method. The model is also tested against real-world flu data, which makes it more useful. The suggested framework can include more complex nonlinear dynamics, memory-driven transmission effects, delayed treatment responses, and fractional epidemics than current standard integer-order and fractional models. When it comes to fitting real data, the fractional-order model is more flexible and has properties (such as bifurcation and chaos) that classical models don't. Combining control techniques

with a fractional discrete-time framework can also help you develop a more valuable and effective instrument for judging public health actions. For this reason, the model is superior for theoretical studies and for real-world plans to control the virus.

The Sitr framework was chosen because our primary goal is to study the effects of treatment dynamics, recovery processes, and memory-dependent transmission on the spread of influenza within a fractional-order framework. The success of control efforts in all three stages, i.e. transition from infected persons, treatment intervention and recovery, depends on implementation of the proposed model with special focus on each. In our research, the exposed (E) compartment was not included because influenza generally has a relatively short latent period. This is because our study does not account for the dynamics during the incubation period for treatment-driven control and fractional-memory effects. Furthermore, the model can employ the Sitr structure for mathematical tractability and to capture essential epidemiological elements for assessments of nonlinear control, stability, bifurcation behavior, and Lyapunov dynamics.

Deterministic mathematical approach

The population is composed of four primary groups: susceptible people (S), infected people (I), infected people receiving treatment (T), and those who have recovered (R). The arrows show possible changes between compartments and describe how recovery occurs, how the flu infection evolves, and how the treatment works.

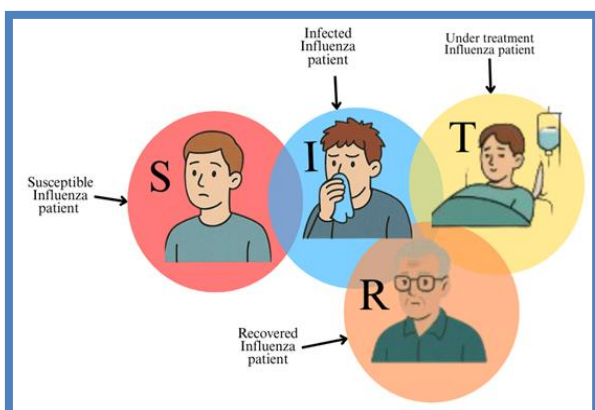


Fig. 1. Schematic diagram of the Sitr epidemic model (1).

The Fig. 2 shows how the populations in each state vary over time, with compartments for sick (I), susceptible (S), treated (T), and recovered (R). At rate μ , natural death happens in all compartments. At rate A , new people are added to the susceptible class.

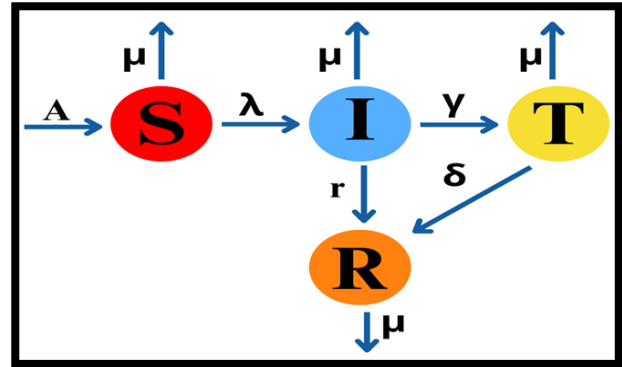


Fig. 2. Flowchart of the Sitr epidemic model (1).

The infection rate for vulnerable individuals is λ . People who are infected can either get better on their own at rate r or receive treatment at rate γ . People who have gotten treatment usually get better more quickly. The arrows show how the model's illness spreads, how it is treated, and how it gets better.

We have extended the model (1) using (Haneche et al., 2024; Yu et al., 2024). Here $S(t)$, $I(t)$, $T(t)$, and $R(t)$ represents the numbers of susceptible, infected, under treatment, recovered and total number of the individuals at the time t , respectively.

$$\begin{cases} \frac{dS}{dt} = A - \lambda SI - \mu S \\ \frac{dI}{dt} = \lambda SI - (\mu + r)I - \gamma I \\ \frac{dT}{dt} = \gamma I - (\delta + \mu)T \\ \frac{dR}{dt} = rI + \delta T - \mu R \end{cases} \quad (1)$$

It is assumed that

$$S(0) > 0, I(0) \geq 0, R(0) \geq 0, T(0) \geq 0. \quad (2)$$

In this setting, A stands for the parameter that shows the rate of population recruitment, μ stands for the constant death rate, r stands for recovery, λ stands for transmission, γ stands for the rate at which infected people move to the treatment compartment, and δ stands for the rate at which treated people recover.

The treatment compartment is included in equation (1) to account for the clinical intervention phase that many influenza patients undergo. In real life, people who have the flu don't move straight from being sick to getting better. Instead, many of them obtain antiviral drugs or medical care, which influences how long they are ill and how quickly they get better. Adding the treatment class makes this model more biologically realistic, as it can distinguish between those infected but untreated and those currently receiving treatment. Treatment also has other benefits, such as a shorter recovery period and a lower chance of spreading the disease. Quantitative indicators of this encompass the transfer rates between the infected and treated groups, as well as between the treated and recovered groups. We may use this approach to better compare different treatments based on how they affect the progression of the sickness, the stability of equilibrium points, and the basic reproduction number. So, the treatment compartment is an integral part of determining how healthy intervention strategies work and predicting more accurately how the flu will spread.

Analysis of the model

Positivity result

Each variable is nonnegative for all $t \geq 0$ because the model represents multiple individuals.

$$\text{Let, } \Omega = \{(S, I, T, R) \in R_+^4 | S + I + T + R \leq \frac{A}{\mu}\} \quad (3)$$

We must show the following result.

Theorem 1. For every $t > 0$, if all of the solutions $(S(t), I(t), T(t), R(t))$ of the system (1) with the initial values fulfill $S(t) > 0, I(t) > 0, T(t) > 0, R(t) > 0$ for all $t > 0$, then system (1) is positivity invariant and attracts inside R_+^4 .

Proof. According to the first equation of (1), we are able to represent it as

$$\begin{aligned} \frac{dS}{dt} &= A - \lambda SI - \mu S \\ \frac{dS}{dt} &= A - S(\lambda I + \mu) \\ &= A - \psi(t)S(t) \end{aligned}$$

Where $\psi(t) = \lambda I + \mu$. The following formula is then obtained by (Samui et al., 2020).

$$S(t) = S_0 \exp(-\int_0^t \psi(s) ds) + A \exp(-\int_0^t \psi(s) ds) (-\int_0^t \psi(u) du) > 0 \quad (4)$$

Similarly, $I(t) > 0, T(t) > 0, R(t) > 0$ are non negative for all t .

After performing the simulations described above, it was found that all solution trajectories of system 1 have positive values for all times $t > 0$.

Table 1. The list of compartments and the model parameter descriptions using the Influenza transmission model (1).

Symbol	Description of Compartments and parameters	Biological meaning
S	Susceptible Influenza patients	People who are suspected of having influenza
I	Infected Influenza patients	People who are sure to have influenza
T	Under treatment Influenza patients	People who are under treatment of influenza
R	Recovered Influenza patients	People who have recovered or removed due to death
A	Recruitment rate of susceptible people	The rate at which the new people enter the population
μ	Constant death rate	Constant rate at which people are dying naturally or from this disease.
r	Recovery rate	The rate at which Infected people are recovering from the disease.
λ	Transmission rate	The rate at which the disease is transmitted from one to another

γ	Treatment Admission Rate	Rate at which the individuals are moved to the treatment compartment
δ	Treatment rate	Rate at which infected individuals are treated

Boundedness

Theorem 2. Consider the initial data $S(0) \geq 0, I(0) \geq 0, T(0) \geq 0, R(0) \geq 0 \in \Omega$ and the set of the solutions $S(t), I(t), T(t), R(t)$ of the system of the equation (1) is non-negative and bounded for all $t \geq 0$

Proof: Combining the equations of system (1) yields (Ahmed et al., 2023)

$$\begin{aligned}\frac{dN}{dt} &= A - \mu(S + I + T + R) \\ \Rightarrow \frac{dN}{dt} &= A - \mu N [\because N = S + I + T + R] \\ \Rightarrow \frac{dN}{dt} + \mu N &= A\end{aligned}$$

Integrating both sides we get, $\int \frac{d}{dT} (Ne^{\mu T}) dT = \int Ae^{\mu T} dT$

$$\begin{aligned}N(t)e^{\mu T} &= \frac{A}{\mu} e^{\mu T} + C \\ N(t) &= \frac{A}{\mu} + C \cdot e^{-\mu T}\end{aligned}$$

The solution is obtained:

$$N(t) = \frac{A}{\mu} + (N(0) - \frac{A}{\mu})e^{-\mu T} \quad (6)$$

Now by evaluating the $\limsup$ as $t \rightarrow \infty$, it is obtained:

$$\lim_{t \rightarrow \infty} \sup N(t) \leq \frac{A}{\mu} \quad (7)$$

This completes the proof of the theorem.

The theorem just mentioned states that all solutions in the model are non-negative and bounded. This supports the idea that the model works in a way that makes biological sense.

Influenza disease free equilibrium point (DFE)

Finding the disease-free equilibrium point requires setting all parameters to zero, including those for the

diseased, treated, and recovered classes, as well as the equation itself.

That is $I = T = R = 0$

$$\begin{cases} A - \lambda SI - \mu S = 0 \\ \lambda SI - (\mu + r)I - \gamma I = 0 \\ \gamma I - (\delta + \mu)T = 0 \\ rI + \delta T - \mu R = 0 \end{cases} \quad (8)$$

Now, $A - \lambda SI - \mu S = 0 \Rightarrow A - \lambda S * 0 - \mu S = 0 \Rightarrow A = \mu S \Rightarrow S = A/\mu$

Again, $\lambda SI - (\mu + r)I - \gamma I = 0 \Rightarrow \lambda S * 0 - (\mu + r) * 0 - \gamma * 0 = 0 \Rightarrow 0 = 0$

$\gamma I - (\delta + \mu)T = 0 \Rightarrow \gamma * 0 - (\delta + \mu) * 0 = 0 \Rightarrow 0 = 0$

$rI + \delta T - \mu R = 0 \Rightarrow r * 0 + \delta * 0 - \mu * 0 = 0 \Rightarrow 0 = 0$

The disease-free equilibrium point can be found by putting the relevant values into the equation $E_0 = (\frac{A}{\mu}, 0, 0, 0)$

Existence of influenza endemic equilibrium point (EE) (5)

It found that the assumed, infected, treated, and recovered classes should all be zero, and we also set the total equation to zero to find the equilibrium endemic point.

The endemic equilibrium point is

Integrating factor: $e^{\int \mu dT} = e^{\mu T}$

By applying the integrating factor to both sides of equation (5), we get the following result:

$$\begin{aligned}e^{\mu T} \cdot \frac{dN}{dT} + e^{\mu T} \cdot \mu N &= e^{\mu T} \cdot A \\ \frac{d}{dT} (Ne^{\mu T}) &= Ae^{\mu T}\end{aligned}$$

$E_1 =$

$$\left(\frac{(\mu+r+\gamma)}{\lambda}, \frac{A\lambda - \mu(\mu+r+\gamma)}{\lambda(\mu+r+\gamma)}, \frac{\gamma\{A\lambda - \mu(\mu+r+\gamma)\}}{\lambda(\delta+\mu)(\mu+r+\gamma)}, \frac{\{A\lambda - \mu(\mu+r+\gamma)\}\{r(\delta+\mu) + \delta\gamma\}}{\lambda\mu(\delta+\mu)(\mu+r+\gamma)} \right)$$

Basic reproduction number

The disease classes are: $\frac{dI}{dT} = \lambda SI - (\mu + r)I - \gamma I$

$$\frac{dT}{dT} = \gamma I - (\delta + \mu)T$$

From the next generation matrix (Samui et al., 2020).

$$\text{Now, } F = \begin{bmatrix} \lambda SI \\ 0 \end{bmatrix} f(I, T) = \lambda SI$$

$$g(I, T) = 0$$

$$F = \begin{bmatrix} \frac{\partial f}{\partial I} & \frac{\partial f}{\partial T} \\ \frac{\partial g}{\partial I} & \frac{\partial g}{\partial T} \end{bmatrix}$$

$$\text{Therefore } F = \begin{bmatrix} \lambda S & 0 \\ 0 & 0 \end{bmatrix}, V = \begin{bmatrix} -(\mu + r + \gamma)I \\ \gamma I - (\delta + \mu)T \end{bmatrix}$$

$$f(I, T) = -(\mu + r + \gamma)I$$

$$g(I, T) = \gamma I - (\delta + \mu)T$$

$$V = \begin{bmatrix} \frac{\partial f}{\partial I} & \frac{\partial f}{\partial T} \\ \frac{\partial g}{\partial I} & \frac{\partial g}{\partial T} \end{bmatrix} \Rightarrow V = \begin{bmatrix} -(\mu + r + \gamma) & 0 \\ \gamma & -(\delta + \mu) \end{bmatrix}$$

Then, $V^{-1} =$

$$\frac{1}{(\mu + r + \gamma)(\delta + \mu)} \begin{bmatrix} -(\delta + \mu) & 0 \\ -\gamma & -(\mu + r + \gamma) \end{bmatrix} =$$

$$\begin{bmatrix} \frac{-(\delta + \mu)}{(\mu + r + \gamma)(\delta + \mu)} & 0 \\ \frac{-\gamma}{(\mu + r + \gamma)(\delta + \mu)} & \frac{-1}{(\delta + \mu)} \end{bmatrix}$$

$$= \begin{bmatrix} \frac{-1}{(\mu + r + \gamma)(\delta + \mu)} & 0 \\ \frac{-\gamma}{(\mu + r + \gamma)(\delta + \mu)} & \frac{-1}{(\delta + \mu)} \end{bmatrix}$$

$$FV^{-1} = \begin{bmatrix} \lambda S & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{-1}{(\mu + r + \gamma)(\delta + \mu)} & 0 \\ \frac{-\gamma}{(\mu + r + \gamma)(\delta + \mu)} & \frac{-1}{(\delta + \mu)} \end{bmatrix}$$

$$= \begin{bmatrix} \frac{-\lambda S}{(\mu + r + \gamma)} & 0 \\ 0 & 0 \end{bmatrix}$$

For finding the eigenvalues: $FV^{-1} - \lambda I =$

$$\begin{bmatrix} \frac{-\lambda S}{(\mu + r + \gamma)} & 0 \\ 0 & 0 \end{bmatrix} - \begin{bmatrix} \lambda & 0 \\ 0 & \lambda \end{bmatrix} = \begin{bmatrix} \frac{-\lambda S}{(\mu + r + \gamma)} - \lambda & 0 \\ 0 & -\lambda \end{bmatrix}$$

$$\text{Now, } |FV^{-1} - \lambda I| = 0 \Rightarrow \begin{vmatrix} \frac{-\lambda S}{(\mu + r + \gamma)} - \lambda & 0 \\ 0 & -\lambda \end{vmatrix} = 0 \Rightarrow$$

$$\frac{-\lambda S}{(\mu + r + \gamma)} = \lambda_1 \Rightarrow \lambda_1 = \left| \frac{-\lambda S}{(\mu + r + \gamma)} \right| \Rightarrow \lambda_1 = \frac{\lambda S}{(\mu + r + \gamma)}$$

$$\text{Hence the reproduction number is, } R_0 = \frac{\lambda S}{(\mu + r + \gamma)} =$$

$$\frac{\lambda A}{\mu(\mu + r + \gamma)} \text{ Suppose } S = 1 \text{ Then, } R_0 = \frac{\lambda}{(\mu + r + \gamma)} \quad (9)$$

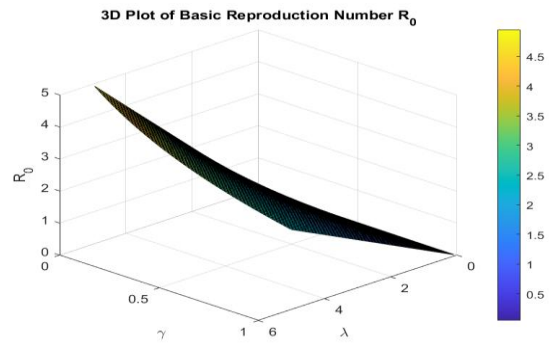


Fig. 3. 3D surface plot of the basic reproduction number R_0 as a function of the transmission rate (λ) and rate at which infected individuals move to treatment compartment (γ).

From the perspective of the three-dimensional surface in Fig. 3, the basic reproduction number R_0 is shown on the z-axis as a function of the transmission rate (x-axis) and the removal rate (y-axis). The graphic clearly shows that R_0 and γ are negatively correlated, suggesting that increasing the clearance rate reduces the disease's transmission rate. The importance of transmission intensity in the disease's persistence is emphasized when it rises, as this raises R_0 . When $\lambda = 5$ and $\gamma = 1$, R_0 approaches zero. Nevertheless, R_0 approaches zero when $\lambda = 0.1$ $\lambda = 0.1$ and $\gamma = 1$ reducing R_0 to less than one highlights the crucial significance of removal mechanisms, such as recovery and intervention efforts, in controlling the epidemic.

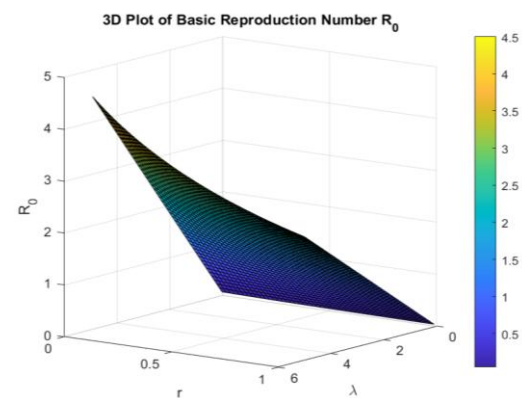


Fig. 4. 3D surface plot of the basic reproduction number R_0 as a function of the transmission rate (λ) and recovery rate (r).

The results are displayed in Fig. 4, which shows that the basic reproduction number R_0 increases with the transmission rate and decreases with the recovery rate r . The most excellent R_0 , which suggests a higher risk of disease transmission, is produced by combining high values of r with low values of r . Higher recovery rates, in contrast, lead to considerably lower R_0 even for moderate transmission. The graphic also clearly shows threshold behavior. If R_0 is greater than one, the disease is likely to persist in the community; if R_0 is less than one, the disease is expected to go away. In light of these results, it is clear that management strategies aimed at reducing transmission and improving recovery are crucial.

Analysis of local stability of DFE

Theorem 3. The disease-free equilibrium point $E_0 = (A/\mu, 0, 0, 0)$ exists and is locally asymptotically stable for $R_0 < 1$ otherwise unstable.

Proof

To find the local stability [56] of $E_0 = (A/\mu, 0, 0, 0)$ it calculates (Ahmed et al., 2023) the Jacobian matrix of the system (1) around the DFE, E_0 is given by

$$J = \begin{bmatrix} -\lambda I - \mu & -\lambda S & 0 & 0 \\ \lambda I & \lambda S - (\mu + r + \gamma) & 0 & 0 \\ 0 & \gamma & -(\delta + \mu) & 0 \\ 0 & r & \delta & -\mu \end{bmatrix} \quad (10)$$

$$J(E_0) = \begin{bmatrix} -\mu & -\frac{A\lambda}{\mu} & 0 & 0 \\ 0 & \frac{A\lambda}{\mu} - (\mu + r + \gamma) & 0 & 0 \\ 0 & \gamma & -(\delta + \mu) & 0 \\ 0 & r & \delta & -\mu \end{bmatrix}$$

The characteristic equation of $J(E_0)$ corresponding to the eigenvalues is $\det(J(E_0) - \lambda I_4) = 0$

The eigenvalues of $J(E_0)$ are

$$\lambda_1 = -\mu < 0, \lambda_2 = \frac{A\lambda}{\mu} - (\mu + r + \gamma) < 0, \lambda_3 = -(\delta + \mu) < 0, \lambda_4 = -\mu < 0$$

Here all the parameters are positive.

It has been observed that $\lambda_1, \lambda_2, \lambda_3, \lambda_4 < 0$ if $R_0 < 1$ and $\lambda_1 - \lambda_4 > 0$ if $R_0 > 1$. Hence the DFE is locally

asymptotically stable if $R_0 < 1$ and it is unstable if $R_0 > 1$.

Global stability of the Influenza disease free state

Theorem 4. The DFE point (E_0) is globally asymptotically stable (GAS) if $R_0 \leq 1$.

Proof. Prior to anything else, a Lyapunov function is considered (Dartiand Suryanto, 2020).

$$L = I - I^0 - I^0 \ln \left(\frac{I}{I^0} \right) T$$

Using system (1), the time derivative of our Lyapunov function is given by,

$$\begin{aligned} \frac{dL_1}{dt} &= \left(1 - \frac{I^0}{I}\right) \frac{dI}{dt} + \frac{dT}{dt} \\ &= \left(1 - \frac{I^0}{I}\right) \lambda SI - (\mu + r)I - \gamma I + \gamma I - (\delta + \mu)T \\ &= \frac{1}{I} (I - I^0) I \{ \lambda S - (\mu + r + \gamma) \} + \gamma I - (\delta + \mu)T \\ &= (I - I^0) \{ \lambda S - (\mu + r + \gamma) \} + \gamma I - (\delta + \mu)T \\ &= (I - I^0) \left\{ \frac{\lambda}{(\mu + r + \gamma)} S - 1 \right\} (\mu + r + \gamma) + \gamma I - (\delta + \mu)T \\ &= (I - I^0) \{ R_0 S - 1 \} (\mu + r + \gamma) + \gamma I - (\delta + \mu)T \\ &= -(I - I^0) \{ 1 - R_0 S \} (\mu + r + \gamma) + \gamma I - (\delta + \mu)T \end{aligned}$$

If $R_0 \leq 1$, then $\frac{dL_1}{dt} \leq 0$ for all $(I_p, T_p) \neq (I_p^0, 0)$. Moreover $\frac{dL_1}{dt} = 0$ if and only if $(I_p, T_p) = (I_p^0, 0)$, showing that singleton (I_p^0) is the only invariant set for which $\frac{dL_1}{dt} = 0$. By the La Salle Invariance Principle, the disease-free equilibrium I_p^0 is globally asymptotically stable for $R_0 \leq 1$.

Forward bifurcation

When a key parameter, usually the basic reproduction number R_0 , is modified in an infectious disease model, the model's behaviour changes qualitatively, causing a bifurcation. Infectious disease research uses this concept. The transition from a disease-free state to an endemic one, brought about by shifts in control or transmission characteristics, is called bifurcation. Influenza transmission is the setting in which this change takes place. The critical point at which forward bifurcation occurs is when $R_0 = 1$. This

happens when there is no longer any influenza in the population, as indicated by the stability of the disease-free equilibrium for $R_0 < 1$. A stable endemic equilibrium emerges gradually as R_0 exceeds 1, leading to a permanent influenza infection. This gradual change makes it feasible to implement effective public health initiatives to eradicate influenza as long as R_0 remains below 1. These treatments aim to lessen the spread of the disease or, if effective, speed up the healing process.

Theorem 5. The model in the system (1) exhibits forward bifurcation (Samui et al., 2020) at $R_0 = 1$, where a unique bifurcation parameter, $\lambda = \lambda^* = (\mu + r + \gamma)$ is presented.

Proof.

Let, $S = x_1, I = x_2, T = x_3$ and $R = x_4$. Then, the model in equation (1) is expressed as,

$$\begin{aligned}\frac{dx_1}{dt} &= A - \lambda x_1 x_2 - \mu x_1 \\ \frac{dx_2}{dt} &= \lambda x_1 x_2 - (\mu + r) x_2 - \gamma x_2 \\ \frac{dx_3}{dt} &= \gamma x_2 - (\delta + \mu) x_3 \\ \frac{dx_4}{dt} &= r x_2 + \delta x_3 - \mu x_4\end{aligned}$$

The transmission rate λ is taken into consideration as a bifurcation parameter so that $R_0 = 1$ if and only if $\lambda = \lambda^* = (\mu + r + \gamma)$. The linearization matrix of the matrix of the equation (10) at the equilibrium point where influenza infection is absent (E_0) is provided as follows:

$$J(E_0) = \begin{bmatrix} -\mu & -\frac{A\lambda}{\mu} & 0 & 0 \\ 0 & \frac{A\lambda}{\mu} - (\mu + r + \gamma) & 0 & 0 \\ 0 & \gamma & -(\delta + \mu) & 0 \\ 0 & r & \delta & -\mu \end{bmatrix}$$

We construct the system of equations as follows by taking into account of the system $J_w = 0$ to find the right eigenvector $w = (w_1, w_2, w_3, w_4)^T$.

$$\begin{aligned}-\mu w_1 - \frac{A\lambda}{\mu} w_2 &= 0, \frac{A\lambda}{\mu} w_2 - (\mu + r + \gamma) w_2 = 0, \\ \gamma w_2 - (\delta + \mu) w_3 &= 0, r + \delta w_3 - \mu w_4 = 0\end{aligned}$$

Solving this equation, we obtain $w_1 = -\frac{\lambda}{\mu} w_2 = 1$,

$$w_3 = \frac{\gamma}{\delta + \mu}, w_4 = \frac{r + \delta \gamma}{\mu}$$

The left eigenvector, $v = (v_1, v_2, v_3, v_4)^T$, is computed from $J_v = 0$, and we obtain,

$$v_1 = 0, v_2 = 1, v_3 = 0, v_4 = 0$$

We don't require the derivatives of f_1, f_3 , and f_4 because the third, fourth and fifth components of v are zero. Only the following derivative of f_2 are non-zero. Let us denote a and b as the bifurcation coefficients derived using the method of Castillo-Chavez-Song theorem: $a =$

$$\sum_{k,i,j=1}^6 v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0), b =$$

$$\sum_{k,i=1}^6 v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta}(E_0),$$

$$\text{Now, } \frac{\partial^2 f_2}{\partial S \partial I} = \lambda, \frac{\partial^2 f_2}{\partial I \partial S} = \lambda, a = w_1 w_2 \lambda + \lambda w_2 w_1 = 2\lambda w_1 w_2$$

$$\text{Therefore, } a = -\frac{2\lambda^2}{\mu(\mu+r+\gamma)} < 0 \text{ and } b = \frac{A}{\mu} > 0$$

Substituting the relevant derivatives and vector components, we obtain: $a < 0$ and $b > 0$.

Due to the positivity of b and the negativity of a , the model experiences a forward bifurcation. This implies that the condition $R_0 < 1$ is both necessary and sufficient for the elimination of the Influenza disease.

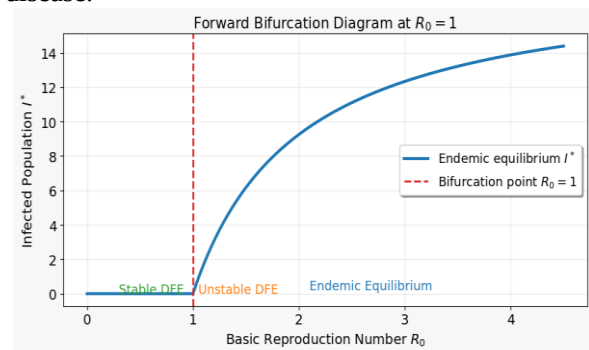


Fig. 5. Forward bifurcation diagram of the infected population I^* with respect to the basic reproduction number R_0 .

Fig.5 shows that the disease-free state is stable when $R_0 < 1$, since the diseased state remains at zero. An endemic equilibrium is reached at $R_0 = 1$, and the system's stability changes. If R_0 is greater than 1, the endemic infected population I^* grows steadily. If R_0 is approximately 4.5, it exceeds 14, indicating the infection persists beyond the threshold.

Fractional order approach

The fractional-order discrete-time SITR model builds on the standard epidemiological frameworks. To do this, the Caputo fractional difference operator is used. This operator lets us show nonlocal temporal

dynamics. In addition, fractional operators automatically account for memory and genetic effects, allowing prior epidemic states to affect present-day dynamics. It is impossible to accurately represent the complex ways diseases spread in the real world without this feature. On the other hand, the fractional order improves both the stability analysis and the system's global dynamics. Stability analysis made easier by the fractional order, while numerical efficiency and compatibility with sampled data are maintained by the discrete-time formulation. The suggested strategy (Haneche et al., 2024) provides better data fit and a more realistic picture of how infectious illnesses spread, making it easier to plan and implement effective control and intervention measures.

Mathematical preliminaries

This section provides an overview of stability results and the central notions of discrete fractional calculus, so the reader can better grasp the rest of the article. It will utilize the one-dimensional time scale $\mathbb{N}_a = \{a, a+1, a+2, \dots\}$, where $a \in \mathbb{R}$.

Definition 1. The γ^{-th} fractional sum of function $u(t): \mathbb{N}_a \rightarrow \mathbb{R}$ is defined [1] as

$$\Delta_0^{-\gamma} u(t) = \frac{1}{\Gamma(\gamma)} \sum_{s=0}^{t-\gamma} (t-s-1)^{(\gamma-1)} u(s)$$

where $\gamma > 0, t \in \mathbb{N}_{a+\gamma}$, and $\Gamma(\cdot)$ is the Gamma function. The term $t^{(\gamma)}$ is the falling function, which is expressed as

$$t^{(\gamma)} = \frac{\Gamma(t+1)}{\Gamma(t+1-\gamma)}$$

Definition 2. The Caputo-like difference operator of $u(t)$ with order γ is defined (Haneche et al., 2024) as

$$\begin{aligned} {}^c\Delta_a^\gamma u(t) &= \Delta_a^{-(1-\gamma)} \Delta u(t) \\ &= \frac{1}{\Gamma(1-\gamma)} \sum_{s=0}^{t-(1-\gamma)} (t-s-1)^{(-\gamma)} \Delta u(s), \end{aligned}$$

where $\gamma \notin \mathbb{N}$ and $t \in \mathbb{N}_{a+1-\gamma}$.

Formulation of fractional order model

By applying forward Euler method scheme to (1) we get,

$$\begin{cases} S_{m+1} = S_m + h(A - \lambda S_m I_m - \mu S_m) \\ I_{m+1} = I_m + h\{\lambda S_m I_m - (\mu + r + \gamma) I_m\} \\ T_{m+1} = T_m + h\{\gamma I_m - (\delta + \mu) T_m\} \\ R_{m+1} = R_m + h(r I_m + \delta T_m - \mu R_m) \end{cases} \quad (11)$$

where h is the step size.

A new fractional-order discrete-time Sitr model for influenza has been developed using the Caputo-like difference operator. To get an infection using (11), take these steps:

$$\begin{cases} {}^c\Delta_a^\gamma S(t) = h\{A - \mu S(t-1+\gamma_1) - \lambda S(t-1+\gamma_1) I(t-1+\gamma_1)\} \\ {}^c\Delta_a^\gamma I(t) = h\{\lambda S(t-1+\gamma_2) I(t-1+\gamma_2) - (\mu + r) I(t-1+\gamma_2) - \gamma I(t-1+\gamma_2)\} \\ {}^c\Delta_a^\gamma T(t) = h\{\gamma I(t-1+\gamma_3) - (\delta + \mu) T(t-1+\gamma_3)\} \\ {}^c\Delta_a^\gamma R(t) = h\{r I(t-1+\gamma_4) + \delta T(t-1+\gamma_4) - \mu R(t-1+\gamma_4)\} \end{cases} \quad (12)$$

Dynamics and numerical simulation

The validation process demonstrated the model's applicability in characterizing the dissemination and control of influenza epidemics, utilizing actual data from Saudi Arabia (Haneche et al., 2024).

Table 2. List of Sitr model parameters, their units, numerical values, and data sources for the influenza transmission model.

Parameter	Units	Values	Data source
A	day ⁻¹	5.55	[1]
h	day ⁻¹	0.78	[1]
λ	day ⁻¹	0.1	[1]
r	day ⁻¹	0.7	[1]
μ	day ⁻¹	0.3	[1]
γ	day ⁻¹	0.9	Assumed
δ	day ⁻¹	0.03	Assumed

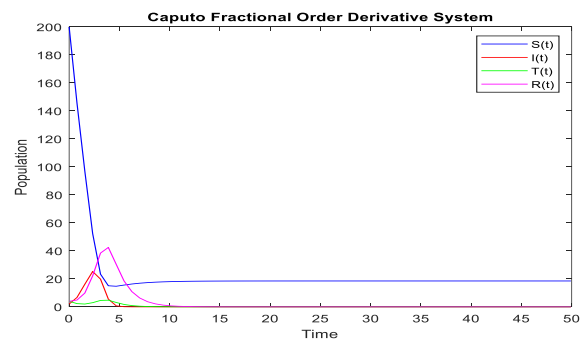


Fig. 6. Dynamics of the influenza disease model (12) using a Caputo fractional difference operator.

Fig. 6 shows that the model starts with many people who are likely to get sick, but this number quickly declines as the disease spreads. In the beginning stages of the disease, the number of infected people rises sharply. After that, it falls as people obtain treatment and get better. The intervention effects are seen in a slight rise in the number of people treated immediately after the peak of the infection. On the other hand, when treatment and natural healing start to work, people who have healed reach their peak later. Fractional-order dynamics, which include memory effects, affect the speed and smoothness of transitions between compartments. This behavior differs from traditional integer-order models, which use integer-order dynamics.

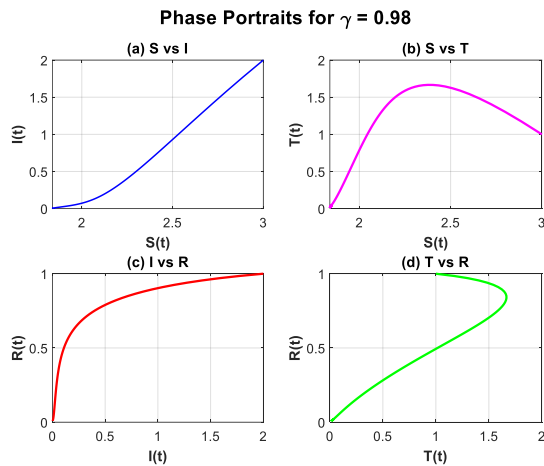


Fig. 7. Phase attractor in 2D projection for the fractional-order SITS model (12) for $\gamma = 0.9$.

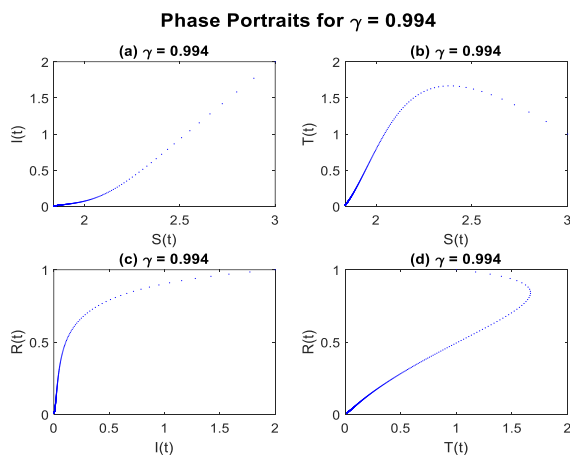


Fig. 8. Phase attractor in 2D projection for the fractional-order SITS model (12) for $\gamma = 0.994$.

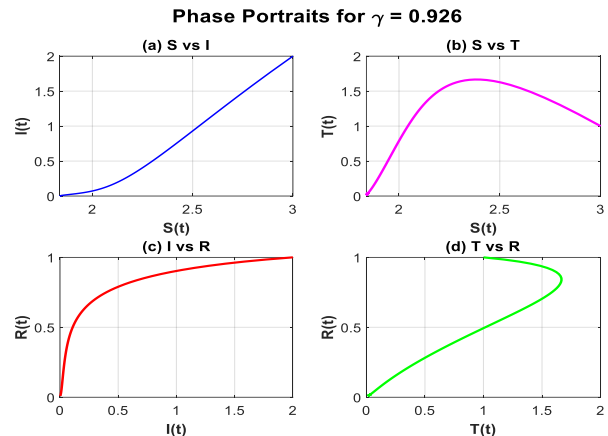


Fig. 9. Phase attractor in 2D projection for the fractional-order SITS model (12) for $\gamma = 0.926$.

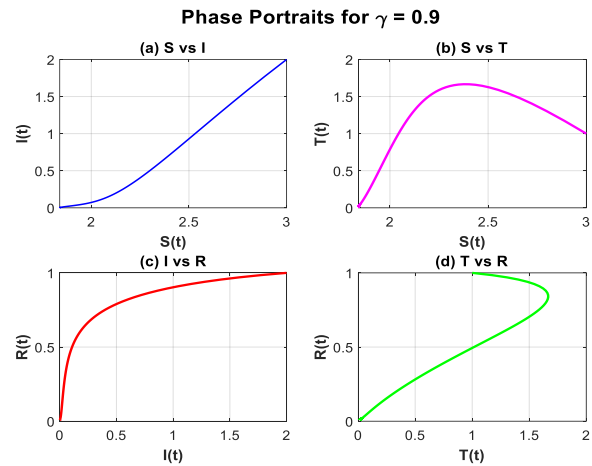


Fig. 10. Phase attractor in 2D projection for the fractional-order SITS model (12) for $\gamma = 0.98$.

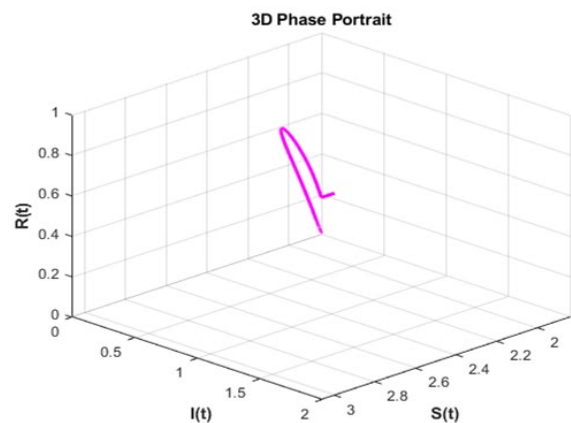


Fig. 11. Phase attractor in 3D projection for the fractional-order SITS model (12) with compartment S, I, R.

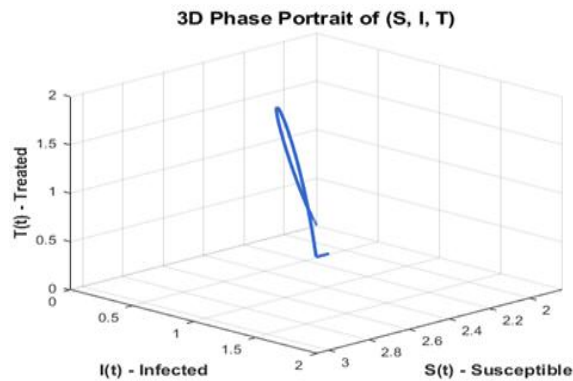


Fig. 12. Phase attractor in 3D projection for the fractional-order Sitr model (12) with compartment S, I, T.

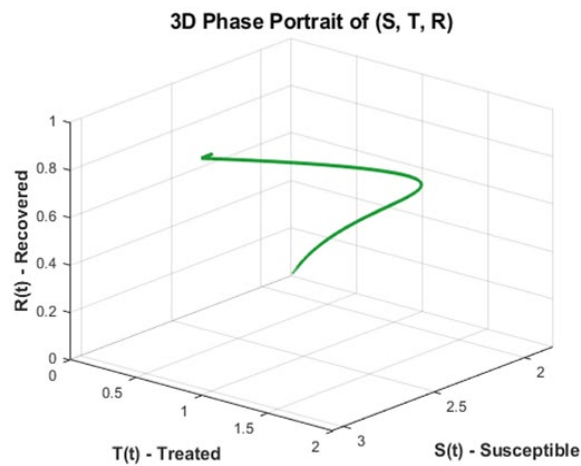


Fig. 13.Phase attractor in 3D projection for the fractional-order Sitr model (12) with compartment S, T, R.

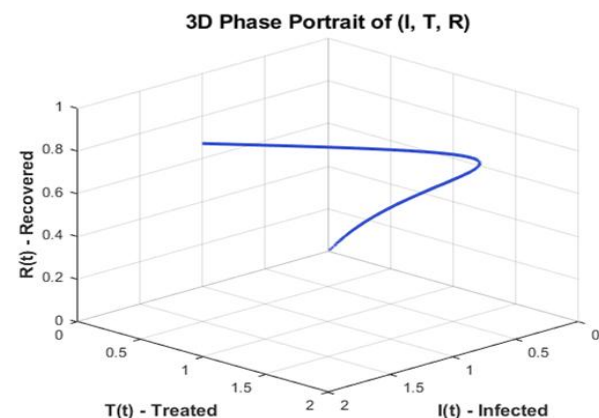


Fig. 14.Phase attractor in 3D projection for the fractional-order Sitr model (12) with compartment I, T, R.

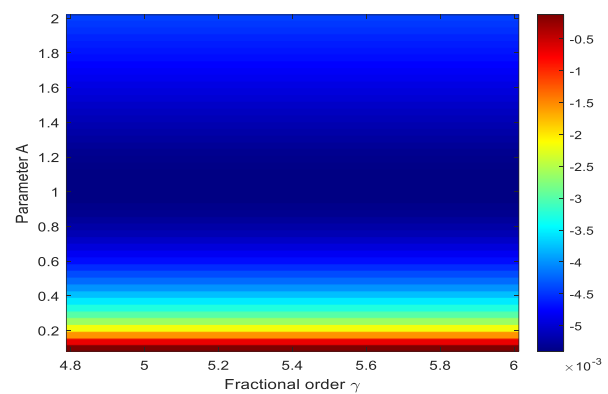


Fig. 15. Chaos diagram of the commensurate fractional-order Sitr model (12) based on the MLE γ - A plane.

Figs. 7–15 illustrate the complexity and memory dependence of the fractional-order Sitr influenza model (12). Fig. 7 demonstrates highly nonlinear interactions among several compartments. Some of these links are saturating recovery behavior, a delayed response to treatment, and a positive susceptibility-infection coupling. Fig. 8 indicates that the Caputo-like fractional operator increases the likelihood that systems will converge to equilibrium by smoothing out phase trajectories. Fig. 9 shows the phase attractors for the classical (integer-order) Sitr model. The distinctive characteristic of this model is the steady-state evolution of nonlinear compartmental interactions that lack memory effects. Fig. 10 depicts fractional memory effects, which allow us to observe actual intervention effects and population decline by adding gradual transitions and delayed responses. Figs. 11–14 present three-dimensional phase portraits that illustrate how the system progresses from the early stages of disease progression to treatment-driven control as it approaches stable equilibrium and disease elimination. During this phase, the number of sick people decreases, while the number of recovered people increases. Fig. 15 indicates that changes in recruitment rate A have a greater effect on the system's behavior than changes in the fractional order γ . This illustrates that a big part of how an epidemic behaves depends on how many new people enter.

The 0-1 test

It is used the 0-1 test from this paragraph to look at how the fractional-order epidemic model (3) changes over time. The 0-1 test can be used to determine whether a time series exhibits chaos when the input is a time

series, and the output is a real scalar, usually between 0 and 1, based on whether the system dynamics are chaotic [1].

Based on the time series $x(n)$ ($n = 1, 2, \dots, N$), where N is the length of the time sequences generated by the iteration, these are the two real-valued sequences that need to be defined [1].

$$\begin{cases} p(n) = \sum_{j=1}^n x(j) \cos(\varphi(j)) \\ q(n) = \sum_{j=1}^n x(j) \sin(\varphi(j)) \end{cases}$$

Where, $\varphi(j) = j\theta + \sum_{i=1}^n x(i)$, and $\theta \in [\frac{\pi}{5}, \frac{4\pi}{5}]$.

It can see how the translation components p and q move in the plane indicated by $(p - q)$. Because the $(p - q)$ trajectories are bounded, the system is in a periodic or convergent state. The unbounded $(p - q)$ trajectories, on

$$K = \text{median}(K_c) \quad (16)$$

When k is closer to zero, the system is more stable. When k is closer to 1, the system is more chaotic. This test is easy to apply because it doesn't require reconstructing phase space and can be applied directly to time series data. This means it works well with deterministic dynamic systems.

Fig. 16 shows the (p, q) plane, with four phase-space graphs. These charts could depict various dynamic behaviours or simulation results of a system, depending on the starting conditions, control parameters, and noise effects. Both the top-left and bottom-left plots show structured, symmetrical polygonal patterns that are centered on the origin. The motion appears to happen repeatedly, or at least closely, and occurs only in a small region.

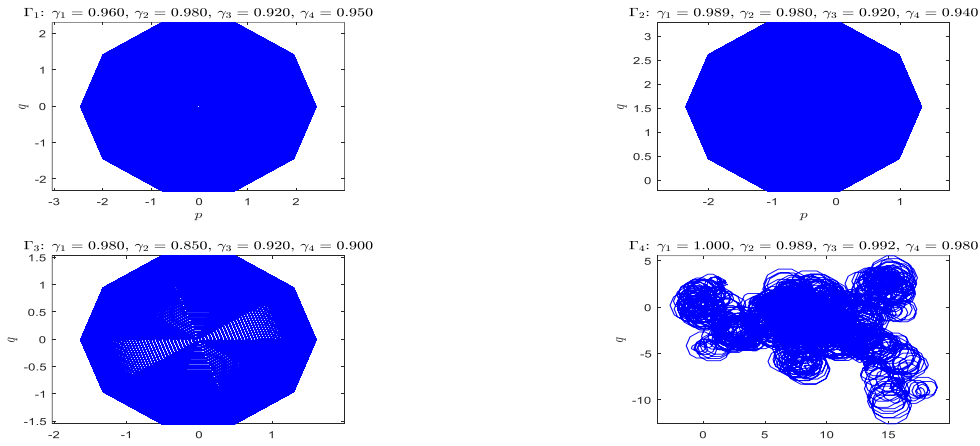


Fig. 16. The 0-1 test of the incommensurate fractional-order Sitr model (12): (Γ_1) for $(\gamma_1, \gamma_2, \gamma_3) = (0.96, 0.98, 0.92)$; (Γ_2) for $(\gamma_1, \gamma_2, \gamma_3) = (0.989, 0.98, 0.92)$; (Γ_3) for $(\gamma_1, \gamma_2, \gamma_3) = (0.98, 0.85, 0.92)$; (Γ_4) for $(\gamma_1, \gamma_2, \gamma_3) = (1, 0.989, 0.992)$.

the other hand, exhibit behavior similar to Brownian motion. It also use the following formula (Haneche et al., 2024) to compute the mean-square displacement for the sequences $p(n)$ and $q(n)$.

$$M_c(n) = \lim_{n \rightarrow \infty} \frac{1}{n} \sum_{j=1}^n [p(j+n) - p(j)]^2 + [q(j+n) - q(j)]^2, \quad (14)$$

For $n \ll N$. Hence, we state that the values of K_c for the random constant c are

$$K_c = \lim_{n \rightarrow \infty} \frac{\log M_c(n)}{\log(n)} \quad (15)$$

Finally, we define the asymptotic growth rate K as the median of all K_c values, i.e;

The presence of a solid polygon in the upper right corner of the plot indicates that the phase space is full. This could be because the oscillations overlap or because there are many paths. The figure in the lower right corner, on the other hand, shows signs of chaotic or diffusive behavior. This is because the trajectory's uneven spread around the plane indicates that the dynamics are complicated, which could mean that they are unstable or random. The Fig. 16 together depict a transition from ordered to disordered motion, helping us understand how the system responds to varied.

With the parameters set at $h = 0.78, \lambda = 0.81, r = 0.7, d = 0.3$, and $A = 5.55$, it examines the incommensurate fractional-order Sitr model (12) using the 0 – 1 test technique. For different fractional order values, the Fig. 16 shows phase graphs of the translation components p and q and the asymptotic growth rate K .

Table 3. Results of the 0-1 test for the fractional-order Sitr model (12) and (16) with different fractional order values.

$(\gamma_1, \gamma_2, \gamma_3, \gamma_4)$	K
$(0.96, 0.98, 0.92, 0.92)$	0.0034
$(0.989, 0.98, 0.92, 0.92)$	0.0064
$(0.98, 0.85, 0.92, 0.92)$	0.0031
$(1, 0.989, 0.992, 0.92)$	0.9873

system (12) exhibits periodic dynamics. On the other hand, the paths of p and q act like Brownian motion when $(\gamma_1, \gamma_2, \gamma_3, \gamma_4) = (1, 0.989, 0.992, 0.92)$. This suggests that fractional-order (12) is mainly chaotic, consistent with the bifurcation diagrams.

Fig. 17 shows a grid of six heat maps that illustrate how the model's output changes when fractional-order parameters are matched in different ways. The heat maps for $\gamma_1, \gamma_2, \gamma_3, \gamma_4$ are labeled as such. The subplots demonstrate how various parameters interact. The intensity of the colors shows how strong the reaction is. The heat maps indicate areas of high and low concentration, corresponding to parameter combinations that cause the system to behave differently. The top-left subplot (γ_1 vs. γ_2), shows a clear vertical band of low values at $\gamma_1 \approx 0.94$. This means that there is a critical threshold. You may observe similar changes in other plots, such as the horizontal band in the top-middle subplot (γ_1 vs. γ_3).

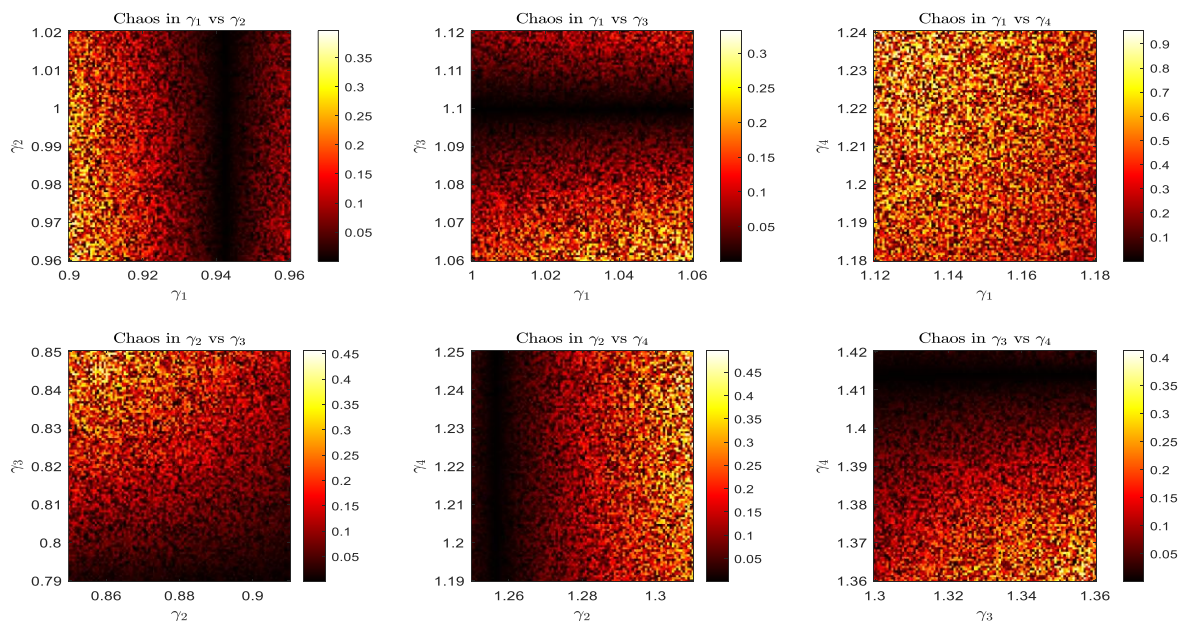


Fig. 17. Chaos diagrams based on the SE analysis for the fractional-order Sitr model (12).

Here are Table 3 and Fig. 18. When $(\gamma_1, \gamma_2, \gamma_3, \gamma_4) = (0.96, 0.98, 0.92, 0.92)$, $(\gamma_1, \gamma_2, \gamma_3, \gamma_4) = (0.989, 0.98, 0.92, 0.92)$ and $(\gamma_1, \gamma_2, \gamma_3, \gamma_4) = (0.98, 0.85, 0.92, 0.92)$, respectively, the paths of p and q are limited. Based on this, it appears that

These visualizations can help one select appropriate parameter ranges for fractional-order systems by highlighting how different fractional orders affect the model's stability and sensitivity Fig. 18 illustrates the spectral entropy (SE) complexity of the fractional-order Sitr model as a function of the fractional

parameters, which range from 0.75 to 1.0. The data indicate that when the total fractional order increases, the SE consistently rises, ranging from approximately 0.45 to roughly 0.75-0.80. This shows that the system's behavior is less predictable and its dynamical complexity is higher. When specific fractional parameters are changed, different numerical patterns show up. For example, the standard error (SE) ranges from about 0.55 to a low of about 0.30-0.35, then rises to about 0.75-0.80, indicating a U-shaped response and a shift from moderate to higher complexity. In another case, the SE stays within a narrower range of about 0.30-0.50, with a clear low near the middle, which suggests slight but not unimportant sensitivity. Second, as the parameters change, SE increases from around 0.35 to 0.40, reaches a peak at 0.80, and then decreases slightly. This example of a non-monotonic effect shows that a significant increase can make things simpler up to a point. One parameter, on the other hand, shows a declining SE trend from about

parameters significantly affect the model's overall complexity.

Theorem 6. The fractional order discrete model (12) defines the asymptotically stable disease-free equilibrium E_0 based on its subsequent requirements [1]: $\frac{\lambda A}{\mu} < \mu + r + \gamma$, $h\mu < 2^\gamma$, $\left| h\left\{ \frac{\lambda A}{\mu} - (\mu + r + \gamma) \right\} \right| < 2^\gamma$

Proof.

The Jacobian Matrix of the fractional order system (12) evaluated at the fixed point E_0 is given by

$$J(E_0) = \begin{pmatrix} -h\mu & -h\frac{\lambda A}{\mu} & 0 & 0 \\ 0 & h\left(\frac{\lambda A}{\mu} - \mu + r + \gamma\right) & 0 & 0 \\ 0 & h\gamma & -h(\delta + \mu) & 0 \\ 0 & hr & h\delta & -h\mu \end{pmatrix}$$

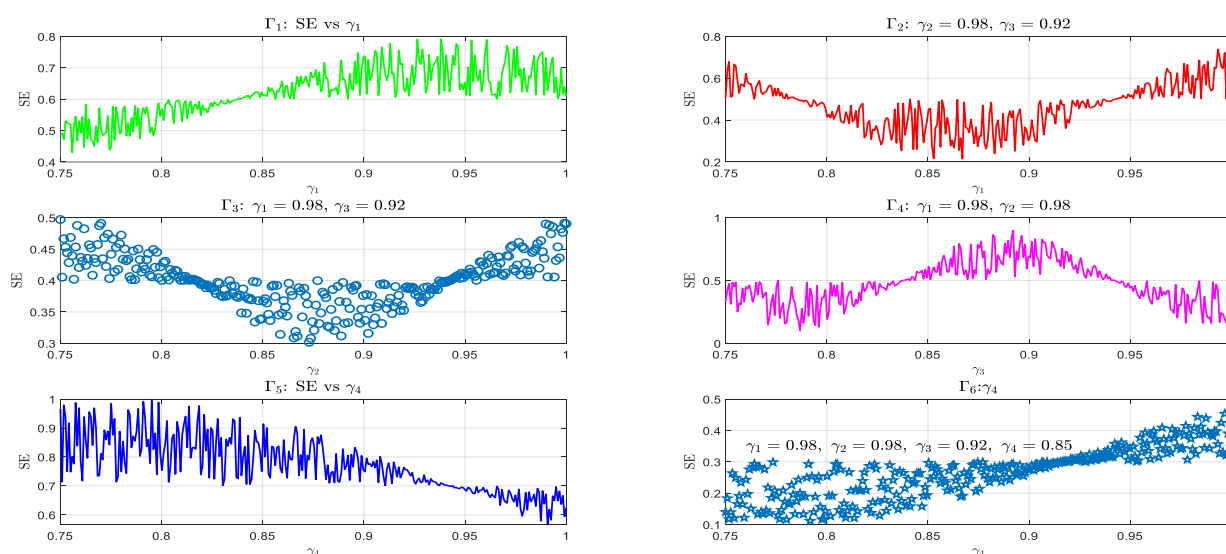


Fig. 18. SE complexity of the fractional-order SITS model (12) under variations of the fractional parameters.

0.95 to 0.65. This means that there is a stabilizing factor that stops complex oscillatory dynamics. Lastly, the standard error (SE) increases from roughly 0.15 to 0.45 as the parameters are varied, indicating that interactions among fractional

It is clear that the eigenvalues of $J(E_0)$ are: $\xi_1 = -h\mu$, $\xi_2 = h\left\{ \frac{\lambda A}{\mu} - (\mu + r + \gamma) \right\}$, $\xi_3 = -h(\delta + \mu)$, $\xi_4 = -h\mu$

One can verify that the arguments of the eigenvalues ξ_1, ξ_3, ξ_4 are π , which satisfy the condition,

$$|\arg(\xi_j)| > \frac{\gamma\pi}{2}, j = 1, 3, 4$$

The condition $|\xi_j| < 2^\gamma, h(\delta + \mu) < 2^\gamma, h\mu < 2^\gamma$

Now, the argument of the eigenvalue ξ_2 is π if $\frac{\lambda A}{\mu} < (\mu + r + \gamma)$

Hence, we have $|\arg(\xi_2)| > \frac{\gamma\pi}{2}$. Taking into consideration the second requirement [1] of Theorem 2, we can $|\xi_2| = \left| h\left\{\frac{\lambda A}{\mu} - (\mu + r + \gamma)\right\} \right| < 2^\gamma$

This completes the proof.

Theorem 7. The fractional order discrete model's endemic equilibrium E_E is considered asymptotically stable if and only if one of the two sets of requirements given below is true.

Then E_E is asymptotically stable if

$$|\xi_i| < 2^{\gamma_i}, \text{ for all } i = 1, 2, 3, 4$$

$$|\arg(\xi_2)| > \frac{\gamma_i\pi}{2}, \text{ for all } i$$

Or

If $\xi_{1,2}$ are complex conjugate eigenvalues, then E_E is asymptotically stable if:

$$|\xi_{1,2}| < (2\cos(\frac{|\arg(\xi_{1,2})| - \pi}{2 - \gamma_i}))^{\gamma_i}, |\arg(\xi_2)| >$$

$$\frac{\gamma_2\pi}{2}, |\xi_3| < 2^{\gamma_3}, |\arg(\xi_3)| > \frac{\gamma_3\pi}{2}$$

$$|\xi_4| < 2^{\gamma_4}, |\arg(\xi_4)| > \frac{\gamma_4\pi}{2}$$

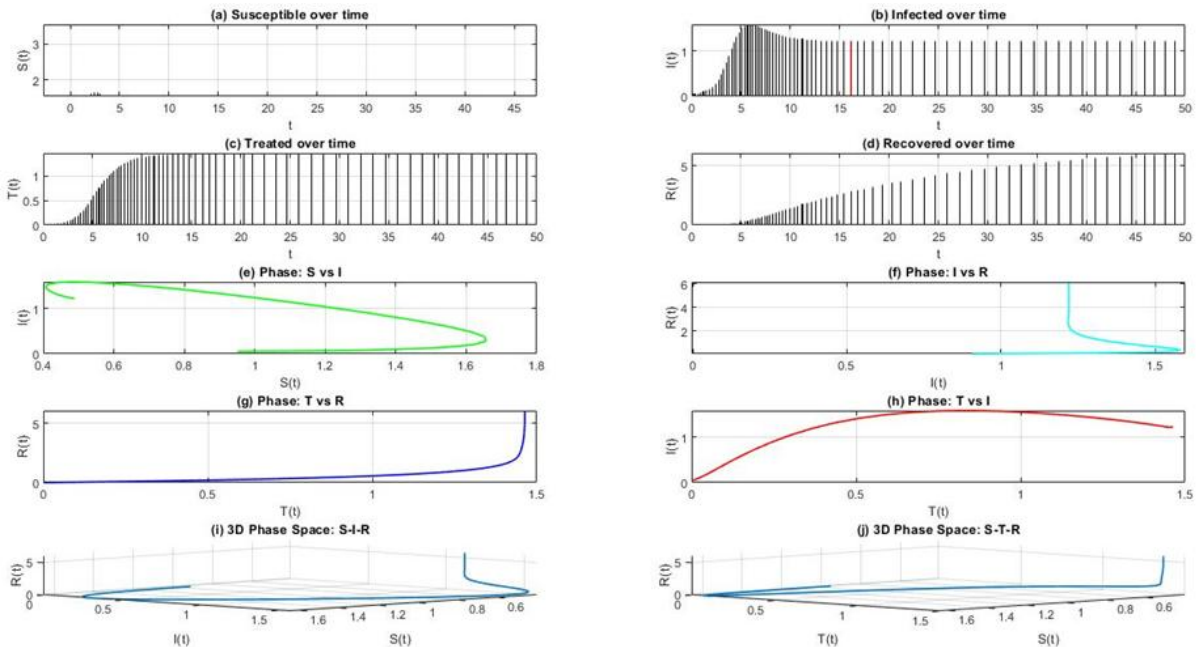


Fig. 19. The states and phase attractor of the controlled fractional-order Sitr model (12): (a) time evolution of $S(t)$ (b) Time evolution of $I(t)$ (c) Time evolution of $T(t)$ (d) Time evolution of $R(t)$ (e) Phase attractor in S - I space (f) Phase attractor in I - R space (g) Phase attractor in T - R space (h) Phase attractor in T - I space (i) Phase attractor in S - I - R space.

$$J(E_E)$$

$$= \begin{bmatrix} -AR_0 & -(\mu + r + \gamma) & 0 & 0 \\ AR_0 - \mu & 0 & 0 & 0 \\ 0 & \gamma & -(\delta + \mu) & 0 \\ 0 & r & \delta & -\mu \end{bmatrix}$$

Let $\xi_{1,2}$ be the eigenvalues of the top left 2×2 block and $\xi_3 = -(\delta + \mu), \xi_4 = -\mu$.

Proof.

Linearization is used for the system (12) near the endemic equilibrium $E_E = (S^*, I^*, T^*, R^*)$. Let:

$$x(t) = \begin{bmatrix} S(t) \\ I(t) \\ T(t) \\ R(t) \end{bmatrix}, \text{ Afterwards, the fractional-order}$$

discrete system approaching Caputo's model changes into

${}^C_0\Delta^\gamma x(t) = J(E_1)x(t)$. Specifically, $J(E_1)$ is the Jacobian matrix of the vector field evaluated at E_1 , and $\gamma = (\gamma_1, \gamma_2, \gamma_3, \gamma_4)$ is the formula that shows the fractional orders of each compartment. One way to write the Jacobian matrix using the endemic model and model equations is:

$$(E_E) = \begin{bmatrix} -AR_0 & -(\mu + r + \gamma) & 0 & 0 \\ AR_0 - \mu & 0 & 0 & 0 \\ 0 & \gamma & -(\delta + \mu) & 0 \\ 0 & r & \delta & -\mu \end{bmatrix}, \quad \text{Two}$$

separate parts can make up this.

Upper-left 2×2 block: $M = \begin{bmatrix} -AR_0 & -(\mu + r + \gamma) \\ AR_0 - \mu & 0 \end{bmatrix}$

Let, $a = -AR_0$, $b = -(\mu + r + \gamma)$, $c = AR_0 - \mu$

The characteristic polynomial of M is: $p(\theta) = \theta^2 - a\theta + (ac - bc)$

The discriminant is: $\Delta = a^2 - 4(ac - bc)$

The eigenvalues of M are: $\xi_{1,2} = \frac{a \pm \sqrt{\Delta}}{2}$

Lower right 2×2 block

This matrix is: $\begin{bmatrix} -(\delta + \mu) & 0 \\ \delta & \mu \end{bmatrix}$

Its eigenvalues are the diagonal entries: $\xi_3 = -(\delta + \mu)$, $\xi_4 = -\mu$

For linear system: ${}^C_0\Delta^\gamma x(t) = J(E_E)x(t)$. Assuming the eigenvalues ξ_i satisfy the condition of discrete fractional order stability, the equilibrium E_E is thought to be locally asymptotically stable.

$$|\arg(\xi_i)| > \frac{\gamma_i \pi}{2}, \text{ and } |\xi_i| < (2 \cos(\frac{|\arg(\xi_i)| - \pi}{2 - \gamma_i}))^{\gamma_i}$$

This applies to: $\xi_3 = -(\delta + \mu)$, $\xi_4 = -\mu$. Possibly ξ_1, ξ_2 if $\Delta \geq 0$ and $\sqrt{\Delta} \in \mathbb{R}$

For these eigenvalues: $\arg(\xi_i) = \pi \Rightarrow |\arg(\xi_i)| > \frac{\gamma_i \pi}{2}$ is always satisfied for $\gamma_i \in (0, 1)$

The magnitude condition reduces to: $|\xi_i| < 2^{\gamma_i}$

In this case, $\xi_{1,2} \in \mathbb{C}$ are complex conjugates.

Let $\xi_{1,2} = \rho e^{\pm i\theta}$. Then the stability conditions are: $|\arg(\xi_{1,2})| = |\theta| > \frac{\gamma_i \pi}{2}$ and $|\xi_{1,2}| = \rho < (2 \cos(\frac{|\theta| - \pi}{2 - \gamma_i}))^{\gamma_i}$

If all eigenvalues ξ_i of the jacobian matrix $J(E_E)$, expressed in terms of the basic reproduction number $R_0 = \frac{\lambda}{(\mu + r + \gamma)}$, satisfy: $|\arg(\xi_i)| > \frac{\gamma_i \pi}{2}$ and $|\xi_i| < (2 \cos(\frac{|\arg(\xi_i)| - \pi}{2 - \gamma_i}))^{\gamma_i}$.

At present, the endemic equilibrium point E_E of the fractional-order discrete-time SITS model is locally asymptotically stable. About the scenario's real and negative eigenvalues:

$$|\xi_i| < 2^{\gamma_i}, |\arg(\xi_i)| = \pi > \frac{\gamma_3 \pi}{2}.$$

Fig. 19 displays the dynamic behavior of a fractional-order discrete-time SITS epidemic model, illustrated using time-series and phase-space graphics. Subplots (a)–(d) demonstrate how the populations of susceptible $S(t)$, infected $I(t)$, treated $T(t)$, and recovered $R(t)$ change over time in fifty steps. The patterns show damped oscillations and, after a while, they settle into stable states. This means that the system is stable. Subplots (e)–(h) show two-dimensional phase diagrams of how compartments interact, such as S vs I and T vs R . The intrinsic equilibrium is asymptotically stable due to the closed or converging trajectories of these subplots. Lastly, (i) was used to display the three-dimensional phase space (S, I, R) , and (j) was used to show the three-dimensional phase space (S, T, R) . These subplots demonstrate how the system behaves globally and how it reaches a stable state in a multi-dimensional space.

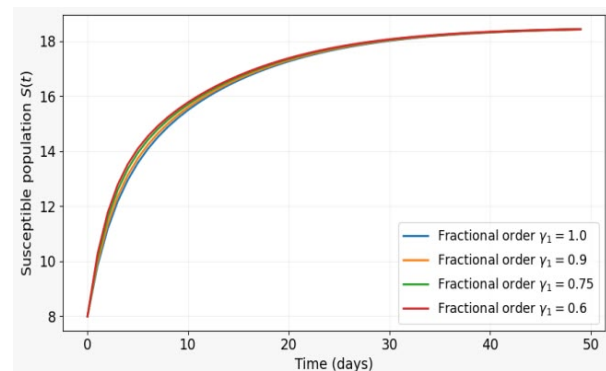


Fig. 20. The comparison of susceptible population over time between the classical ($\gamma_1 = 1$) and fractional model.

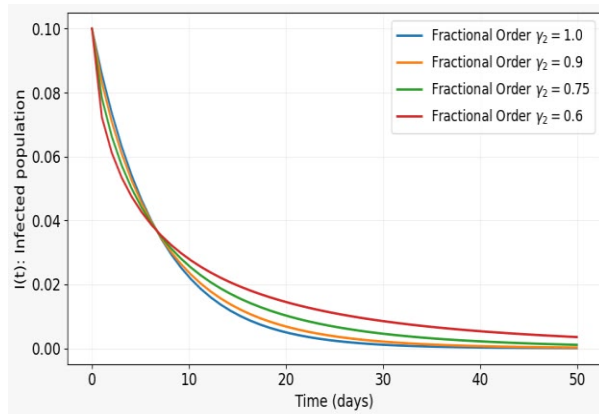


Fig. 21. The comparison of infected population over time between the classical ($\gamma_1 = 1$) and fractional model.

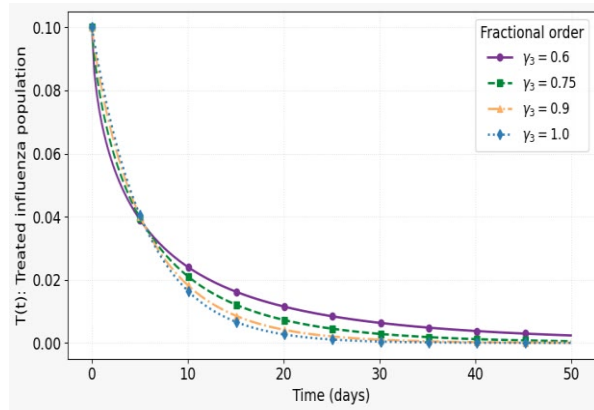


Fig. 22. The comparison of treated influenza population over time between the classical ($\gamma_1 = 1$) and fractional model.

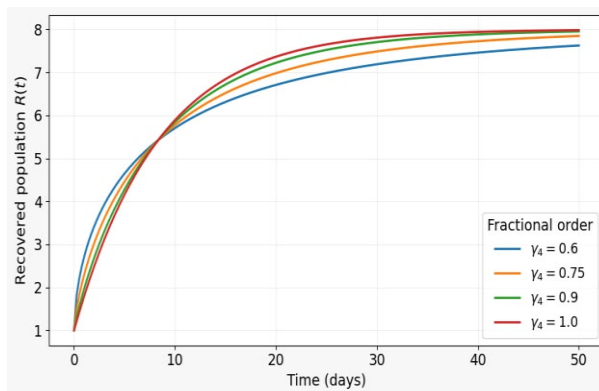


Fig. 23. The comparison of recovered population over time between the classical ($\gamma_1 = 1$) and fractional model.

Figs. 20–23 illustrate the substantial alterations in the temporal evolution of influenza dynamics resulting from the use of fractional-order memory, as compared with the conventional (integer-order) model. As the fractional order α decreases, the memory effects in all figures become stronger. This implies that both current circumstances and the

broader history of the epidemic shape the disease's current state.

Peak deviation formula: $\% \Delta I_{max}(\gamma_1) =$

$$\frac{I_{max}(\gamma_1) - I_{max}(1)}{I_{max}(1)} \times 100\% \quad (17)$$

$$(I_{influenza}^*) = I(t_{final}) = I(50 \text{ days})$$

For $\gamma_1 = 1$, $I_{max}(\gamma_1) = I_{max}(1) = 0.10$, Therefore deviation level $\% \Delta I_{max}(\gamma_1) = 0$

$$I_{max}(1) = 0.0010,$$

$$I_{max}(\gamma_1) = 0.0021$$

$$\text{For } \gamma_1 = 0.9, \% \Delta I_{max}(\gamma_1) = \frac{0.0021 - 0.0010}{0.0010} \times 100\% = +110$$

$$\text{For } \gamma_1 = 0.75, \% \Delta I_{max}(\gamma_1) = \frac{0.0032 - 0.0010}{0.0010} \times 100\% = +220$$

$$\text{For } \gamma_1 = 0.6, \% \Delta I_{max}(\gamma_1) = \frac{0.0048 - 0.0010}{0.0010} \times 100\% = +380$$

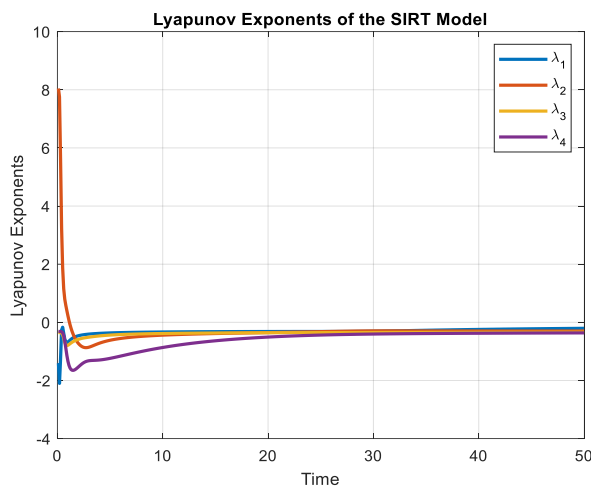
Table 4 displays the numerical results for the infected influenza population, clearly illustrating the influence of the fractional-order parameter on memory effects. In all simulations, the initial infected population was set to $I(0) = 0.10$, and the end observation time was set to $t = 50$ days. At $\gamma_1 = 1$, the infected population decreases to a low endemic level, $I(50) = 0.0010$, when the classical model is applied. This is the standard case that has not been altered. Memory effects cause a dramatic spike in the frequency of chronic illnesses when the fractional order is decreased. The infected population increases by 110% to 0.0021 at $t = 50$ when $\gamma_1 = 0.9$. By lowering the fractional order to $\gamma_1 = 0.75$, we get an endemic level of 0.0032, which is the same as a deviation of +220%. When the fractional order is set to $\gamma_1 = 0.6$, there is a 380% deviation from the classic example, resulting in an infected population of 0.0048. Even if the peak infection level remains the same because the initial conditions are the same, these data suggest that lowering the fractional order extends the infection's duration. This shows how vital memory effects are in the pathogenesis of chronic influenza.

Table 4. Numerical deviation in the infected influenza population for classical and fractional-order model.

Model type	Fractional order	Peak infected($I_{influenza}^{max}$)	Time to peak (days)	Endemic infection level($I_{influenza}^*$)	% $\Delta I_{max}(\gamma_1)$ Deviation in endemic level
Classical (ODE)	1.0	0.1	0	0.0010	0
Fractional	0.9	0.1	0	0.0021	+110
Fractional	0.75	0.1	0	0.0032	+220
Fractional	0.6	0.1	0	0.0048	+380

Lyapunov Exponents

Lyapunov exponents (Islam et al., 2025) can be used to describe how the influenza virus spreads through the body. To accomplish this, we assess whether the epidemic dynamics are stable, unstable, or chaotic by evaluating the system's sensitivity to initial conditions.

**Fig. 24. Lyapunov exponents ($\lambda_1, \lambda_2, \lambda_3, \lambda_4$) of the influenza model (1) over time.**

The Fig. 24 shows that the initial conditions may be sensitive and chaotic because the first two exponents, it is observed that the first two exponents, $\lambda_3 \approx$

0.015 and $\lambda_2 \approx 0.012$, are slightly positive, indicating sensitivity to initial conditions and suggesting possible chaotic dynamics. The third exponent, $\lambda_3 \approx 0.005$, implies weak expansion in another direction, while the fourth exponent, $\lambda_4 \approx -0.10$, is negative, contributing a stabilizing contraction to the system. The existence of both positive and negative Lyapunov exponents indicates a low-dimensional strange attractor and a potentially chaotic yet constrained course. This conduct underscores the complex dynamics of the examined epidemic, where slight alterations to initial conditions can yield significantly divergent outcomes, an essential insight for disease models in reality and the formulation of control methods.

Normalized sensitivity analysis

Normalized sensitivity analysis eliminates scale and unit differences, facilitating an equitable evaluation of factor impacts and ultimately pinpointing the parameters with the most significant influence. This is a fundamental reason why normalized sensitivity analysis is so important.

Table 5. Normalized sensitivity indices.

Parameter	Sensitivity index($\psi_{\rho}^{R_0}$)	Numerical value	Sign	Effect on R_0
λ	$\psi_{\lambda}^{R_0} = \frac{\partial R_0}{\partial \lambda} \times \frac{\lambda}{R_0} = 1$	+1.0000	Positive	Strong increase
A	$\psi_A^{R_0} = \frac{\partial R_0}{\partial A} \times \frac{A}{R_0} = 1$	+1.0000	Positive	Strong increase
μ	$\psi_{\mu}^{R_0} = \frac{\partial R_0}{\partial \mu} \times \frac{\mu}{R_0}$ $= -\left(1 + \frac{\mu}{\mu + r + \gamma}\right)$	-1.1579	Negative	Strong decrease
r	$\psi_r^{R_0} = \frac{\partial R_0}{\partial r} \times \frac{r}{R_0}$ $= -\left(\frac{r}{\mu + r + \gamma}\right)$	-0.3684	Negative	Moderate decrease
γ	$\psi_{\gamma}^{R_0} = \frac{\partial R_0}{\partial \gamma} \times \frac{\gamma}{R_0}$ $= -\left(\frac{\gamma}{\mu + r + \gamma}\right)$	-0.4737	Negative	Moderate decrease

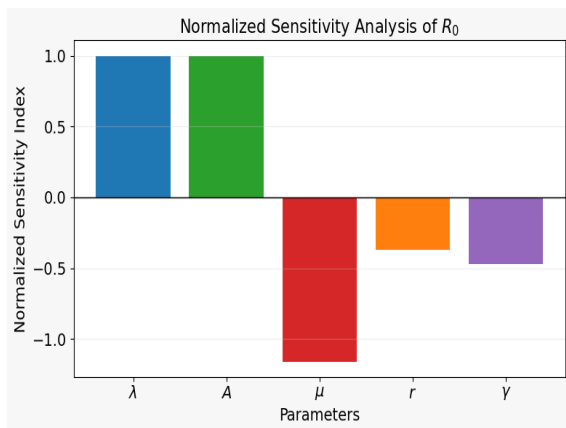
**Fig. 25. Normalized sensitivity analysis of the basic reproduction number R_0 .**

Table 5 and Fig. 25 show the normalized sensitivity indices for the fundamental reproduction number R_0 . These indices are shown in the table. The transmission rate λ and the recruitment rate A are the

By adjusting certain factors, the sensitivity analysis determines the effect on R_0 . With regard to a parameter ρ , the sensitivity index of R_0 is provided by:

$$\psi_{\rho}^{R_0} = \frac{\partial R_0}{\partial \rho} \times \frac{\rho}{R_0} \quad (18)$$

most critical positive sensitivity indices, with values of +1.0000 and +1.0000, respectively. In this way, R_0 increases significantly when these elements increase. Still, the natural death rate μ has the most extensive negative sensitivity index (−1.1579) among the parameters. This means that it stops the spread of disease in a big way. The recovery rate r and the treatment rate γ exhibit very low sensitivity, indicating that they stabilize the dynamics of influenza.

Global sensitivity analysis

Global sensitivity analysis is an essential tool for influenza disease models. It examines all possible parameter ranges to identify the factors that significantly affect how the disease spreads and what happens to people who get it. If we can separate the most important drivers, like transmission and recruitment rates, from the less important ones, the model's predictions will be more accurate. This information helps ensure that interventions focus on the most critical epidemiological processes, support smart policy decisions, and direct control efforts in the right direction. PRCC (Partial Rank Correlation Coefficient) is significant as it evaluates the

effects of nonlinear and interacting variables and quantifies the impact of parameters on model outputs.

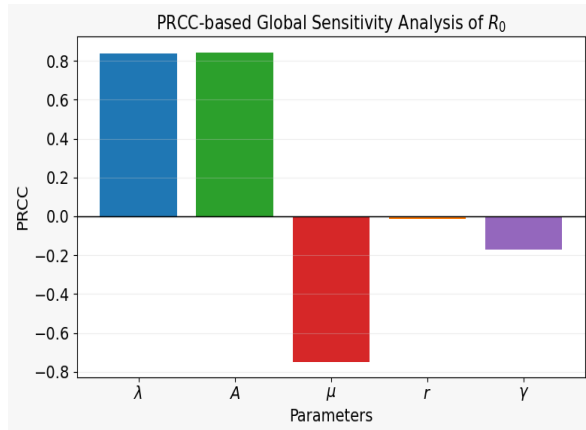


Fig. 26. PRCC-based global sensitivity analysis of the basic reproduction number(R_0).

From Fig. 26, the PRCC-based global sensitivity analysis indicates that the key factors affecting how quickly influenza spreads are the recruitment rate A and the transmission rate λ . Both rates substantially improve R_0 , with PRCC values exceeding 0.8. But recuperation r and therapy γ have a modest-to-moderate negative influence, which helps keep things stable. On the other hand, the natural death rate μ has a severe suppressive effect on R_0 .

Table 6. PRCC-based global sensitivity analysis showing the sign and qualitative impact of key model parameters on influenza disease transmission dynamics.

Parameter	Color	PRCC sign	Interpretation
λ	Blue	Positive(+)	Strongly increases R_0 ; primary driver of influenza transmission
A	Green	Positive(+)	Expands susceptible population, thereby increasing R_0
μ	Red	Negative(-)	Strongly suppresses transmission by reducing infectious population
r	Orange	Negative(-)	Slightly decreases R_0 ; shortens infectious period
γ	Purple	Negative(-)	Reduces transmission by moving infected individuals to treatment

$$\begin{cases} {}^c\Delta_{\alpha}^{\gamma_1} S(t) = h\{A - \mu S(t-1+\gamma_1) - \lambda S(t-1+\gamma_1)I(t-1+\gamma_1)\} + f_1(t-1+\gamma_1) \\ {}^c\Delta_{\alpha}^{\gamma_2} I(t) = h\{\lambda S(t-1+\gamma_2)I(t-1+\gamma_2) - (\mu+r)I(t-1+\gamma_2) - \gamma I(t-1+\gamma_2)\} + f_2(t-1+\gamma_2) \\ {}^c\Delta_{\alpha}^{\gamma_3} T(t) = h\{\gamma I(t-1+\gamma_3) - (\delta+\mu)T(t-1+\gamma_3)\} + f_3(t-1+\gamma_3) \\ {}^c\Delta_{\alpha}^{\gamma_4} R(t) = h\{rI(t-1+\gamma_4) + \delta T(t-1+\gamma_4) - \mu R(t-1+\gamma_4)\} + f_4(t-1+\gamma_4) \end{cases}$$

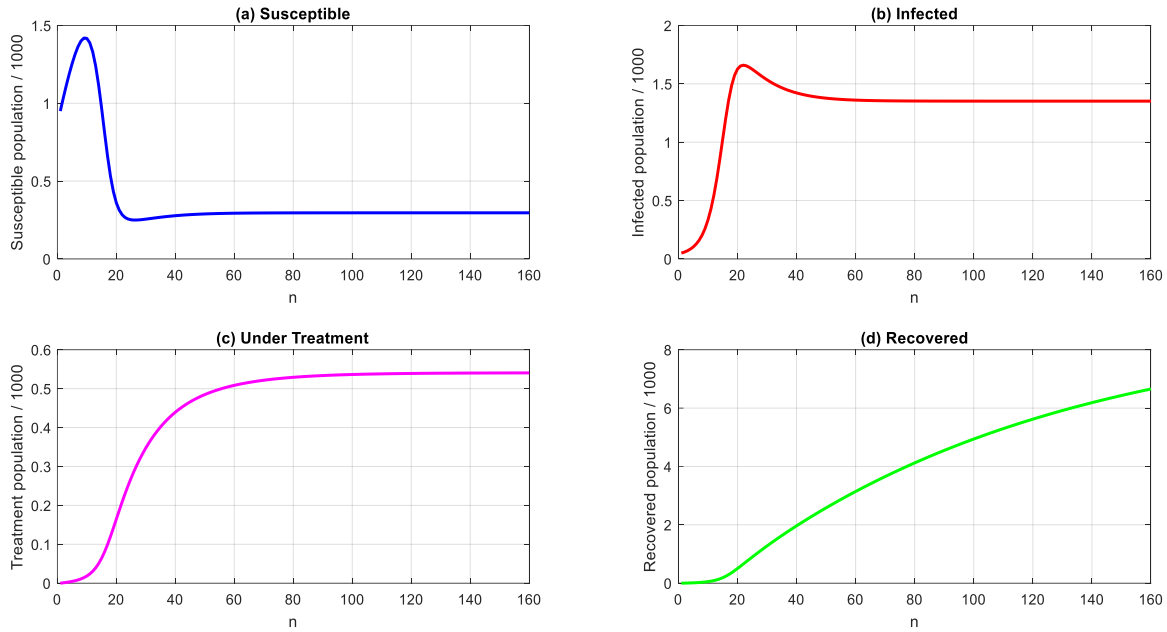


Fig. 27. Fitting results of the fractional order SITS model (12) using Table 2: (a) Susceptible population (b) Infected population (c) Population under treatment (d) Recovered population.

$$\begin{aligned} {}^C\Delta_{\alpha}^{\gamma_1} S(t) &= -h\mu S(t-1 + \gamma_1) \\ {}^C\Delta_{\alpha}^{\gamma_2} I(t) &= -h(\mu + r + \gamma)I(t-1 + \gamma_2) \\ {}^C\Delta_{\alpha}^{\gamma_3} T(t) &= -h(\delta + \mu)T(t-1 + \gamma_3) \\ {}^C\Delta_{\alpha}^{\gamma_4} R(t) &= -h\mu R(t-1 + \gamma_4) \end{aligned}$$

Which can be simplified as,

$${}^C\Delta_{\alpha}^{\gamma_i} x(t) = Ax(t-1 + \gamma_i)$$

$$\text{Where, } x(t) = \begin{bmatrix} {}^C\Delta_{\alpha}^{\gamma_1} S(t) \\ {}^C\Delta_{\alpha}^{\gamma_2} I(t) \\ {}^C\Delta_{\alpha}^{\gamma_3} T(t) \\ {}^C\Delta_{\alpha}^{\gamma_4} R(t) \end{bmatrix}$$

Obviously, the eigenvalues are: $\xi_1 = -h\mu, \xi_2 = -h(\mu + r + \gamma), \xi_3 = -h(\delta + \mu), \xi_4 = -h\mu$

The results of the global sensitivity analysis make it clear that reducing contact rates and recruiting people at risk should be the most critical steps to stop the virus's spread. Strengthening therapy and recovery procedures can also have secondary moderating effects.

Control law

Theorem 8. An asymptotic stabilization to zero equilibrium is possible for the fractional order discrete model (12) in accordance with the following non-linear feedback control law (Haneche et al., 2024).

$$\begin{cases} f_1(t) = h(\lambda S(t)I(t) - A) \\ f_2(t) = -h\lambda S(t)I(t) \\ f_3(t) = -h\gamma I(t) \\ f_4(t) = -h[rI(t) + \delta T(t)] \end{cases}$$

for $t \in \mathbb{N}_{1-\gamma_i}$

Proof

The fractional-order discrete SITS model (12) with controller $f = (f_1, f_2, f_3, f_4)^T$ can be designed as

These are real and negative, so $|\arg(\xi_i)| = \pi$. The fractional stability conditions are:

$$|\xi_i| < (2\cos\frac{|\arg(\xi_i)|-\pi}{2-\gamma_i})^{\gamma_i} \quad \text{and} \quad |\arg(\xi_i)| = \pi > \frac{\pi\gamma_i}{2}, \text{ for all } i = 1, 2, 3, 4$$

The system's zero equilibrium is asymptotically stable under the restrictions given above if the requirements are satisfied for the parameter values given above. Therefore, it follows that the states of the fractional-order discrete Sitr model asymptotically converge to zero.

Fig. 27 displays the temporal dynamics of the susceptible, infected, undergoing treatment, and recovered populations within the framework of the fractional-order Sitr model. The number of people likely to get sick drops quickly at the outset of an outbreak because so many are getting sick, but it returns to a steady level after a while. The number of ill people rises quickly, then slowly declines to a stable level. This pattern shows that therapy or recovery is working. Because the medical intervention works so well, the number of people receiving therapy rises quickly until it reaches its limit. The number of people who have recovered is also increasing, indicating that the medication and the body's immune response are working together. The system remains in a constant endemic state for the rest of its life, as predicted by the simulated dynamics, following an initial unstable breakout phase.

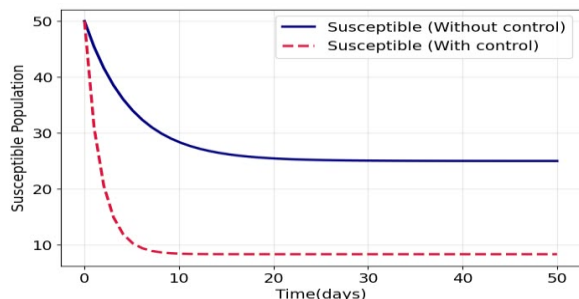


Fig. 28. Dynamics of the susceptible population $S(t)$ with and without control over a 50-day simulation period.

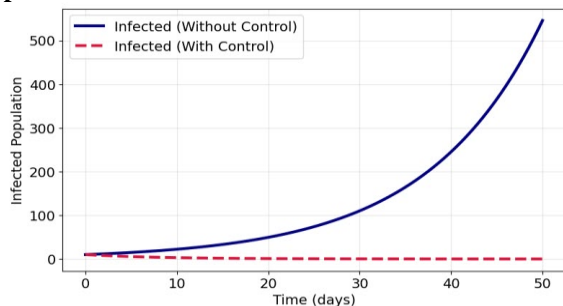


Fig. 29. Dynamics of the infected population $I(t)$ with and without control over a 50-day simulation period.

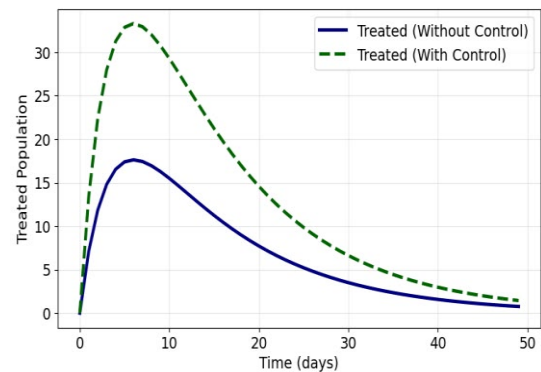


Fig. 30. Dynamics of the treated population $T(t)$ with and without control over a 50-day simulation period.

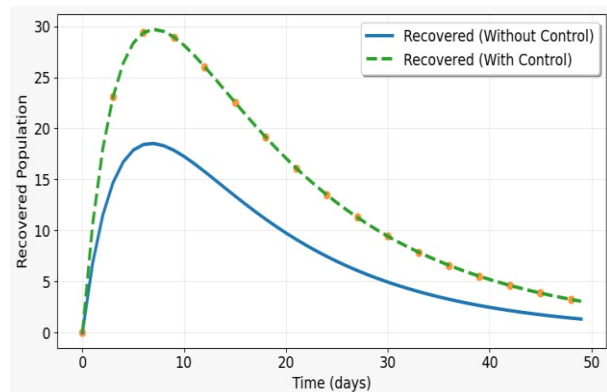


Fig. 31. Dynamics of the recovered population $R(t)$ with and without control over a 50-day simulation period.

As shown in Fig. 28, the sensitive population without control starts at $S(0) = 50$ and slowly drops until it reaches a steady value of about 25 by day 50. This happens when the population is almost stable. In the control group, $S(t)$ declines by about 70% in the first 10 days compared to the uncontrolled group. After that, it stays around 8, indicating that the control effect is statistically significant. The uncontrolled infected population grows quickly over the fifty-day simulation period; reaching over 500 by day fifty (see Fig. 29). This shows that the pandemic is still spreading. But in the early stages of the sickness, the number of afflicted people declines sharply and approaches zero, indicating that the control mechanism is working. Fig. 30 shows that the treated population grows initially in both situations, reaching its peak between days 6 and 8. The highest number of patients who can have treatment without control is about 17, and then it slowly decreases. The treated

group has a higher peak of about 32 than the control group, and that size stays the same throughout the simulation. Control strategies improve treatment coverage, as shown below. Fig. 31 shows that the number of people who have recovered increases initially, reaching a peak between days 7 and 9. In an unregulated setting, the most that can be regained is a population of roughly 18. After that, the number of people starts to go down slowly. Control measures have helped the recovery process by enabling the recovered population to reach a higher peak of approximately 30 during the simulation and to remain higher throughout.

Conclusions

A deterministic, fractional-order discrete-time Sitr influenza model was built and tested in this study to address the limitations of existing integer-order frameworks in modeling memory-dependent transmission dynamics and treatment effects. These good qualities, along with stability and boundedness, were confirmed by a thorough theoretical investigation. It has found that the basic reproduction number R_0 is a vital threshold that prevents the disease from dying out. Figs. 3 and 4 shows that $R_0 > 1$ increase as the disease spreads and decreases as the rates of recovery. At $R_0 = 1$, a threshold is crossed. When R_0 less than 1, the disease-free state is stable. Infections are still very much present, even though R_0 is getting close to 4.5. The results show that fractional-order dynamics better capture the spread of pandemics through memory than integer-order models. Fig. 6 indicates that sensitive individuals get sick quickly, and then have an early peak in infections, a peak in treatment, and a later peak in recovery.

The use of fractional-order memory improves stability, accounts for delayed interventions, and explains nonlinear epidemic dynamics more thoroughly (as shown in Figs. 7–15). The recruiting rate takes precedence over the fractional order in terms of importance. Fig. 17 shows the findings of the numerical heat map. The results show that bands with low responsiveness are produced by significant

thresholds at $\gamma_1 \approx 0.94$. The model stability is particularly sensitive to specific combinations of fractional-order parameters, as shown by the similar patterns on both the horizontal and vertical axes. Fig. 18 shows that spectral entropy increases from about 0.45 to 0.75–0.80 as the overall fractional order increases. This indicates an increase in complexity, alongside stabilizing and sensitivity-driven SE tendencies specific to the parameters and non-monotonic. Fig. 19 shows that the damped oscillations in all compartments reach a steady state. The closed and converging phase trajectories in Fig. 19 further confirm the asymptotic stability and global equilibrium of the fractional-order discrete Sitr model. Figs. 20–23 show that lowering the fractional order α improves memory benefits. This has a significant effect on how influenza spreads when compared to the integer-order model. This is because both the present and the past states are essential for the system to change. Table 4 shows that the infected population grows rapidly at $t = 50$ because the fractional order γ_1 decreases. A rise from 0.0010 to 0.0048 (γ of chronic influenza). The Lyapunov exponents (λ) in Fig. 24 lie within a positive range (0.012 to 0.015), indicating that the model is sensitive to initial conditions and could be chaotic. In contrast, contraction is guaranteed by a negative exponent ($\lambda_4 \approx -0.10$), indicating that the epidemic defines this trend, emphasizing the significant influence of memory effects on the duration dynamics, which are limited from a low-dimensional perspective.

Fig. 25 and Table 5 demonstrate that the two variables with the most significant positive effects on R_0 are the recruitment rate A and the transmission rate λ , both with a value of +1.0000. The natural death rate μ , on the other hand, is -1.1579, which is the worst case. On the other hand, the low sensitivity of recovery r and therapy γ helps stabilize the flu dynamics. Fig. 26 shows that the primary factors driving influenza spread ($PRCC > 0.8$) are the recruitment rate A and the transmission rate λ . This was found using the PRCC-based global sensitivity

analysis. On the other hand, recovery r and therapy γ lower R_0 a lot, but the natural death rate λ lowers it much more. Figures 28–31 show that control methods significantly reduce the number of people who are susceptible or infected, swiftly stop the spread of infection, improve treatment coverage, and increase long-term recovery compared to the uncontrolled scenario. Overall, our results demonstrate that the fractional-order model is a more realistic and adaptable approach for modeling virus spread than conventional models. This is especially true when representing dynamics that are important for control, nonlinear behavior, and memory effects. Subsequent research should build on this groundwork by employing stochastic fractional and variable-order models to incorporate uncertainty, seasonal influences, and temporal fluctuations in influenza transmission. Using machine learning methods such as neural networks and hybrid physics-informed learning models, it will also be possible to estimate parameters, make predictions, and optimize control strategies in real time using epidemiological data.

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Authors contribution

Md. Asraful Islam: Conceptualization, methodology, formal analysis, investigation, data curation, and visualization of the study. Sajjad Hossain and Md. Asraful Islam: Development of the methodology. Sajjad Hossain: Writing the original draft of the manuscript. Md. Asraful Islam: Review and editing of the manuscript.

Conflict of interest

The author's state that they are not involved in any conflicts of interest.

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