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The fuzzy fractional SIQR model of computer virus propagation in wireless sensor network using Caputo Atangana–Baleanu derivatives

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Abstract

This work is devoted to study the uncertain attacking behavior of computer viruses in wireless sensor network involving fuzzy fractional derivatives with non-local Mittag-Leffler function kernel. Based on epidemic theory and fractional calculus, we propose a fuzzy fractional Susceptible – Infectious – Quarantine – Recovered (SIQR) model to describe dynamics of virus propagation with quarantine in the network. The concept of Atangana–Baleanu fuzzy fractional derivative in Caputo sense is proposed with some important properties to investigate the fractional SIQR model with fuzzy data. The fuzzy Laplace transform of Atangana–Baleanu derivative is proposed to represent the analytic mild solutions of the fractional SIQR model. Then, the existence and uniqueness of mild solutions is proved by using generalized contraction principle. An efficient numerical scheme to solve numerical solutions of the fractional SIQR model is introduced. Finally, some graphical representations are given to show the uncertain attack behavior of computer virus and dynamical behavior of the model.

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Keywords: Fuzzy fractional SIQR model; Wireless sensor network; Fuzzy Atangana-Baleanu fractional derivatives; Computer virus propagation

1. Introduction

Wireless Sensor Networks (WSNs) are known as dense systems consists of numerous small-sized, energy-limited, and multi-functional sensor nodes that are deployed to collect data from an environment or monitor of a phenomenon and then, transmit the collected data to users and administrators. The appearance of WSNs in the past two decades has led to the dramatic change in the way that data is transferred and information exchange takes place and open up for the high development of digital age. Recently, WSNs have received extensive attention due to their great potential in civil

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Fig. 1. The diagram of wireless sensor network.

and military applications, such as supervision of the transportation of pollutants, industrial monitoring, military target tracking, disaster recovery, biological detection, seismic sensing and health monitoring. Over the past few decades, along with the rapid spread of the internet and wireless sensor networks, the spreads of malware objects, such as viruses, worms, trojans, etc, have also been staggering. In addition, due to the restriction of memory resources of sensor nodes and disadvantage in their locations, WSNs often have weak defenses and become attractive targets of malware objects, especially cyber attacks by computer viruses. See Fig. 1.

Therefore, antivirus software has become an important way to safeguard computer systems and WSNs. Unfortunately, no antivirus software can detect and clear all kinds of viruses, and the development of antivirus software, in turn, always lags behind that of viruses. Therefore, the most important prerequisite to optimally minimize the influences of computer virus is to understand the features of computer viruses and the characteristics of their spreads and then, to predict their evolutionary directions. For this goal, based on the similarity between the spread of diseases in populations and the spread of computer viruses in WSNs, some epidemic models have been applied to model and study the spread of computer viruses in WSNs. The mathematical modeling of malware objects transmissions has been approached by a number of researchers with variety of models over past decades. In [29], Mishra and Saini proposed a delay SEIRS epidemic model to study the spread of malicious objects in computer networks and the stability of malicious objects-free equilibrium point. Next, the paper [30] formulated the transmission of worms in wireless sensor network by the SEIRS-V model to describe the dynamics of the transmission with respect to time and investigate the relation between its basic reproduction number and the stability of its equilibrium points. After that, a predator-prey model [31] was proposed to analyze the impact of energy conservation in the context of worm attacks in wireless sensor network and determine the conditions for the stability of different equilibrium points. Recently, Singh et al. [44] studied a fractional epidemiological model for the spread of computer viruses and discussed the influence of arbitrary order with the dynamical behaviors of virus transmission.

The infection of malware objects such as viruses, worms or trojans in WSNs are based on the transmission of electric-waves and signals between wireless sensor nodes. However, in particular, signal transmissions are known as memory and hereditary processes that often have the significant dependence on the flexibility of environment, the history of functions or the texture and characteristic properties of the material, that are difficult to describe correctly by mean of integer order differential systems. Fractional derivatives and fractional integrals have non-local property, i.e., these derivatives and integrals can present both the past information and distributed effect of any physical systems. This proves the great ability of fractional calculus to represent complex real-world phenomena more accurately and efficiently than ordinary calculus. Indeed, fractional calculus is now known as an important branch of mathematical analysis with a long history of development and various applications in science and engineering. Many scientists considered fractional-order derivative as an effective tool to describe memory phenomena. Here, the kernel function of fractional derivative is called memory function. Each physical phenomena can be better modeled by a different type of kernel functions. For example, in order to express the properties of viscoelastic materials with memory in Kevin model, Voigt model, Maxwell model, they proposed to use the non-singular kernel function $(t - s)^{\alpha}$ while in the case

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of expressing decay physical processes, the singular kernel of the forms $\exp(t-s)^{\alpha}$ or $\mathbb{E}_{\alpha,\beta}((t-s)^{\alpha})$ were suggested. This leads to there are many concepts of fractional derivatives with different capability of applications. Some wellknown derivatives, such as Caputo, Riemann-Liouville fractional derivatives or the definition of fractional derivative in sense of Grünwald, that uses the power law as singular kernel, see [24,39,42]. Alongside with the development of fractional calculus, fractional differential equations and fractional partial differential equations have been also dramatically studied with the high applicability, see [4,10-12,16,23,32,44]. In several decades, the study of fractional calculus in uncertain environment has achieved some noticeable results. For instance, R. Agarwal [2] investigated Cauchy problem for fuzzy fractional equations, in which the proposed fuzzy fractional differential equations were studied without using fuzzy fractional derivatives. Next, Allahviranloo et al. [6] investigated the initial value problem for fuzzy fractional differential equations with the used of fuzzy Riemann-Liouville fractional derivatives. In the field of fuzzy partial differential equations, Long et al. studied local and non-local problems for fuzzy fractional partial differential equations under Caputo gH-differentiability, see [26,27]. Recently, there have been several literature that considered fractional epidemic models and their applications in computer networks and WSNs such as Hassouna et al. [17], Huo and Zhao [20], J. Singh et al. [44]. In the last decades, many researchers working within the field of fractional calculus have stated that the all physical problems were claimed to follows the power-law process, i.e., the fractional operators have singular kernel. However, it is implied from [19] that some models of dissipative phenomena cannot be adequately described by the fractional derivative operators with singular kernel. Indeed, there are quite a lot of physical problems that are modeled better by other type of kernel than by power-law such as the problem of fatigue expressed by a runner or the decay process of a dead body in force air. Recently, Caputo and Fabrizio [15] have suggested an alternative concept of fractional calculus with non-singular kernel by using the exponential decay as kernel. An other concept of fractional differentiation with non-singular kernel is proposed by Atangana and Baleanu [8]. The Atangana-Baleanu (AB) fractional calculus uses the Mittag-Leffler function as its non-local kernel, that follows at the same time the exponential decay and power law. The new concepts of Atangana-Baleanu fractional calculus are expected to better modeling the effect of memory in complex physical systems and recently, these types of fractional calculus have achieved lots of noticeable results. For example, in the literature [9], Atangana applied the concept of Caputo-Fabrizio fractional derivative to study a modified fractional model of nonlinear Fisher's reaction-diffusion equation. The Caputo-Fabrizio fractional derivative in Riemann-Liouville sense then continued to be applied to investigate the numerical algorithm of a fractional partial differential equation of parabolic type in [35]. An application of Caputo-Fabrizio fractional calculus was discussed in [23] with the use of Caputo-Fabrizio fractional derivative to model the dynamics of hepatitis E virus. Another result on the fractional calculus of fuzzy-valued functions was proposed by Son et al. [48] with the foundation on some different types of fuzzy fractional derivatives under Fréchet differentiability. In addition, Adams-Bashforth numerical scheme was applied to describe behavior of the HEV disease model. In the literature [10], the authors demonstrated the application of fractional differential operators with no index law properties to statistic and dynamical systems compared with fractional operators obeying index law. In addition, some important properties and theorems for the Atangana-Baleanu fractional derivative of an analytic function were established. After that, Atangana and Hammouch [12] constructed a new class of partial integro-differential equations with fractional operators based on the new generalized Mittag-Leffler function and presented a detailed discussion underpinning the conditions for which the new partial integro-differential equation is well-posed. Next, Owolabi [36] discussed an ecological system consisting of a predator and two preys with the newly derived two-step fractional Adams-Bashforth method via the Atangana-Baleanu derivative in the Caputo sense. In the field of engineering, Alkahtani [4] used the Caputo Atangana–Baleanu fractional derivative to study Chua's circuit model and gave a numerical scheme for numerically solving the modified model. In the literature [40], Saad et al. compared three concepts of fractional derivatives: Liouville-Caputo, Caputo-Fabrizio, Mittag-Leffler fractional time derivatives and applied to study three modified fractional models of Burgers equation and corresponding approximate solutions based on Homotopy analysis transform method.

It is natural that all real-world phenomena always contain vague and uncertain factors. Hence, there is often a considerable degree of uncertainty when we model, solve and interpret the problems in natural environments. In addition, rather than the particular value, we may have only imprecise, and incomplete information about the variables and parameters due to applying different operating conditions or a result of errors in measurement and experiment. As a consequence, fuzzy differential equations appeared as a natural way to model the propagation of epistemic uncertainty in a dynamical environment. Especially, for epidemic models in wireless sensor network, because the signal transmission environment has memory property and always contains uncertain parameters, it is necessary to inter-

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pret and control the uncertain trajectory of the transmission in these models. This class of differential equations then demonstrated its important role in describing real-world problems with various applications in many fields of science such as biological processes [38,53], control problems [1] and engineering [22,43], etc. In addition, to deal with nonlocal real-world phenomena with memory property, an interesting type of differential equations, combined both the notions of fractional order derivative and fuzzy, was proposed by Agarwal [2]. Fuzzy fractional differential equations are considered as effective tools to enable researchers to describe the uncertain behaviors of dynamical systems in time and space. Along with mathematical progress, they also have a wide range of applications in various fields of science and engineering such as physics, chemistry, applied mathematics, biology, economics. For example, the paper [3] studied the optimality condition for a fuzzy optimal control problem subject to fuzzy fractional differential system with the fractional derivative in Caputo sense. In the literature [18], the authors studied the existence and uniqueness of fuzzy solution to an initial value problem of Caputo-Katugampola fractional differential equations in fuzzy setting. In the perspectives of control theory, Mani et al. [32] addressed the synchronization problem of chaotic fractionalorder fuzzy cellular neural networks through designing the novel adaptive control scheme, while in [33], the authors proposed a novel adaptive T-S fuzzy variable structure control technique for nonlinear fractional-order systems in spite of the saturating input and control fluctuations. In [34], the authors presented a novel adaptive interval type-2 fuzzy fractional-order back-stepping sliding mode control method to design controllers for some classes of nonlinear fully-actuated and under-actuated mechanical systems with uncertainty. The stability of fuzzy fractional dynamic systems also get more attention. In [52], Tyagi and Martha discussed some criteria for the finite-time stability of a class of fractional fuzzy differential systems with proportional delay and applied to study the stability of neural network. Beside analytic results, some numerical methods for fractional differential systems were also studied in [33,53]. For more applications, see [37,45,47,54,55].

In this work, we formulate a fractional mathematical model of computer virus propagation with quarantine and uncertain initial data in WSNs via Caputo Atangana–Baleanu fractional derivative. Here, we consider the fractional derivative with non-local and non-singular kernel since it has a great ability in describing dramatic spread like that of computer viruses and permits the incorporation of memory effect in the model. In summary, the paper's contributions can be highlighted as follows:

- (i) In Section 2, the formulation of a fractional mathematical model of virus propagation in WSNs is presented. The proposed model consists of four compartments: Susceptible (S) Infectious (I) Quarantine (Q) Recovered (R) that describes the uncertain dynamical behaviors of the virus attack with quarantine in the network and fuzziness in initial data.
- (ii) A new concept of fractional derivative with non-local and non-singular kernel of fuzzy-valued functions, namely Caputo Atangana–Baleanu fractional derivative, is introduced in Section 4. Here, the proposed Atangana– Baleanu fractional derivative has the non-local kernel of Mittag-Leffler function type. Additionally, the concept of Riemann–Liouville Atangana–Baleanu fractional integral associated with the proposed fractional derivative is also derived. Moreover, the fuzzy Laplace transform for the new fractional order derivative is presented in Appendix section.
- (iii) An initial value problem (6) for the fuzzy fractional differential system with Atangana–Baleanu fractional generalized Hukuhara derivative in sense of Caputo is studied at Section 5. Then, the integral formulas of mild solutions in both type 1 and type 2 of the problem (6) are constructed and proved by using fuzzy Laplace transform (Theorem 5.1).
- (iv) The existence and uniqueness of mild solutions of the initial value problem (6) are obtained by virtue of generalized contraction principle in Theorem 5.2 and Theorem 5.3.
- (v) A numerical algorithm to numerically solve the initial value problem (1)–(2) for fractional SIQR model with fuzzy initial data is proposed and demonstrates its effectiveness by simulations in Section 6.

2. The attacking model of viruses in WSNs

In this work, we consider the fractional-order model of virus propagation in wireless sensor network. In this network, it is assumed that all sensor nodes belong to one of four possible states during the process:

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Fig. 2. The SIQR model of virus propagation.

- (S) The state (S) consists of all wireless sensor nodes which haven't been infected by any virus, however, these nodes are vulnerable to virus and hence, they are called susceptible nodes.
- (I) The wireless sensor nodes in the state (I) have been infected by viruses in WSNs and moreover, they may infect to other sensor nodes in the state (S). The sensor nodes in this state are said to be infectious nodes.
- (Q) The state (Q) consists of all wireless sensor nodes which are quarantined from the nodes in the state (I) and these nodes are said to be quarantine nodes.
- (**R**) The sensor nodes in the state (**R**) are clear of viruses, and they are temporarily immune. However, these sensor nodes then become susceptible towards the possible attack of viruses.

Denote S(t), I(t), Q(t) and R(t) by the numbers of susceptible, infectious, quarantine and recovered sensor nodes at time t, respectively. Then, the model of virus propagation can be schematically described in following diagram, see Fig. 2.

To defend WSNs against virus's attacking, we need to accurately understand the dynamic characteristics of the spread of viruses. Hence, the mathematical modeling for virus propagation is considered as an effective tool for not only describing the process of information and disease diffusion in human society, but also predicting the outbreak of malware propagation in WSNs. In this work, we proposed the SIQR model to describe the spread of computer viruses in WSNs. Here, we assume that every independent sensor node in these states (S), (I), (Q) or (R) leaves the network with a rate μ . Then, the mathematical SIQR model can be formulated as follows:

State (S): Under assumption that the sensor nodes outside the WSNs enter the network at a rate A, the number of susceptible nodes decreases since these nodes are infected by viruses with probability $\lambda I(t)$ and leave the network with a rate μ , while there is an increase in the number of susceptible nodes coming from the recovered and quarantine states with rates σ and ω , respectively. Finally, the rate of change of the state (S) can be formulated in following differential equation:

$$\frac{dS(t)}{dt} = A - \lambda S(t)I(t) + \omega Q(t) + \sigma R(t) - \mu S(t)$$

It should be noted that the transmission of computer viruses in WSNs is a complex physical phenomena having non-local property with memory effects. Thus, fractional derivatives with non-singular kernel are suggested to represent this process for more accurately and efficiently. In this work, we introduce the concepts of Caputo Atangana–Baleanu derivative–a fractional derivative with non-local kernel of Mittag–Leffler function type, to represent the rate of change of virus propagation in WSNs. In addition, the approach of fuzzy dynamic systems is commonly employed to deal with the problem of virus's attacking in the real-world environments that contain vague factors and often have a lack of information or incomplete signals. This approach allows us to depict and present the uncertain behaviors of propagation in real-world environment. Moreover, this interpretation can be closer to the origin of reality models, and it has enough ability to be extended to the other models. Therefore, we propose here the concept of fuzzy Atangana–Baleanu fractional derivative to represent the rate of change of the quantity (S):

$${}^{abc}\mathfrak{D}^{\beta}_{+}S(t) = A - \lambda S(t)I(t) + \omega Q(t) + \sigma R(t) - \mu S(t).$$

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Here, ${}^{abc}\mathfrak{D}^{\beta}_{+}$ is the fuzzy Atangana–Baleanu fractional differential operator of order β and $S, I, Q, R : \mathbb{R} \to \mathscr{E}$ are given fuzzy-valued functions.

By the same arguments, we also formulate the fractional differential equations to describe the rate of change of three states (I), (Q) and (R) as follows:

- State (I): It is a fact that viruses infect sensor nodes in the state (S) and transfer them into the infectious state with a probability $\lambda I(t)$. Each infectious node then belongs to one of three following cases:
 - (i) The infectious node leaves the WSNs with a rate μ .
 - (ii) Under the action of detection programs, every infectious node can be isolated to be a quarantined node with probability γ .
 - (iii) By antivirus programs, each infectious node becomes recovered node with a rate v.

Hence, the fractional differential equation describing the rate of change of (I) is given by

$${}^{abc}\mathfrak{D}^{p}_{+}I(t) = \lambda S(t)I(t) - (\nu + \gamma + \mu)I(t).$$

- State (Q): It is well-known that the infectious node can be isolated to be a quarantined node with a probability γ . Then, each sensor node in the state (Q) can be transferred into one of following states:
 - (i) Each quarantine node can leave the WSNs with a rate μ or be released to the recovered state with a rate η .
 - (ii) The quarantine node can be released to the susceptible state with a rate ω by reinstalling the systems.
 - Then, the fractional differential equation describing the rate of change of (\mathbf{Q}) is given by

$${}^{abc}\mathfrak{D}^{\beta}_{+}Q(t) = \gamma I(t) - (\eta + \mu + \omega)Q(t).$$

State (R): The rate of change of the recovered state (R) is described as follows

- (i) Each recovered node leaves the WSNs with a rate μ and each quarantine node is released to the recovered state with a rate η .
- (ii) By using antivirus program, each infectious node is recovered with a rate ν . These nodes then enter the WSNs and become susceptible nodes with a rate σ .

Hence, the fractional differential equation with respect to the state (**R**) is

$$^{abc}\mathfrak{D}^{\beta}_{+}R(t) = \nu I(t) + \eta Q(t) - (\sigma + \mu)R(t).$$

In summary, uncertain dynamic behavior of the virus propagation is modeled by the following fuzzy fractional differential system

$$\begin{cases} a^{bc} \mathfrak{D}^{\beta}_{+} S(t) = A - \lambda S(t) I(t) + \omega Q(t) + \sigma R(t) - \mu S(t) \\ a^{bc} \mathfrak{D}^{\beta}_{+} I(t) = \lambda S(t) I(t) - (\nu + \gamma + \mu) I(t) \\ a^{bc} \mathfrak{D}^{\beta}_{+} Q(t) = \gamma I(t) - (\eta + \mu + \omega) Q(t) \\ a^{bc} \mathfrak{D}^{\beta}_{+} R(t) = \nu I(t) + \eta Q(t) - (\sigma + \mu) R(t) \end{cases}$$
(1)

subject to the fuzzy initial conditions

$$S(0) = S_0, \ I(0) = I_0, \ Q(0) = Q_0, \ R(0) = R_0.$$
 (2)

Here, the parameters of the fractional differential system (1) are discussed in Table 1.

Some related properties to evaluate virus's attacking behavior of fractional SIQR model are given by

• Denote by N(t) the total number of sensor nodes in WSNs, that is

$$N(t) = S(t) + I(t) + Q(t) + R(t), \qquad \forall t \ge 0.$$

• The basic reproduction number is an epidemiologic metric used to describe the transmissibility of infectious agents such as viruses, worms or trojans. In the SIQR model, the basic reproduction number \Re of fractional differential system (1) is given by $\Re = \frac{\lambda A}{\mu(\nu + \nu + \mu)}$.

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Variables and some parameters.						
	Meaning					
Variable						
S(t)	The number of susceptible nodes at time <i>t</i>					
I(t)	The number of infectious nodes at time t					
Q(t)	The number of quarantine nodes at time t					
R(t)	The number of recovered nodes at time t					
Parameter						
ν	The recovery rate					
σ	The rate of being susceptible from the recovered state					
λ	The rate of being infected from the susceptible state					
μ	The rate of logging out the network of sensor nodes					
η	The rate for quarantine node is released to recovered node					
ω	The rate of being susceptible from the quarantine state					
γ	The probability for an infectious node is isolated to be a quarantined node					
Α	The rate of logging in the network of sensor nodes					

Table 1	
Variables and	some parameters.

• The equilibrium points of the fuzzy fractional differential system (1) are obtained by solving the corresponding stationary system. Then, we directly obtain that the set of equilibrium points consists of a virus-free equilibrium point $E_1 = \left(\frac{A}{\mu}, 0, 0, 0\right)$ and a virus-endemic equilibrium point $E_2 = (S^*, I^*, Q^*, R^*)$.

3. Preliminaries

In this section, we recall some essential facts from basic concepts of fuzzy analysis and fuzzy fractional calculus [13,14,25,49,24,39]. Since there are some derivative and integral operators used in this paper then in order to avoid confusing for the readers, we introduce a table of used notations (see Table 2 in Appendix section).

Firstly, we recall from [13] that a fuzzy number u on \mathbb{R} is a mapping u: $[a, b] \subseteq \mathbb{R} \to [0, 1]$ satisfying four following conditions: normal, upper semi-continuous, fuzzy convex and compact supported. Denote by \mathscr{E} the space of all fuzzy numbers on the real line \mathbb{R} . Then, for each $u, v \in \mathscr{E}$, it is well-known that

(i) The level sets or α -cuts of the fuzzy number u, denoted by $[u]^{\alpha}$, is defined by

$$[u]^{\alpha} = \begin{cases} \{x \in \mathbb{R} : u(x) \ge \alpha\} & \text{if } \alpha \in [0, 1] \\ \text{cl(supp } u) & \text{if } \alpha = 0 \end{cases}$$

and can be written in the parametric form $[u]^{\alpha} = [u_{\alpha}^{-}, u_{\alpha}^{+}], \alpha \in [0, 1].$

(ii) For each $\alpha \in [0, 1]$, the length of α -cuts of the fuzzy number u is denoted by

$$\operatorname{len}([u]^{\alpha}) = u_{\alpha}^{+} - u_{\alpha}^{-}.$$

(iii) The generalized Hukuhara difference (or gH-difference for short) of u and v, denoted by $u \ominus_{gH} v$, is an element $w \in \mathscr{E}$ such that

$$u \ominus_{gH} v = w \Longleftrightarrow \begin{bmatrix} (i) & u = v + w \\ (ii) & u = v \ominus (-1)w \end{bmatrix}$$

where + and \ominus are Minkovski addition and Hukuhara difference of u and v, respectively.

(iv) The space \mathscr{E} is a complete metric space endowed with the metric

$$d_{\infty}(u, v) = \sup_{\alpha \in [0, 1]} d_H\left([u]^{\alpha}, [v]^{\alpha}\right) = \sup_{\alpha \in [0, 1]} \max\left\{\left|u_{\alpha}^{-} - v_{\alpha}^{-}\right|, \left|u_{\alpha}^{+} - v_{\alpha}^{+}\right|\right\}.$$

In addition, the norm $\|.\|$ on the space \mathscr{E} is defined by

$$||u|| = d_{\infty}(u, \hat{0}) = \sup_{\alpha \in [0, 1]} d_H\left(\left[u_{\alpha}^{-}, u_{\alpha}^{+}\right], [0, 0]\right),$$

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where $\hat{0} \in \mathscr{E}$ is zero fuzzy number and $\left[\hat{0}\right]^{\alpha} = [0, 0]$ for all $\alpha \in [0, 1]$. However, the metric space $(\mathscr{E}, d_{\infty})$ is neither linearity space nor locally compact space, see [13].

(v) Denote $\mathscr{E}^n := \underbrace{\mathscr{E} \times \mathscr{E} \times \cdots \times \mathscr{E}}_n$. Then, we define the vector-valued metric on the space \mathscr{E}^n by a mapping \mathbb{D}_n :

$$\mathscr{E}^n \times \mathscr{E}^n \to \mathbb{R}^n$$
, in which

$$\mathbb{D}_n(u,v) = \begin{pmatrix} d_{\infty}(u_1,v_1) & d_{\infty}(u_2,v_2) & \cdots & d_{\infty}(u_n,v_n) \end{pmatrix}^{\mathsf{L}}$$

for all $u = (u_1, ..., u_n)$, $v = (v_1, ..., v_n) \in \mathscr{E}^n$ and t denotes for the transposition of a vector or a matrix. It should be noted that the space $(\mathscr{E}^n, \mathbb{D}_n)$ is a generalized complete metric space. A preceding result for the case n = 2 was presented in [28]. The proof for general case is left as an exercise to the readers.

Example 3.1. Let us consider an L–R fuzzy number A given by

$$A = \begin{cases} x^2 & \text{if } 0 \le x < 1\\ \frac{(3-x)^2}{4} & \text{if } 1 \le x \le 3\\ 0 & \text{if } x \notin [0,3], \end{cases}$$

which illustrates the concept: "A number is near 1". Then, the α -cuts of the fuzzy number A are given by $[A]^{\alpha} = \left[\sqrt{\alpha}, 3 - 2\sqrt{\alpha}\right], \alpha \in [0, 1]$. In addition, the norm of A is given by

$$\|A\| = d_{\infty}\left(A, \hat{0}\right) = \sup_{\alpha \in [0,1]} \max\left\{\left|\sqrt{\alpha}\right|, \left|3 - 2\sqrt{\alpha}\right|\right\} = 3.$$

Let $f : [0, b] \subset \mathbb{R} \to \mathscr{E}$ be a fuzzy-valued function. Then, the detailed concepts on the limit of function, the continuity, the gH-differentiability and integrability can be referred from [14,25]. In the following, we recall some of the most essential properties of fuzzy-valued functions:

Definition 3.1 ([13,14]). The fuzzy-valued function f is called generalized Hukuhara differentiable (or gHdifferentiable for short) on the interval (0, b) if and only if it is gH-differentiable at each point $t_0 \in (0, b)$, i.e., there exists $f'_{oH}(t_0) \in \mathscr{E}$ such that for all h > 0 sufficiently small, we have

$$f(t_0 + h) \ominus_{gH} f(t_0) = f'_{gH}(t_0)h + \varepsilon(h),$$

where the function $\varepsilon_h : \mathbb{R}^+ \to \mathscr{E}$ satisfies $\lim_{h\to 0} \frac{1}{h} d_{\infty}(\varepsilon_h(h), \hat{0}) = 0$. The fuzzy number $f'_{gH}(t_0)$ is then called generalized Hukuhara derivative (or gH-derivative for short) of function f(t) at the point t_0 . Denote by $C^1((0, b), \mathscr{E})$ the space of all continuously gH-differentiable fuzzy functions on (0, b).

Definition 3.2 ([14]). Assume that the function $f \in C^1((0, b), \mathscr{E})$ and its α -cuts are written in following parametric form $[f(t)]^{\alpha} = [f_{\alpha}^{-}(t), f_{\alpha}^{+}(t)]$ for each $t \in (0, b)$ and $\alpha \in [0, 1]$.

(i) If f is gH-differentiable in type 1 at t_0 then $\left[f'_{1-gH}(t_0)\right]^{\alpha} = \left[(f^-_{\alpha})'(t_0), (f^+_{\alpha})'(t_0)\right]$. (ii) If f is gH-differentiable in type 2 at t_0 then $\left[f'_{2-gH}(t_0)\right]^{\alpha} = \left[(f^+_{\alpha})'(t_0), (f^-_{\alpha})'(t_0)\right]$.

Definition 3.3 ([25]). Assume that the α -cuts of a fuzzy-valued function f can be written in parametric form $[f(t)]^{\alpha} = [f_{\alpha}^{-}(t), f_{\alpha}^{+}(t)]$ for each $t \in [0, b], \alpha \in [0, 1]$ and $f_{\alpha}^{-}(t), f_{\alpha}^{+}(t)$ are measurable and Lebesgue integrable on the interval [0, b]. Then, the Lebesgue integral of f(t) is denoted by $\int_{0}^{b} f(t)dt$ whose α -cuts are given by

$$\left[\int_{0}^{b} f(t)dt\right]^{\alpha} = \left[\int_{0}^{b} f_{\alpha}^{-}(t)dt, \int_{0}^{b} f_{\alpha}^{+}(t)dt\right] \text{ for each } \alpha \in [0, 1].$$

The space of all Lebesgue integrable fuzzy-valued functions on [0, b] is denoted by $L^1([0, b], \mathscr{E})$ whose the properties used throughout this paper can be referred in [13,25,46].

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Definition 3.4 ([7,41]). The Riemann–Liouville fractional integral of order $\beta > 0$ of a fuzzy-valued function $f \in L^1([0, b], \mathscr{E})$ is defined by

$$\mathcal{I}^{\beta}_{+}f(t) := \frac{1}{\Gamma(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1} f(\tau) d\tau.$$

4. The Atangana-Baleanu fractional derivative and integral for fuzzy-valued functions

In this section, we introduce two interesting concepts: Riemann–Liouville Atangana–Baleanu fractional integral and Atangana–Baleanu fractional derivative of Caputo type for fuzzy-valued functions. Then, some characteristic properties of these fractional calculus concepts are also discussed.

Definition 4.1 ([8,9]). Let $f : [0,b] \to \mathbb{R}$ be a function of class $C^1([0,b],\mathbb{R})$. Then, Caputo Atangana–Baleanu fractional derivative of order $\beta \in (0, 1)$ of the real function f(t) is defined by

$${}^{abc}\mathbf{D}^{\beta}_{+}f(t) := \frac{\Phi(\beta)}{1-\beta} \int_{0}^{t} \mathbb{E}_{\beta} \left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'(\tau) d\tau,$$

where $\Phi(\beta)$ is a normalization function such that $\Phi(0) = \Phi(1) = 1$ and $\mathbb{E}_{\beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(k\beta + 1)}$ is the Mittag-Leffler

function (see [24]). The Laplace transform of Caputo Atangana–Baleanu fractional derivative ${}^{abc}D^{\beta}_{+}f(t)$ is

$$\mathscr{L}\left\{^{abc}\mathrm{D}^{\beta}_{+}f(t)\right\}(s) = \frac{\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left[s^{\beta}\mathscr{L}\left\{f(t)\right\}(s) - s^{\beta-1}f(0)\right].$$
(3)

Next, we will recall the concept of a new type of fractional integral associating to the Caputo Atangana–Baleanu fractional derivative with non-local kernel, namely Riemann–Liouville Atangana–Baleanu fractional integral.

Definition 4.2 ([8,9]). Let $f : [0,b] \subset \mathbb{R} \to \mathbb{R}$ be a function of class $L^1([0,b],\mathbb{R})$. Then, Riemann–Liouville Atangana–Baleanu fractional integral of order $\beta \in (0, 1)$ of the real function f(t) is defined by

$${}^{ab}\mathbf{I}^{\beta}_{+}f(t) := \frac{1-\beta}{\Phi(\beta)}f(t) + \frac{\beta}{\Phi(\beta)\Gamma(\beta)}\int_{0}^{t}(t-\tau)^{\beta-1}f(\tau)d\tau.$$

Next, we introduce the concept of Caputo Atangana-Baleanu fractional derivative for a fuzzy-valued function.

Definition 4.3. Assume that the function f(t) is of class $C^1([0, b], \mathscr{E})$. Then, Caputo Atangana–Baleanu fractional derivative of order $\beta \in (0, 1)$ of the function f(t) is defined by

$${}^{abc}\mathfrak{D}^{\beta}_{+}f(t) := \frac{\Phi(\beta)}{1-\beta} \int_{0}^{t} \mathbb{E}_{\beta} \left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{gH}(\tau) d\tau.$$

Remark 4.1. According to the properties in Proposition 8.23 of [13], Definition 4.3 implies that

$${}^{abc}\mathfrak{D}^{\beta}_{+}f(t) = \frac{\Phi(\beta)}{1-\beta} \int_{0}^{t} \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta(t-\tau)^{\beta}}{1-\beta}\right)^{k}}{\Gamma(k\beta+1)} f'_{gH}(\tau) d\tau$$
$$= \frac{\Phi(\beta)}{1-\beta} \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta}{1-\beta}\right)^{k}}{\Gamma(k\beta+1)} \int_{0}^{t} (t-\tau)^{k\beta} f'_{gH}(\tau) d\tau$$

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$$= \frac{\Phi(\beta)}{1-\beta} \sum_{k=0}^{\infty} \left(\frac{-\beta}{1-\beta}\right)^k \mathcal{I}_+^{k\beta+1} f'_{gH}(t).$$

Remark 4.2. The fuzzy Laplace transform for Caputo Atangana–Baleanu fractional derivative is given by

$$\tilde{\mathscr{L}}\left\{{}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s) = \begin{cases} \frac{\Phi(\beta)}{1-\beta} \frac{s^{\beta} \tilde{\mathscr{L}}\{f(t)\}(s) \ominus s^{\beta-1}f(0)}{s^{\beta} + \frac{\beta}{1-\beta}} & \text{if } f \text{ is gH-differentiable in type 1} \\ \frac{(-1)\Phi(\beta)}{1-\beta} \frac{s^{\beta-1}f(0) \ominus s^{\beta} \tilde{\mathscr{L}}\{f(t)\}(s)}{s^{\beta} + \frac{\beta}{1-\beta}} & \text{if } f \text{ is gH-differentiable in type 2.} \end{cases}$$

Proof. See Proposition 7.1 in Appendix section. \Box

Proposition 4.1. Assume that the fuzzy-valued function $f \in C^1([0, b], \mathscr{E})$ and its α -cuts are written in the parametric form $[f(t)]^{\alpha} = [f_{\alpha}^{-}(t), f_{\alpha}^{+}(t)]$ for each $t \in [0, b], \alpha \in [0, 1]$.

(i) If f is gH-differentiable in type 1 then $\begin{bmatrix} abc \mathfrak{D}^{\beta}_{+} f(t) \end{bmatrix}^{\alpha} = \begin{bmatrix} abc D^{\beta}_{+} f^{-}_{\alpha}(t), abc D^{\beta}_{+} f^{+}_{\alpha}(t) \end{bmatrix}$. (ii) If f is gH-differentiable in type 2 then $\begin{bmatrix} abc \mathfrak{D}^{\beta}_{+} f(t) \end{bmatrix}^{\alpha} = \begin{bmatrix} abc D^{\beta}_{+} f^{-}_{\alpha}(t), abc D^{\beta}_{+} f^{-}_{\alpha}(t) \end{bmatrix}$.

In order to illustrate the theoretical results, let us consider following example

Example 4.1. Let $A_1 = (0, 1, 2)$ be a triangular fuzzy number. Here, the fuzzy number A_1 represents for the concept "The number is near to 1". Then, let us consider $f : [0, \pi] \subset \mathbb{R} \to \mathscr{E}$ is a fuzzy-valued function defined by $f(t) = (t, t + \sin t, t + 2\sin t)$. Note that the function f(t) can be rewritten in the compact form

$$f(t) = A_1 \sin t + t.$$

In addition, the function f(t) is gH-differentiable in both type 1 and type 2 on $[0, \pi]$. Indeed,

(i) The function f(t) is gH-differentiable in type 1 on the interval $\left[0, \frac{\pi}{2}\right]$ and its gH-derivative is given by $f'_{1-gH}(t) = (1, \cos t + 1, 2\cos t + 1)$ whose level sets are

$$\left[f_{1-gH}'(t)\right]^{\alpha} = \left[1 + \alpha \cos t, 2\cos t + 1 - \alpha \cos t\right].$$

(ii) The function f(t) is gH-differentiable in type 2 on the interval $\left[\frac{\pi}{2}, \pi\right]$ and its gH-derivative is given by $f'_{2-gH}(t) = (2\cos t + 1, \cos t + 1, 1)$ whose level sets are

$$\left[f_{2-gH}'(t)\right]^{\alpha} = \left[2\cos t + 1 - \alpha\cos t, 1 + \alpha\cos t\right].$$

The plot of the function f(t) and its two types of gH-differentiability are shown in Fig. 3.

Now, we will calculate the Caputo Atangana–Baleanu fractional derivative of the fuzzy-valued function f(t) on the interval $[0, \pi]$. Firstly, for each $t \in [0, \frac{\pi}{2}]$, by using fractional calculus and some fundamental computation (see Table 9.1, [42]), we have

$${}^{abc}\mathfrak{D}^{\beta}_{+}f(t) = \frac{\Phi(\beta)}{1-\beta} \int_{0}^{t} \mathbb{E}_{\beta} \left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{gH}(\tau) d\tau = \frac{\Phi(\beta)}{1-\beta} \int_{0}^{t} \mathbb{E}_{\beta} \left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{1-gH}(\tau) d\tau$$

is a triangular fuzzy-valued function $(a_1, b_1, c_1) := (a_1(t), b_1(t), c_1(t))$ with three components

$$a_1 = \frac{t\Phi(\beta)}{1-\beta} \mathbb{E}_{\beta,2}\left[\frac{-\beta t^{\beta}}{1-\beta}\right],$$

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Fig. 3. The function f(t) and its gH-derivatives on the interval $[0, \pi]$.

$$b_{1} = \frac{\Phi(\beta)}{1-\beta} \left(t \mathbb{E}_{\beta,2} \left[\frac{-\beta t^{\beta}}{1-\beta} \right] + \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta t^{\beta}}{1-\beta} \right)^{k} \left[{}_{1}F_{1}\left(1; k\beta + 1; it \right) + {}_{1}F_{1}\left(1; k\beta + 1; -it \right) \right]}{2\Gamma(k\beta + 2)} \right),$$

$$c_{1} = \frac{\Phi(\beta)}{1-\beta} \left(t \mathbb{E}_{\beta,2} \left[\frac{-\beta t^{\beta}}{1-\beta} \right] + \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta t^{\beta}}{1-\beta} \right)^{k} \left[{}_{1}F_{1}\left(1; k\beta + 1; it \right) + {}_{1}F_{1}\left(1; k\beta + 1; -it \right) \right]}{\Gamma(k\beta + 2)} \right),$$

where the confluent hypergeometric Kummer function (see [24]) is defined by

$${}_{1}F_{1}(a;b;z) = \frac{\Gamma(b)}{\Gamma(b-a)\Gamma(a)} \int_{0}^{1} e^{zs} t^{a-1} (1-s)^{b-a-1} ds.$$

Next, for each $t \in \left[\frac{\pi}{2}, \pi\right]$, we have

$${}^{abc}\mathfrak{D}^{\beta}_{+}f(t) = \frac{\Phi(\beta)}{1-\beta} \left(\int\limits_{0}^{\frac{\pi}{2}} \mathbb{E}_{\beta} \left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{1-gH}(\tau) d\tau + \int\limits_{\frac{\pi}{2}}^{t} \mathbb{E}_{\beta} \left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{2-gH}(\tau) d\tau \right)$$

By similar arguments and computation, we directly get that the function ${}^{abc}\mathfrak{D}^{\beta}_+f(t)$ is also a triangular fuzzy-valued function of the form $(a_2, b_2, c_2) := (a_2(t), b_2(t), c_2(t))$, where

$$\begin{split} a_{2} &= \frac{\pi \mathbb{E}_{\beta,2} \left[\frac{-\beta \pi^{\beta}}{2^{\beta} (1-\beta)} \right]}{2} + \frac{(2t-\pi) \mathbb{E}_{\beta,2} \left[\frac{-\beta(2t-\pi)^{\beta}}{2^{\beta} (1-\beta)} \right]}{2} \\ &+ \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta(2t-\pi)^{\beta}}{2^{\beta} (1-\beta)} \right)^{k} \left[{}_{1}F_{1}\left(1; k\beta + 1; it \right) + {}_{1}F_{1}\left(1; k\beta + 1; -it \right) \right]}{\Gamma(k\beta + 2)}, \\ b_{2} &= \frac{\pi \mathbb{E}_{\beta,2} \left[\frac{-\beta \pi^{\beta}}{2^{\beta} (1-\beta)} \right]}{2} + \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta \pi^{\beta}}{2^{\beta} (1-\beta)} \right)^{k} \left[{}_{1}F_{1}\left(1; k\beta + 1; it \right) + {}_{1}F_{1}\left(1; k\beta + 1; -it \right) \right]}{2\Gamma(k\beta + 2)}, \end{split}$$

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$$+ \frac{(2t-\pi)\mathbb{E}_{\beta,2}\left[\frac{-\beta(2t-\pi)^{\beta}}{2^{\beta}(1-\beta)}\right]}{2} + \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta(2t-\pi)^{\beta}}{2^{\beta}(1-\beta)}\right)^{k} \left[{}_{1}F_{1}\left(1;k\beta+1;it\right) + {}_{1}F_{1}\left(1;k\beta+1;-it\right)\right]}{\Gamma(k\beta+2)},$$

$$c_{2} = \frac{\pi\mathbb{E}_{\beta,2}\left[\frac{-\beta\pi^{\beta}}{2^{\beta}(1-\beta)}\right]}{2} + \frac{(2t-\pi)\mathbb{E}_{\beta,2}\left[\frac{-\beta(2t-\pi)^{\beta}}{2^{\beta}(1-\beta)}\right]}{2} + \sum_{k=0}^{\infty} \frac{\left(\frac{-\beta\pi^{\beta}}{2^{\beta}(1-\beta)}\right)^{k} \left[{}_{1}F_{1}\left(1;k\beta+1;it\right) + {}_{1}F_{1}\left(1;k\beta+1;-it\right)\right]}{\Gamma(k\beta+2)}.$$

In addition, the concept of Riemann–Liouville Atangana–Baleanu fractional integral for fuzzy-valued functions associated to the Atangana–Baleanu fractional derivatives with non-local kernel is given as follows:

Definition 4.4. Let $f : [0, b] \subset \mathbb{R} \to \mathscr{E}$ be a fuzzy-valued function. Then, the Riemann–Liouville Atangana–Baleanu fractional integral of order $\beta \in (0, 1]$ of the function f(t) is defined by

$${}^{ab}\mathcal{I}^{\beta}_{+}f(t) := \frac{1-\beta}{\Phi(\beta)}f(t) + \frac{\beta}{\Phi(\beta)\Gamma(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1}f(\tau)d\tau = \frac{1-\beta}{\Phi(\beta)}f(t) + \frac{\beta}{\Phi(\beta)}\mathcal{I}^{\beta}_{+}f(t).$$

In addition, for each $\alpha \in [0, 1]$, α -cuts of ${}^{ab}\mathcal{I}^{\beta}_{+}f(t)$ are $\left[{}^{ab}\mathcal{I}^{\beta}_{+}f(t)\right]^{-} = \left[{}^{ab}I^{\beta}_{+}f^{-}_{\alpha}(t), {}^{ab}I^{\beta}_{+}f^{+}_{\alpha}(t)\right]$.

Remark 4.3. In some special case, the Riemann–Liouville Atangana–Baleanu fractional integral is identified with the well-known concepts.

(i) If $\beta = 0$, the Riemann–Liouville Atangana–Baleanu fractional integral is

$${}^{ab}\mathcal{I}^{0}_{+}f(t) = \frac{1-0}{\Phi(0)}f(t) + \frac{0}{\Phi(0)\Gamma(0)}\int_{0}^{t}(t-\tau)^{-1}f(\tau)d\tau = f(t).$$

(ii) If $\beta = 1$, the Riemann–Liouville Atangana–Baleanu fractional integral is

$${}^{ab}\mathcal{I}^{1}_{+}f(t) = \frac{1-1}{\Phi(1)}f(t) + \frac{1}{\Phi(1)\Gamma(1)}\int_{0}^{t}(t-\tau)^{1-1}f(\tau)d\tau = \int_{0}^{t}f(\tau)d\tau.$$

Example 4.2. Let $f : [0, T] \subset \mathbb{R} \to \mathscr{E}$ be a fuzzy-valued function given by $f(t) = (0, e^{\lambda t}, 2e^{\lambda t} + t^2)$, where λ and T are positive constants. Then, the Riemann–Liouville Atangana–Baleanu fractional integral of order $\beta \in (0, 1)$ of the function f(t) is given by

$${}^{ab}\mathcal{I}^{\beta}_{+}f(t) = \frac{1-\beta}{\Phi(\beta)}f(t) + \frac{\beta}{\Phi(\beta)}\mathcal{I}^{\beta}_{+}f(t).$$

Firstly, we compute the fractional integral $\mathcal{I}_{+}^{\beta}f(t) = (\mathcal{I}_{+}^{\beta}(0), \mathcal{I}_{+}^{\beta}(e^{\lambda t}), \mathcal{I}_{+}^{\beta}(2e^{\lambda t} + t^{2}))$, in which

$$\begin{aligned} \mathcal{I}^{\beta}_{+}(0) &= \int_{0}^{t} (t-\tau)^{\beta-1} 0 \, d\tau = 0, \\ \mathcal{I}^{\beta}_{+}(e^{\lambda t}) &= \int_{0}^{t} (t-\tau)^{\beta-1} e^{\lambda \tau} d\tau = t^{\beta} \mathbb{E}_{1,\beta+1}(\lambda t), \end{aligned}$$

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$$\mathcal{I}^{\beta}_{+}(2e^{\lambda t}+t^{2}) = \int_{0}^{t} (t-\tau)^{\beta-1} \left(2e^{\lambda \tau}+\tau^{2}\right) d\tau = 2t^{\beta} \mathbb{E}_{1,\beta+1}\left(\lambda t\right) + \frac{\Gamma(3)}{\Gamma(\beta+3)} t^{\beta+3-1}.$$

Hence, we obtain

$${}^{ab}\mathcal{I}^{\beta}_{+}f(t) = \frac{1-\beta}{\Phi(\beta)}\left(0, e^{\lambda t}, 2e^{\lambda t} + t^{2}\right) + \frac{\beta}{\Phi(\beta)}\left(0, t^{\beta}\mathbb{E}_{1,\beta+1}\left(\lambda t\right), 2t^{\beta}\mathbb{E}_{1,\beta+1}\left(\lambda t\right) + \frac{2}{\Gamma(\beta+3)}t^{\beta+2}\right).$$

Especially, if $\beta = 0$ then we have ${}^{ab}\mathcal{I}^0_+ f(t) = \frac{1}{\Phi(0)} \left(0, e^{\lambda t}, 2e^{\lambda t} + t^2\right) = f(t)$ while in case $\beta = 1$, we obtain

$${}^{ab}\mathcal{I}^{1}_{+}f(t) = \frac{1}{\Phi(1)} \left(0, t\mathbb{E}_{1,2}(\lambda t), 2t\mathbb{E}_{1,2}(\lambda t) + \frac{2}{\Gamma(4)}t^{3} \right)$$
$$= \left(0, \frac{t(e^{\lambda t} - 1)}{\lambda t}, \frac{2t(e^{\lambda t} - 1)}{\lambda t} + \frac{1}{3}t^{3} \right)$$
$$= \left(0, \frac{e^{\lambda t} - 1}{\lambda}, \frac{2(e^{\lambda t} - 1)}{\lambda} + \frac{1}{3}t^{3} \right)$$
$$= \int_{0}^{t} \left(0, e^{\lambda s}, 2e^{\lambda s} + s^{2} \right) ds,$$

that verify the statements (i) and (ii) in Remark 4.3.

It is well-known that Newton–Leibniz formula is a required element in the derivation of fractional calculus theory. Hence, a formula of Newton–Leibniz type for fuzzy Caputo Atangana–Baleanu fractional derivative may enable us to construct significant results for fractional calculus in the Atangana–Baleanu model, too.

Theorem 4.1. Let $\beta \in (0, 1)$ and $f : [0, T] \subset \mathbb{R} \to \mathscr{E}$ be a gH-differentiable fuzzy-valued function with no switching point on [0, T]. Then, Riemann–Liouville Atangana–Baleanu fractional integral and Caputo Atangana–Baleanu fractional derivative of the function f(t) satisfy the following Newton–Leibniz formula

$${}^{ab}\mathcal{I}^{\beta}_{+}\left({}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right) = f(t)\ominus_{gH}f(0), \qquad t\in[0,T].$$

Proof. Since the function f is gH-differentiable with no switching point on the interval [0, T], it implies that f is either gH-differentiable in type 1 or gH-differentiable in type 2 on this interval.

Case 1: If *f* is gH-differentiable in type 1 then for each $t \in [0, T]$ and $\alpha \in [0, 1]$, we have

$$\begin{bmatrix} ab \mathcal{I}^{\beta}_{+} \begin{pmatrix} abc \mathfrak{D}^{\beta}_{+} f(t) \end{pmatrix} \end{bmatrix}^{\alpha} = \begin{bmatrix} ab I^{\beta}_{+} \begin{pmatrix} abc D^{\beta}_{+} f^{-}_{\alpha}(t) \end{pmatrix}, ab I^{\beta}_{+} \begin{pmatrix} abc D^{\beta}_{+} f^{+}_{\alpha}(t) \end{pmatrix} \end{bmatrix}.$$

Here, by the definition of Riemann–Liouville Atangana–Baleanu fractional integral together with the series formula for Caputo Atangana–Baleanu fractional derivative, we have

$$\begin{split} {}^{ab}\mathrm{I}^{\beta}_{+} \left({}^{abc}\mathrm{D}^{\beta}_{+}f^{-}_{\alpha}(t) \right) &= \frac{1-\beta}{\Phi(\beta)}{}^{abc}\mathrm{D}^{\beta}_{+}f^{-}_{\alpha}(t) + \frac{\beta}{\Phi(\beta)}\mathrm{I}^{\beta}_{+} \left({}^{abc}\mathrm{D}^{\beta}_{+}f^{-}_{\alpha}(t) \right) \\ &= \sum_{k=0}^{\infty} \left(\frac{-\beta}{1-\beta} \right)^{k}\mathrm{I}^{k\beta+1}_{+} \left({}^{-}_{\alpha} \right)'(t) + \frac{\beta}{1-\beta}\mathrm{I}^{\beta}_{+} \left[\sum_{k=0}^{\infty} \left(\frac{-\beta}{1-\beta} \right)^{k}\mathrm{I}^{k\beta+1}_{+} \left({}^{-}_{\alpha} \right)'(t) \right] \\ &= \sum_{k=0}^{\infty} \left(\frac{-\beta}{1-\beta} \right)^{k}\mathrm{I}^{k\beta+1}_{+} \left({}^{-}_{\alpha} \right)'(t) - \left(\frac{-\beta}{1-\beta} \right) \sum_{k=0}^{\infty} \left(\frac{-\beta}{1-\beta} \right)^{k}\mathrm{I}^{(k+1)\beta+1}_{+} \left({}^{-}_{\alpha} \right)'(t) \\ &= \sum_{k=0}^{\infty} \left(\frac{-\beta}{1-\beta} \right)^{k}\mathrm{I}^{k\beta+1}_{+} \left({}^{-}_{\alpha} \right)'(t) - \sum_{k=0}^{\infty} \left(\frac{-\beta}{1-\beta} \right)^{k+1}\mathrm{I}^{(k+1)\beta+1}_{+} \left({}^{-}_{\alpha} \right)'(t) \\ &= \mathrm{I}^{1}_{+} \left({}^{-}_{\alpha} \right)'(t) = {}^{-}_{\alpha}(t) - {}^{-}_{\alpha}(0). \end{split}$$

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By doing similar arguments, we also receive ${}^{ab}I^{\beta}_{+}\left({}^{abc}D^{\beta}_{+}f^{+}_{\alpha}(t)\right) = f^{+}_{\alpha}(t) - f^{+}_{\alpha}(0)$. Hence, the level sets of the expression ${}^{ab}\mathcal{I}^{\beta}_{+}\left({}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right)$ are given by

$$\begin{bmatrix} ab \mathcal{I}^{\beta}_{+} \begin{pmatrix} abc \mathfrak{D}^{\beta}_{+} f(t) \end{pmatrix} \end{bmatrix}^{\alpha} = \begin{bmatrix} f^{-}_{\alpha}(t) - f^{-}_{\alpha}(0), f^{+}_{\alpha}(t) - f^{+}_{\alpha}(0) \end{bmatrix} \text{ for all } \alpha \in [0, 1],$$

which implies that

$${}^{ab}\mathcal{I}^{\beta}_{+}\left({}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right) = f(t)\ominus f(0). \tag{4}$$

Case 2: If *f* is gH-differentiable in type 2 then for each $t \in [0, T]$ and $\alpha \in [0, 1]$, we have

$$\begin{bmatrix} ab \mathcal{I}^{\beta}_{+} \left(abc \mathfrak{D}^{\beta}_{+} f(t) \right) \end{bmatrix}^{\alpha} = \begin{bmatrix} ab \mathbf{I}^{\beta}_{+} \left(abc \mathbf{D}^{\beta}_{+} f^{+}_{\alpha}(t) \right), ab \mathbf{I}^{\beta}_{+} \left(abc \mathbf{D}^{\beta}_{+} f^{-}_{\alpha}(t) \right) \end{bmatrix}.$$

By similar computation as in Case 1, we also receive

$${}^{ab}\mathbf{I}^{\beta}_{+} \left({}^{abc}\mathbf{D}^{\beta}_{+}f^{-}_{\alpha}(t)\right) = f^{-}_{\alpha}(t) - f^{-}_{\alpha}(0)$$

$${}^{ab}\mathbf{I}^{\beta}_{+} \left({}^{abc}\mathbf{D}^{\beta}_{+}f^{+}_{\alpha}(t)\right) = f^{+}_{\alpha}(t) - f^{+}_{\alpha}(0),$$
implies that
$$\left[{}^{ab}\mathcal{I}^{\beta}_{+} \left({}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right)\right]^{\alpha} = (-1)\left[f^{-}_{\alpha}(0) - f^{-}_{\alpha}(t), f^{+}_{\alpha}(0) - f^{+}_{\alpha}(t)\right]$$
 for all $\alpha \in [0, 1]$. Therefore, we

which implies that directly obtain

$${}^{ab}\mathcal{I}^{\beta}_{+}\left({}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right) = (-1)\left(f(0)\ominus f(t)\right).$$
(5)

By combining equations (4) and (5), we receive ${}^{ab}\mathcal{I}^{\beta}_{+}\left({}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right) = f(t)\ominus_{gH}f(0)$, which completes the proof. \Box

5. The existence and uniqueness of mild solution for fuzzy initial value problem (FIVP) under Caputo Atangana–Baleanu fractional gH-differentiability

Next, we investigate the existence and uniqueness of mild solution of the following fuzzy initial value problem to fuzzy fractional differential system under the Caputo Atangana–Baleanu fractional gH-differentiability

$$\begin{cases} a^{bc} \mathfrak{D}^{\beta}_{+} x(t) &= \mathbf{F}(t, x(t)), \quad t \in J = [0, T] \\ x(0) &= x_{0}, \end{cases}$$
(6)

where ${}^{abc}\mathfrak{D}^{\beta}_+x(t)$ denotes for Caputo Atangana–Baleanu fractional derivative of state vector x(t), initial condition $x_0 \in \mathscr{E}^n$ and $F : [0, T] \times \mathscr{E}^n \to \mathscr{E}^n$ is a fuzzy vector-valued function that satisfies following assumptions:

- (**HF1**) The fuzzy vector-valued function $F(\cdot, \xi) : [0, T] \to \mathscr{E}^n$ is strongly measurable for each $\xi \in \mathscr{E}^n$ and the function $F(t, \cdot) : \mathscr{E}^n \to \mathscr{E}^n$ is continuous for a.e. $t \in [0, T]$;
- (**HF2**) There exists a matrix M_0 such that $\mathbb{D}_n(F(t,\xi),\overline{0}) \le M_0 \mathbb{D}_n(\xi,\overline{0})$ for all $\xi \in \mathscr{E}^n$.
- (**HF3**) There exists a matrix M_1 such that $\mathbb{D}_n(F(t,\xi), F(t,\overline{\xi})) \leq M_1 \mathbb{D}_n(\xi,\overline{\xi})$ for all $\xi, \overline{\xi} \in \mathscr{E}^n$.

In the following, we recall from [28] the space of all continuous fuzzy vector-valued functions on [0, T].

Definition 5.1 ([28]). Denote $C([0, T], \mathscr{E}^n) = \{\varphi : [0, T] \to \mathscr{E}^n : \varphi(t) \text{ is continuous on } [0, T]\}$. According to Long et al. [28], it is well-known that the space $C([0, T], \mathscr{E}^n)$ is a generalized complete metric space endowed with the generalized weighted metric

$$\mathcal{H}_{\lambda}(\varphi,\psi) = \sup_{[0,T]} \left\{ \mathbb{D}_{n}(\varphi(t),\psi(t))e^{-\lambda t} \right\},\,$$

for all $\varphi, \psi \in C([0, T], \mathscr{E}^n)$ and $\lambda > 0$ is big enough parameter that will be specified later.

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In this section, for simplicity in representation, we assume that all components of the fuzzy vector-valued function x(t) have the same type of gH-differentiability with no switching point in whole time domain J = [0, T]. The following theorem plays a key role in defining the mild solution of (FIVP).

Theorem 5.1. Assume that $x \in C([0, T], \mathcal{E}^n)$ satisfies the fractional differential system (6).

(i) If x is gH-differentiable in type 1 then it satisfies following integral equation

$$x(t) = x_0 + \frac{1-\beta}{\Phi(\beta)} F(t, x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} F(\tau, x(\tau)) d\tau.$$
(7)

(ii) If x is gH-differentiable in type 2 then it satisfies following integral equation

$$x(t) = x_0 \ominus (-1) \left[\frac{1-\beta}{\Phi(\beta)} F(t, x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} F(\tau, x(\tau)) d\tau \right].$$
(8)

Proof. For each $t \in [0, T]$, by applying fuzzy Laplace transform to both sides of the fractional fuzzy differential system of (6), we have

$$\tilde{\mathscr{L}}\left\{{}^{abc}\mathfrak{D}^{\beta}_{+}x(t)\right\}(s) = \tilde{\mathscr{L}}\left\{\mathsf{F}(t,x(t))\right\}(s).$$
⁽⁹⁾

Then, by using Proposition 7.1, the above equation can be rewritten in two following forms depending on the types of gH-differentiability of the fuzzy-valued function x(t).

Case 1: If x is gH-differentiable in type 1 on [0, T] then (9) becomes

$$\frac{\Phi(\beta)}{1-\beta}\frac{s^{\beta}\tilde{\mathscr{L}}\{x(t)\}(s)\ominus s^{\beta-1}x(0)}{s^{\beta}+\frac{\beta}{1-\beta}}=\tilde{\mathscr{L}}\{\mathsf{F}(t,x(t))\}(s).$$

Thus, we obtain

$$s^{\beta} \tilde{\mathscr{L}} \{x(t)\}(s) \ominus s^{\beta-1} x_{0} = \frac{(1-\beta)s^{\beta}+\beta}{\Phi(\beta)} \tilde{\mathscr{L}} \{F(t,x(t))\}(s)$$

$$\Leftrightarrow \tilde{\mathscr{L}} \{x(t)\}(s) = \frac{1}{s} x_{0} + \frac{(1-\beta)s^{\beta}+\beta}{s^{\beta}\Phi(\beta)} \tilde{\mathscr{L}} \{F(t,x(t))\}(s)$$

$$\Leftrightarrow \tilde{\mathscr{L}} \{x(t)\}(s) = \frac{1}{s} x_{0} + \frac{(1-\beta)}{\Phi(\beta)} \tilde{\mathscr{L}} \{F(t,x(t))\}(s) + \frac{\beta}{s^{\beta}\Phi(\beta)} \tilde{\mathscr{L}} \{F(t,x(t))\}(s)$$

$$\Leftrightarrow \tilde{\mathscr{L}} \{x(t)\}(s) = \frac{1}{s} x_{0} + \frac{(1-\beta)}{\Phi(\beta)} \tilde{\mathscr{L}} \{F(t,x(t))\}(s) + \frac{\beta}{\Phi(\beta)} \tilde{\mathscr{L}} \{F(t,x(t))\}(s)$$

Then, the convolution theorem of Laplace transform can be applied to obtain

$$\tilde{\mathscr{L}}\{x(t)\}(s) = \frac{1}{s}x_0 + \frac{(1-\beta)}{\Phi(\beta)}\tilde{\mathscr{L}}\{F(t,x(t))\}(s) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\tilde{\mathscr{L}}\left\{\int_0^t (t-\tau)^{\beta-1}F(\tau,x(\tau))d\tau\right\}(s).$$

Finally, by applying inverse Laplace transform, it yields

$$x(t) = x_0 + \frac{(1-\beta)}{\Phi(\beta)} \mathbf{F}(t, x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} \mathbf{F}(\tau, x(\tau)) d\tau.$$

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Case 2: If x is gH-differentiable in type 2 on [0, T] then (9) becomes

$$\frac{(-1)\Phi(\beta)}{1-\beta}\frac{s^{\beta-1}x(0)\ominus s^{\beta}\tilde{\mathscr{L}}\{x(t)\}(s)}{s^{\beta}+\frac{\beta}{1-\beta}}=\tilde{\mathscr{L}}\left\{\mathsf{F}(t,x(t))\right\}(s).$$

The above equality is equivalent to

$$s^{\beta-1}x(0) \ominus s^{\beta}\tilde{\mathscr{L}}\{x(t)\}(s) = \frac{(1-\beta)s^{\beta}+\beta}{\Phi(\beta)}\tilde{\mathscr{L}}\{F(t,x(t))\}(s)$$

$$\Leftrightarrow s^{\beta}\tilde{\mathscr{L}}\{x(t)\}(s) = s^{\beta-1}x_{0} \ominus \frac{(1-\beta)s^{\beta}+\beta}{\Phi(\beta)}\tilde{\mathscr{L}}\{F(t,x(t))\}(s)$$

$$\Leftrightarrow \tilde{\mathscr{L}}\{x(t)\}(s) = \frac{1}{s}x_{0} \ominus \frac{(1-\beta)s^{\beta}+\beta}{\Phi(\beta)s^{\beta}}\tilde{\mathscr{L}}\{F(t,x(t))\}(s)$$

$$\Leftrightarrow \tilde{\mathscr{L}}\{x(t)\}(s) = \frac{1}{s}x_{0} \ominus \left[\frac{(1-\beta)}{\Phi(\beta)}\tilde{\mathscr{L}}\{F(t,x(t))\}(s) + \frac{\beta}{\Phi(\beta)s^{\beta}}\tilde{\mathscr{L}}\{F(t,x(t))\}(s)\right].$$

Next, by applying the convolution theorem of Laplace transform, we obtain

$$\tilde{\mathscr{L}}\{x(t)\}(s) = \frac{1}{s}x_0 \ominus \left[\frac{(1-\beta)}{\Phi(\beta)}\tilde{\mathscr{L}}\{F(t,x(t))\}(s) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\tilde{\mathscr{L}}\left\{\int_0^t (t-\tau)^{\beta-1}F(\tau,x(\tau))d\tau\right\}(s)\right].$$

Then, the inverse Laplace transform is applied to show that if x is gH-differentiable in type 2 then

$$x(t) = x_0 \ominus \left[\frac{(1-\beta)}{\Phi(\beta)} F(t, x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} F(\tau, x(\tau)) d\tau \right]$$

Hence, the proof is completed. \Box

Remark 5.1. Based on the concept of generalized Hukuhara difference, two integral equations (7) and (8) can be rewritten in following form

$$x(t) \ominus_{gH} x_0 = \frac{1-\beta}{\Phi(\beta)} F(t, x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} F(\tau, x(\tau)) d\tau.$$

Now, we introduce a suitable concept of mild solutions of (FIVP).

Definition 5.2.

- (i) A continuous fuzzy vector-valued function $x : [0, T] \subset \mathbb{R} \to \mathscr{E}^n$ is said to be a mild solution of type (i) of (FIVP) if it satisfies the integral equation (7).
- (ii) A continuous fuzzy vector-valued function $x : [0, T] \subset \mathbb{R} \to \mathscr{E}^n$ is said to be a mild solution of type (ii) of (FIVP) if it satisfies the integral equation (8).

Next, the existence and uniqueness of mild solution in type 1 of the problem (6) is presented.

Theorem 5.2. Under the assumptions (**HF1**), (**HF2**) and (**HF3**), the fuzzy initial value problem (6) has exactly one mild solution in type 1 defined on [0, T] provided that the spectral radii of the matrices $\frac{(1-\beta)}{\Phi(\beta)}M_0$ and $\frac{(1-\beta)}{\Phi(\beta)}M_1$ are all less than 1.

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Proof. Define an operator $\mathscr{P}: C([0,T], \mathscr{E}^n) \to C([0,T], \mathscr{E}^n)$ by

$$\mathscr{P}[x](t) = x_0 + \frac{1-\beta}{\Phi(\beta)} F(t, x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} F(\tau, x(\tau)) d\tau, \quad t \in [0, T].$$

It is easy to see that \mathscr{P} is a linear operator mapping $C([0, T], \mathscr{E}^n)$ into itself. Then, the unique existence of mild solution in type 1 of (FIVP) is equivalent to the fixed point problem for the operator \mathscr{P} .

Firstly, since the spectral radius of the matrix $\frac{(1-\beta)}{\Phi(\beta)}M_0$ is less than 1, it implies that the matrix $\frac{(1-\beta)}{\Phi(\beta)}M_0$ also converges to zero matrix. In addition, since the elements of matrix $\frac{G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}M_0$ can be as small as desired, the matrix $\frac{(1-\beta)\Gamma(\beta)+G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}M_0$ also converges to zero matrix.

Now, let us denote
$$r = \left[I_n - \frac{(1-\beta)\Gamma(\beta) + G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}M_0\right]^{-1} \mathbb{D}_n(x_0,\overline{0})$$
 and

$$\Omega_r = \left\{ x \in C\left([0, T], \mathscr{E}^n\right) : \mathcal{H}_{\lambda}\left(x, \overline{0}\right) \leq r \right\},\$$

where I_n is the unit matrix of order *n*. Now, we proceed the proof by following steps:

Step 1. The operator \mathscr{P} satisfies $\mathscr{P}(\Omega_r) \subseteq \Omega_r$. Indeed, for $t \in [0, T]$ and $x \in \Omega_r$, we have

$$\begin{split} \mathbb{D}_{n}(\mathscr{P}[x](t),\overline{0}) &= \mathbb{D}_{n}\left(x_{0} + \frac{1-\beta}{\Phi(\beta)}\mathrm{F}(t,x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\int_{0}^{t}(t-\tau)^{\beta-1}\mathrm{F}(\tau,x(\tau))d\tau,\overline{0}\right) \\ &\leq \mathbb{D}_{n}\left(x_{0},\overline{0}\right) + \frac{1-\beta}{\Phi(\beta)}\mathbb{D}_{n}\left(\mathrm{F}(t,x(t)),\overline{0}\right) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\int_{0}^{t}(t-\tau)^{\beta-1}\mathbb{D}_{n}\left(\mathrm{F}(\tau,x(\tau)),\overline{0}\right)d\tau \\ &\leq \mathbb{D}_{n}\left(x_{0},\overline{0}\right) + \frac{1-\beta}{\Phi(\beta)}\mathrm{M}_{0}\mathbb{D}_{n}\left(x(t),\overline{0}\right) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\int_{0}^{t}(t-\tau)^{\beta-1}\mathrm{M}_{0}\mathbb{D}_{n}\left(x(\tau),\overline{0}\right)e^{-\lambda\tau}e^{\lambda\tau}d\tau \\ &\leq \mathbb{D}_{n}\left(x_{0},\overline{0}\right) + \frac{1-\beta}{\Phi(\beta)}\mathrm{M}_{0}\mathcal{H}_{\lambda}\left(x,\overline{0}\right)e^{\lambda t} + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\left(\int_{0}^{t}(t-\tau)^{\beta-1}e^{\lambda\tau}d\tau\right)\mathrm{M}_{0}\mathcal{H}_{\lambda}\left(x,\overline{0}\right) \\ &< \mathbb{D}_{n}\left(x_{0},\overline{0}\right) + \frac{1-\beta}{\Phi(\beta)}\mathrm{M}_{0}\mathcal{H}_{\lambda}\left(x,\overline{0}\right)e^{\lambda t} + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\left(\frac{2}{\lambda^{\frac{\beta}{4}}} + \frac{1}{\lambda^{1+\frac{\beta}{2}}}\right)\mathrm{M}_{0}\mathcal{H}_{\lambda}\left(x,\overline{0}\right). \end{split}$$

Here, we use the estimation $\int_{0}^{t} (t-\tau)^{\beta-1} e^{\lambda \tau} d\tau < \frac{e^{\lambda t}}{\beta} G(\lambda,\beta), \text{ where } \lambda > 0, G(\lambda,\beta) = \left(\frac{2}{\lambda^{\frac{\beta}{4}}} + \frac{1}{\lambda^{1+\frac{\beta}{2}}}\right) \text{ and } t \in [0,T]$

(see Lemma 7.1 in [28]). Next, by dividing both sides by $e^{\lambda t}$ and taking supremum for $t \in [0, T]$, we receive

$$\begin{aligned} \mathcal{H}_{\lambda}\left(\mathscr{P}[x],\overline{0}\right) &< \mathbb{D}_{n}\left(x_{0},\overline{0}\right) + \left[\frac{1-\beta}{\Phi(\beta)} + \frac{G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}\right] \mathcal{M}_{0}\mathcal{H}_{\lambda}\left(x,\overline{0}\right) \\ &\leq \mathbb{D}_{n}\left(x_{0},\overline{0}\right) + \frac{(1-\beta)\Gamma(\beta) + G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}\mathcal{M}_{0}r \leq r, \end{aligned}$$

which means that the operator \mathscr{P} maps Ω_r into itself.

Moreover, the operator $\mathscr{P} : \Omega_r \to \Omega_r$ is a continuous operator. Indeed, assume that $\{x_n\} \subset \Omega_r$ is a sequence such that $x_n \rightrightarrows x \in \Omega_r$. Here, for each $t \in [0, T]$, we have

$$\mathscr{P}[x_n](t) = x_0 + \frac{1-\beta}{\Phi(\beta)} F(t, x_n(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} F(\tau, x_n(\tau)) d\tau.$$

For all $0 \le s \le t \le T$, the hypothesis (**HF1**) implies that

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$$(t-\tau)^{\beta-1}\mathbf{F}(\tau,x_n(\tau)) \to (t-\tau)^{\beta-1}\mathbf{F}(\tau,x(\tau)) \quad \text{as } n \to \infty.$$

Next, by employing hypotheses (HF1) and Lebesgue's dominated theorem, we have

$$\mathbb{D}_{n}\left(\mathscr{P}[x_{n}](t),\mathscr{P}[x](t)\right) \leq \frac{1-\beta}{\Phi(\beta)} \mathbb{D}_{n}\left(\mathrm{F}(t,x_{n}(t)),\mathrm{F}(t,x(t))\right) \\ + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-s)^{\beta-1} \mathbb{D}_{n}\left(\mathrm{F}(\tau,x_{n}(\tau)),\mathrm{F}(\tau,x(\tau))\right) d\tau$$

tends to $\vec{0} \in \mathbb{R}^n$ as $n \to \infty$, which follows that the operator \mathscr{P} is continuous on Ω_r .

Step 2. The operator \mathscr{P} is a contraction. For this aim, let $x, \overline{x} \in \Omega_r$ be arbitrary. Then, it suffices to show that there exists a convergent to zero matrix \mathcal{M} such that

 $\mathcal{H}_{\lambda}\left(\mathscr{P}[x], \mathscr{P}[\overline{x}]\right) < \mathcal{M}\mathcal{H}_{\lambda}\left(x, \overline{x}\right).$

Indeed, for each $t \in [0, T]$, we have

$$\begin{split} \mathbb{D}_{n}\left(\mathscr{P}[x](t),\mathscr{P}[\overline{x}](t)\right) \\ &\leq \frac{1-\beta}{\Phi(\beta)}\mathbb{D}_{n}\left(\mathrm{F}(t,x(t)),\mathrm{F}(t,\overline{x}(t))\right) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1}\mathbb{D}_{n}\left(\mathrm{F}(\tau,x(\tau)),\mathrm{F}(\tau,\overline{x}(\tau))\right)d\tau \\ &\leq \frac{1-\beta}{\Phi(\beta)}\mathrm{M}_{1}\mathbb{D}_{n}\left(x(t),\overline{x}(t)\right) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1}\mathrm{M}_{1}\mathbb{D}_{n}\left(x(\tau),\overline{x}(\tau)\right)d\tau \\ &= \frac{1-\beta}{\Phi(\beta)}\mathrm{M}_{1}\mathbb{D}_{n}\left(x(t),\overline{x}(t)\right)e^{-\lambda t}e^{\lambda t} + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1}\mathrm{M}_{1}\mathbb{D}_{n}\left(x(\tau),\overline{x}(\tau)\right)e^{-\lambda \tau}e^{\lambda \tau}d\tau \\ &\leq \frac{1-\beta}{\Phi(\beta)}\mathrm{M}_{1}\mathcal{H}_{\lambda}\left(x,\overline{x}\right)e^{\lambda t} + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\left(\int_{0}^{t} (t-\tau)^{\beta-1}e^{\lambda \tau}d\tau\right)\mathrm{M}_{1}\mathcal{H}_{\lambda}\left(x,\overline{x}\right) \\ &< \frac{1-\beta}{\Phi(\beta)}\mathrm{M}_{1}\mathcal{H}_{\lambda}\left(x,\overline{x}\right)e^{\lambda t} + \frac{e^{\lambda t}G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}\mathrm{M}_{1}\mathcal{H}_{\lambda}\left(x,\overline{x}\right). \end{split}$$

Then, by dividing both sides by $e^{\lambda t}$ and taking supremum for $t \in [0, T]$, we immediately obtain

$$\mathcal{H}_{\lambda}\left(\mathscr{P}[x],\mathscr{P}[\overline{x}]\right) < \frac{(1-\beta)\Gamma(\beta) + G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)} \mathbf{M}_{1}\mathcal{H}_{\lambda}\left(x,\overline{x}\right).$$

Since the assumption that the spectral radius of the matrix $\frac{(1-\beta)}{\Phi(\beta)}M_1$ is less than 1 and the elements of $\frac{G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}M_1$ can be as small as desired, we directly deduce that the matrix

$$\frac{(1-\beta)\Gamma(\beta) + G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}\mathbf{M}_{1} = \frac{(1-\beta)}{\Phi(\beta)}\mathbf{M}_{1} + \frac{G(\lambda,\beta)}{\Gamma(\beta)\Phi(\beta)}\mathbf{M}_{1}$$

converges to zero matrix.

Hence, it follows that the operator \mathscr{P} is a generalized contraction. Finally, by applying contraction principle, we can conclude that the operator \mathscr{P} has a unique fixed point $x^* \in \Omega_r$ that is the unique mild solution of the fuzzy initial value problem (6). \Box

The rest of this section is devoted to prove the existence and uniqueness of mild solution in type 2 of (FIVP). For each $x \in C([0, T], \mathscr{E}^n)$, we define

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$$\mathscr{F}[x](t) = x_0 \ominus \left[\frac{(1-\beta)}{\Phi(\beta)} F(t, x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} F(\tau, x(\tau)) d\tau \right].$$
(10)

Denote $\hat{C}([0, T], \mathscr{E}^n) = \{x \in C([0, T], \mathscr{E}^n) : \text{the equality (10) holds for all } t \in [0, T]\}$. By doing similar arguments as in [28], we can also prove that $(\hat{C}([0, T], \mathscr{E}^n), \mathbb{D}_n)$ is also a complete metric space.

Theorem 5.3. Assume that:

- (i) The hypotheses (HF1), (HF2) and (HF3) are fulfilled.
- (ii) The set $\hat{C}([0, T], \mathscr{E}^n) \neq \emptyset$.
- (iii) The spectral radii of the matrices $\frac{(1-\beta)}{\Phi(\beta)}M_0$ and $\frac{(1-\beta)}{\Phi(\beta)}M_1$ are all less than 1.

Then, the fuzzy initial value problem (6) has exactly one mild solution in type 2 defined on [0, T].

Proof. By doing similar arguments as in Theorem 5.2, the existence and uniqueness of mild solution in type 2 of (FIVP) is equivalent to the solvability of the functional equation $\mathscr{F}[x] = x$. Hence, it suffices to show that the operator \mathscr{F} has a unique fixed point $x^* \in \hat{C}([0, T], \mathscr{E}^n)$. Adapting to Theorem 5.2, we directly obtain the operator \mathscr{F} is a contraction mapping and hence, it has a unique fixed point x^* in $\hat{C}([0, T], \mathscr{E}^n)$, that is the unique mild solution in type 2 of (FIVP). \Box

Remark 5.2. In this section, we only need to consider that all components of fuzzy mild solution x(t) have the same type of gH-differentiability without switching point on the time domain J = [0, T]. If components of the fuzzy mild solution $x(t) = (x_1(t), \ldots, x_n(t))^{\top}$ have different types of gH-differentiability on J, the existence and uniqueness result of Theorem 5.2 and 5.3 are still guaranteed. Indeed, no loss generality, we assume that the fuzzy-valued functions $x_1(t), x_2(t), \ldots, x_p(t)$ are gH-differentiable in type 1 and the fuzzy-valued functions $x_{p+1}(t), x_{p+2}(t), \ldots, x_n(t)$ are gH-differentiable in type 2. Then, we define the solution operator $\mathscr{P}[x]$ as a vector-valued function $\mathscr{P}[x] = (\mathscr{P}_1[x], \ldots, \mathscr{P}_n[x])^{\top}$, where

$$\mathscr{P}_{i}[x](t) = \begin{cases} x_{0}^{i} + \frac{1-\beta}{\Phi(\beta)} F_{i}(t,x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1} F_{i}(\tau,x(\tau)) d\tau & \text{for } i = 1, \dots, p \\ \\ x_{0}^{i} \ominus \left[\frac{(1-\beta)}{\Phi(\beta)} F_{i}(t,x(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1} F_{i}(\tau,x(\tau)) d\tau \right] & \text{for } i = p+1,\dots,n. \end{cases}$$

Since the inequalities $d_{\infty}(u + v, w + e) \le d_{\infty}(u, w) + d_{\infty}(v, e)$ and $d_{\infty}(u \ominus v, w \ominus e) \le d_{\infty}(u, w) + d_{\infty}(v, e)$ are fulfilled for all $u, v, w, e \in \mathscr{E}$ then for all $i = \overline{1, n}$, we always get

$$d_{\infty}\left(\mathscr{P}_{i}[x](t)\right) \leq d_{\infty}\left(x_{0}^{i},\hat{0}\right) + \frac{1-\beta}{\Phi(\beta)}d_{\infty}\left(\mathsf{F}_{i}(t,x(t)),\hat{0}\right) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)}\int_{0}^{t}(t-\tau)^{\beta-1}d_{\infty}\left(\mathsf{F}_{i}(\tau,x(\tau)),\hat{0}\right)d\tau.$$

Hence, it implies that the proof of Theorem 5.2 and 5.3 are still true. However, in this case, we cannot conclude that the obtained solution is a mild fuzzy solution of type 1 or type 2.

6. Numerical solution of the fuzzy fractional SIQR model for the attacking of viruses in WSNs

In Section 2, we proposed a fuzzy initial value problem for the fuzzy fractional SIQR model (1) of virus propagation with the initial conditions (2). Now, we propose a novel algorithm for numerically solving the fuzzy initial value problem (1)–(2) using the Caputo Atangana–Baleanu fractional derivative. Here, for simplicity, we denote $X(t) = (S(t) \ I(t) \ Q(t) \ R(t))^{t}$, $X_0 = (S_0 \ I_0 \ Q_0 \ R_0)^{t} \in \mathcal{E}^4$. Then, the problem (1)–(2) can be rewritten in following compact form

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$$\begin{cases} abc \mathfrak{D}^{\beta}_{+} X(t) &= \mathbf{G}(t, X(t)) \\ X(0) &= X_{0}. \end{cases}$$
(11)

Here, the fuzzy vector function G(t, X(t)), given by

$$G(t, X(t)) = \begin{pmatrix} G_1(t, X(t)) & G_2(t, X(t)) & G_3(t, X(t)) & G_4(t, X(t)) \end{pmatrix}$$

satisfies all hypotheses (HF1), (HF2) and (HF3). Based on Theorem 5.1, mild solution of the initial value problem (11) can be given in one of following forms

$$X(t) = X_0 + \frac{1-\beta}{\Phi(\beta)}G(t, X(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1}G(\tau, X(\tau))d\tau,$$
(12)

$$X(t) = X_0 \ominus (-1) \left[\frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t, X(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_0^t (t-\tau)^{\beta-1} \mathbf{G}(\tau, X(\tau)) d\tau \right],$$
(13)

where \ominus is well-known Hukuhara difference and the function $\Phi(\beta) = 1 - \beta + \frac{\beta}{\Gamma(\beta)}$ satisfies $\Phi(0) = \Phi(1) = 1$.

6.1. Numerical solution of the fuzzy fractional initial value problem (1)–(2)

In the following, we develop a numerical method to numerically solve the integral equation (12). After that, a similar method will be also applied to obtain the numerical scheme for numerically solving the integral solution (13). First of all, we consider a uniform grid

$$\Pi = \{t_k = kh : k = 0, 1, \dots, N\},\$$

where N is positive integer such that $h = \frac{T}{N}$.

For each $\alpha \in [0, 1]$, the α -cuts form of the expression (12) is given by

$$\begin{split} X_{\alpha}^{-}(t) &= X_{0,\alpha}^{-} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t, X_{\alpha}^{-}(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1} \mathbf{G}(\tau, X_{\alpha}^{-}(\tau)) d\tau, \\ X_{\alpha}^{+}(t) &= X_{0,\alpha}^{+} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t, X_{\alpha}^{+}(t)) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \int_{0}^{t} (t-\tau)^{\beta-1} \mathbf{G}(\tau, X_{\alpha}^{+}(\tau)) d\tau. \end{split}$$

Then, based on the combination of the two-step Lagrange polynomial and the fundamental theorem of fractional calculus, the iterative scheme to implement the numerical solution in type 1 of the fuzzy fractional initial value problem (11) is given as follows:

For each n = 0, 1, 2, ..., denote $X_{\alpha,n}^- = X_{\alpha}^-(t_n), X_{\alpha,n}^+ = X_{\alpha}^+(t_n)$. Then, the terms $X_{\alpha,n+1}^-$ and $X_{\alpha,n+1}^+$ can be determined by approximating following integral equality

$$X_{\alpha}^{-}(t_{n+1}) = X_{0,\alpha}^{-} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_n, X_{\alpha,n}^{-}) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \sum_{k=0}^{n} \int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta-1} \mathbf{G}(\tau, X_{\alpha}^{-}(\tau)) d\tau,$$
(14)

$$X_{\alpha}^{+}(t_{n+1}) = X_{0,\alpha}^{+} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_{n}, X_{\alpha,n}^{+}) + \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \sum_{k=0}^{n} \int_{t_{k}}^{t_{k+1}} (t_{n+1} - \tau)^{\beta-1} \mathbf{G}(\tau, X_{\alpha}^{+}(\tau)) d\tau.$$
(15)

Next, we estimate the integrals on the right-hand sides of (14) and (15) over the interval $[t_k, t_{k+1}]$ by using the two-step Lagrange interpolation polynomial

$$G(\tau, X_{\alpha}^{*}(\tau)) \cong \frac{G(t_{k+1}, X_{\alpha,k+1}^{*})}{h}(\tau - t_{k}) - \frac{G(t_{k}, X_{\alpha,k}^{*})}{h}(\tau - t_{k+1})$$

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where "*" denotes for the signs "+" or "-". Then, by applying above approximation, we have

$$\begin{split} &\int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta - 1} \mathcal{G}(\tau, X_{\alpha}^*(\tau)) d\tau \\ &\cong \frac{\mathcal{G}(t_{k+1}, X_{\alpha,k+1}^*)}{h} \int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta - 1} (\tau - t_k) d\tau - \frac{\mathcal{G}(t_k, X_{\alpha,k}^*)}{h} \int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta - 1} (\tau - t_{k+1}) d\tau. \end{split}$$

By changing variable $s = t_{n+1} - \tau$, we receive

$$\int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta - 1} (\tau - t_k) d\tau = \frac{h^{\beta + 1}}{\beta(\beta + 1)} \Big[(n + 1 - k)^{\beta + 1} - (n - k)^{\beta} (n + 1 - k + \beta) \Big]$$

$$\int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta - 1} (\tau - t_{k+1}) d\tau = \frac{h^{\beta + 1}}{\beta(\beta + 1)} \Big[(n - k)^{\beta + 1} + (n - k + 1)^{\beta} (n - k + \beta) \Big].$$

Hence, it yields

$$\int_{t_{k}}^{t_{k+1}} (t_{n+1} - \tau)^{\beta - 1} \mathbf{G}(\tau, X_{\alpha}^{*}(\tau)) d\tau \cong \frac{h^{\beta} \mathbf{G}(t_{k+1}, X_{\alpha,k+1}^{*})}{\beta(\beta + 1)} \left[(n+1-k)^{\beta + 1} - (n-k)^{\beta}(n+1-k+\beta) \right] - \frac{h^{\beta} \mathbf{G}(t_{k}, X_{\alpha,k}^{*})}{\beta(\beta + 1)} \left[(n-k)^{\beta + 1} + (n-k+1)^{\beta}(n-k+\beta) \right].$$
(16)

By replacing the integral terms on the right-hand sides of integral equations (14)–(15) by the estimation (16), we immediately obtain the approximate solution

$$X_{\alpha,n+1}^{-} = X_{0,\alpha}^{-} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_n, X_{\alpha,n}^{-}) + h^{\beta} \sum_{k=0}^{n} \left[C_{k,n}^{\beta} \mathbf{G}(t_{k+1}, X_{\alpha,k+1}^{-}) - \overline{C}_{k,n}^{\beta} \mathbf{G}(t_k, X_{\alpha,k}^{-}) \right],$$
(17)

$$X_{\alpha,n+1}^{+} = X_{0,\alpha}^{+} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_n, X_{\alpha,n}^{+}) + h^{\beta} \sum_{k=0}^{n} \left[C_{k,n}^{\beta} \mathbf{G}(t_{k+1}, X_{\alpha,k+1}^{+}) - \overline{C}_{k,n}^{\beta} \mathbf{G}(t_k, X_{\alpha,k}^{+}) \right],$$
(18)

where the coefficients $C_{k,n}^{\beta}$, $\overline{C}_{k,n}^{\beta}$ are given by

$$\begin{split} C_{k,n}^{\beta} &= \frac{\beta}{\Phi(\beta)\Gamma(\beta+2)} \Big[(n+1-k)^{\beta+1} - (n-k)^{\beta}(n+1-k+\beta) \Big] \\ \overline{C}_{k,n}^{\beta} &= \frac{\beta}{\Phi(\beta)\Gamma(\beta+2)} \Big[(n-k)^{\beta+1} + (n-k+1)^{\beta}(n-k+\beta) \Big]. \end{split}$$

Finally, the numerical solution in type 1 of the fuzzy initial value problem (11) is established from two difference systems (17) and (18).

Remark 6.1 (*error estimation*). In order to establish the error estimation while approximating the fractional differential equation using our suggested method, we assume that the vector $X_{\alpha}^{*}(\cdot) \in C^{2}([0, T], \mathbb{R}^{4})$ for all $\alpha \in [0, 1]$, which follows that the second-order partial derivative $\frac{\partial^{2}G(t, X_{\alpha}^{*}(t))}{\partial t^{2}}$ is continuous on [0, T] and hence, the function $\frac{\partial^{2}G(t, X_{\alpha}^{*}(t))}{\partial t^{2}}$ is bounded on this interval.

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In the approximation (16), we estimate the integral term $\int (t_{n+1} - \tau)^{\beta-1} G(\tau, X^*_{\alpha}(\tau)) d\tau$ by using two-step La-

grange interpolation polynomial. Hence, the error of integral term's estimation is given by

$$\left\| G(\tau, X_{\alpha}^{*}(\tau)) - \left[\frac{G(t_{k+1}, X_{\alpha,k+1}^{*})}{h}(\tau - t_{k}) - \frac{G(t_{k}, X_{\alpha,k}^{*})}{h}(\tau - t_{k+1}) \right] \right\| \leq \frac{M_{n}}{2!}(\tau - t_{k})(t_{k+1} - \tau),$$

where $M_n = \sup_{[0,t_{n+1}]} d_{\infty} \left(\frac{\partial_{gH}^2 G(\tau, X(\tau))}{\partial \tau^2}, \overline{0} \right)$ and $\tau \in [t_k, t_{k+1}].$

Hence, the error in our approximation is given by

$$R_n^{\alpha} = \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \sum_{k=0}^n \int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta-1} \frac{M_n}{2!} (\tau - t_k) (t_{k+1} - \tau) d\tau.$$

Note that the mapping $\tau \mapsto (t_{n+1} - \tau)^{\beta-1} (\tau - t_k)$ is positive on the interval $[t_k, t_{k+1}]$. Thus, by using Mean Value Theorem for integral, there exists $c_k \in [t_k, t_{k+1}]$

$$\begin{split} R_n^{\alpha} &= \frac{\beta}{\Gamma(\beta)\Phi(\beta)} \sum_{k=0}^n \frac{M_n}{2} (t_{k+1} - c_k) \int_{t_k}^{t_{k+1}} (t_{n+1} - \tau)^{\beta - 1} (\tau - t_k) d\tau \\ &= \frac{h^{\beta + 1} \beta}{2\Gamma(\beta + 2)\Phi(\beta)} \sum_{k=0}^n M_n (t_{k+1} - c_k) \left[(n+1-k)^{\beta + 1} - (n-k)^{\beta} (n+1-k+\beta) \right] \\ &\leq \frac{M_n h^{\beta + 2} \beta}{2\Gamma(\beta + 2)\Phi(\beta)} \sum_{k=0}^n \left[(n+1-k)^{\beta + 1} - (n-k)^{\beta} (n+1-k+\beta) \right] \\ &= \frac{M_n h^{\beta + 2} \beta}{2\Gamma(\beta + 2)\Phi(\beta)} \sum_{k=0}^n \left\{ (n+1-k+\beta) \left[(n+1-k)^{\beta} - (n-k)^{\beta} \right] - \beta (n+1-k)^{\beta} \right\}. \end{split}$$

By using Proposition 7.3, it yields $(n+1-k)^{\beta} - (n-k)^{\beta} \le (n+1-k-(n-k))^{\beta} = 1$ and hence,

$$R_n^{\alpha} \leq \frac{M_n h^{\beta+2} \beta}{2\Gamma(\beta+2)\Phi(\beta)} \sum_{k=0}^n \left[(n+1-k+\beta) - \beta(n+1-k)^{\beta} \right]$$
$$= \frac{M_n \beta}{2\Gamma(\beta+2)\Phi(\beta)} \left[\frac{n(n+1)}{2} + n\beta - \frac{(n+1)^{\beta}}{\beta} \right] h^{\beta+2}.$$

Hence, we can see that the error bound of our proposed numerical scheme is $Ch^{\beta+2}$ that is similar to the result in [51] and it shows the faster convergent rate with the result in [21]. Indeed, in the literature [21], the authors' approach gave the error bound of approximation with convergent rate $\tilde{C}h$. Let us recall that the error of approximation comes from the approximation of G(t, X(t)) by its two-step Lagrange interpolating polynomial. The smaller error is expected to be attained with higher-order interpolating polynomial.

Remark 6.2. By similar method, we also obtain the numerical scheme for the numerical solution in type 2 of the fuzzy fractional initial value problem (11):

$$\begin{aligned} X_{\alpha,n+1}^{-} &= X_{0,\alpha}^{-} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_n, X_{\alpha,n}^{+}) + h^{\beta} \sum_{k=0}^{n} \left[C_{k,n}^{\beta} \mathbf{G}(t_{k+1}, X_{\alpha,k+1}^{+}) - \overline{C}_{k,n}^{\beta} \mathbf{G}(t_k, X_{\alpha,k}^{+}) \right], \\ X_{\alpha,n+1}^{+} &= X_{0,\alpha}^{+} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_n, X_{\alpha,n}^{-}) + h^{\beta} \sum_{k=0}^{n} \left[C_{k,n}^{\beta} \mathbf{G}(t_{k+1}, X_{\alpha,k+1}^{-}) - \overline{C}_{k,n}^{\beta} \mathbf{G}(t_k, X_{\alpha,k}^{-}) \right]. \end{aligned}$$

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Remark 6.3 (*Numerical algorithm*). Now, we summarize the above approximate scheme and give a numerical algorithm to implement the numerical solution of the problem (1)–(2) as follows:

Algorithm 1: The proposed numerical method **Input:** The fractional order β – Number of partitions N – The parameters in Table 1 – The initial and final time – The initial condition X_0 . Output: The numerical solution of the FFIVP (11) **Data:** Initialization a = 0; T = 50;// set up starting and ending points 1 h = (T - a)/N;2 // set up step size $\Phi(\beta) = 1 - \beta + \frac{\beta}{\Gamma(\beta)};$ 3 // set up normalization function /* preallocation size */ $x = \operatorname{zeros}(1, N); y = \operatorname{zeros}(1, N); z = \operatorname{zeros}(1, N); v = \operatorname{zeros}(1, N);$ 4 5 X = zeros(4, N);// The solution vector $X = (x, y, z, v)^{t}$ /* Numerical solution of the problem (11) */ 6 for n = 1, 2, ..., N do $C_{k,n}^{\beta} = \frac{\beta}{\Phi(\beta)\Gamma(\beta+2)} \left[(n+1-k)^{\beta+1} - (n-k)^{\beta}(n+1-k+\beta) \right];$ 7 $\overline{C}_{k,n}^{\beta} = \frac{\beta}{\Phi(\beta)\Gamma(\beta+2)} \left[(n-k)^{\beta+1} + (n-k+1)^{\beta}(n-k+\beta) \right];$ 8 $X_{\alpha,n+1}^{-} = X_{0,\alpha}^{-} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_n, X_{\alpha,n}^*) + h^{\beta} \sum_{k=0}^{n} \left[C_{k,n}^{\beta} \mathbf{G}(t_{k+1}, X_{\alpha,k+1}^*) - \overline{C}_{k,n}^{\beta} \mathbf{G}(t_k, X_{\alpha,k}^*) \right];$ 9 $X_{\alpha,n+1}^{+} = X_{0,\alpha}^{+} + \frac{1-\beta}{\Phi(\beta)} \mathbf{G}(t_n, X_{\alpha,n}^{*}) + h^{\beta} \sum_{k=0}^{n} \left[C_{k,n}^{\beta} \mathbf{G}(t_{k+1}, X_{\alpha,k+1}^{*}) - \overline{C}_{k,n}^{\beta} \mathbf{G}(t_k, X_{\alpha,k}^{*}) \right];$ 10 // for k = 0, 1, 2, ..., n, $\alpha \in [0, 1]$ /* Plot the solution */ plot(t, X)11 // plot the numerical solution

6.2. Numerical simulation

In this section, the numerical simulations are carried out by using Matlab program and some different cases of parameters in Table 1 to show the uncertain behavior versus time of numerical solution of the fractional SIQR model (1) and the effect of the fractional derivative β on the transmission of viruses in WSNs for $\beta \in \{0.3, 0.6, 0.9\}$.

(a) We simulate the uncertain behavior of mild solution of the fuzzy fractional initial value problem for the fuzzy fractional SIQR model (1) with parameters

A = 3	$\mu = 0.45$	$\lambda = 0.55$	v = 0.25
$\omega = 0.02$	$\sigma = 0.02$	$\gamma = 0.001$	$\eta = 0.005$

and uncertain initial data $S_0 = (1.3, 1.5, 1.7)$, $I_0 = (2.5, 2.75, 3.0)$, $Q_0 = (1.0, 1.25, 1.5)$ and $R_0 = (0.01, 0.02, 0.03)$. Fig. 4 presents the time series of numerical solution of the fuzzy fractional SIQR model for some different values of fractional order β . For the above parameters, we can directly compute the basic reproduction number \Re corresponding to the considered model is $\Re = 0.814 < 1$, which implies that the virus-free equilibrium E_1 is globally asymptotically stable. In fact, from Fig. 4, we can see that the infectious component I(t) of the solution tends to be vanished as the time is increasing, that means the spread will die out or equivalently, viruses can be removed completely out the WSNs.

Three figures (Figs. 5–7) show the dynamical behaviors of the fuzzy fractional SIQR model (1) for some different values of fractional order β .

(b) We simulate the uncertain behavior of mild solution of the fuzzy fractional initial value problem for the fuzzy fractional SIQR model (1) with parameters

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Fig. 4. Time series of numerical solution of the fuzzy fractional SIQR model. (For interpretation of the colors in the figure(s), the reader is referred to the web version of this article.)



Fig. 5. The dynamical behavior of the fuzzy fractional SIQR model with $\beta = 0.3$.

A = 2	$\mu = 0.45$	$\lambda = 0.52$	v = 0.1
$\omega = 0.05$	$\sigma = 0.1$	$\gamma = 0.75$	$\eta = 0.075$

and uncertain initial data $S_0 = (2.3, 2.5, 2.7)$, $I_0 = (3.6, 3.75, 3.8)$, $Q_0 = (0.015, 0.025, 0.035)$ and $R_0 = (3.9, 4.0, 4.2)$. Fig. 8 presents the time series of numerical solution of the fuzzy fractional SIQR model for some different values of fractional order β . With the above parameters, we can directly compute the basic reproduction number \Re corresponding to the considered model is $\Re = 1.524 > 1$, that means the spread won't die out. In fact, by Fig. 8, we can see that the infectious component I(t) is asymptotic to some positive values as the time increasing. This means that the spread of viruses remains in the network.

Three figures (Figs. 9–11), we show the dynamical behaviors of the fuzzy fractional SIQR model (1) for some different values of fractional order β .

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Fig. 6. The dynamical behavior of the fuzzy fractional SIQR model with $\beta = 0.6$.



Fig. 7. The dynamical behavior of the fuzzy fractional SIQR model with $\beta = 0.9$.

7. Appendix

In the following, we introduce Table 2 of fractional calculus's notations.

Next, we recall the concept of Laplace transform for fuzzy-valued functions proposed by Allahviranloo and Barkhordari [5]. Let $f : [0, \infty) \to \mathscr{E}$ be a continuous fuzzy-valued function such that $f(t)e^{-st}$ is integrable on $[0, \infty)$. Then, the fuzzy Laplace transform of f(t) is defined by

$$\mathscr{F}(s) = \tilde{\mathscr{L}}\{f(t)\}(s) = \int_{0}^{\infty} f(t)e^{-st}dt,$$

whose α -cuts are given by $\left[\tilde{\mathscr{L}}\{f(t)\}(s)\right]^{\alpha} = \left[\mathscr{L}\{f_{\alpha}^{-}(t)\}(s), \mathscr{L}\{f_{\alpha}^{+}(t)\}(s)\right]$ where $\alpha \in [0, 1]$ and the functions $\mathscr{L}\{f_{\alpha}^{-}(t)\}(s), \mathscr{L}\{f_{\alpha}^{+}(t)\}(s)$ are classical Laplace transforms of real-valued functions.

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Fig. 8. Time series of numerical solution of the fractional SIQR model.



Fig. 9. The dynamical behavior of the fuzzy fractional SIQR model with $\beta = 0.3$.

Remark 7.1. In the multidimensional case, if $f : [0, \infty) \to \mathscr{E}^m$ is a fuzzy vector-valued function, defined by $t \mapsto f(t) = (f_1(t) \quad f_2(t) \quad \cdots \quad f_m(t))^{t}$, then the fuzzy Laplace transform of f is known as the vector of fuzzy Laplace transforms of all its components, i.e.,

$$\tilde{\mathscr{L}}{f(t)}(s) = \left(\tilde{\mathscr{L}}{f_1(t)}(s) \quad \tilde{\mathscr{L}}{f_2(t)}(s) \quad \cdots \quad \tilde{\mathscr{L}}{f_m(t)}(s)\right)^{\mathsf{t}}.$$

In the following, we present the Laplace transform of Caputo Atangana-Baleanu fractional derivative

Proposition 7.1. Assume that $f \in C^1([0, b], \mathcal{E})$. Then, we have

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Fig. 10. The dynamical behavior of the fuzzy fractional SIQR model with $\beta = 0.6$.



Fig. 11. The dynamical behavior of the fuzzy fractional SIQR model with $\beta = 0.9$.

Table 2 Some used notations.

Notation	Description	Location
$\overline{\mathcal{I}_{+}^{\beta}f(t)}$	The Riemann–Liouville fractional integral of order β of a fuzzy-valued function $f(t)$	Definition 3.4
$^{abc}\mathrm{D}^{\beta}_{+}f(t)$	The Caputo Atangana–Baleanu fractional derivative of order β of a real function $f(t)$	Definition 4.1
$^{ab}\mathbf{I}^{\beta}_{+}f(t)$	The Riemann–Liouville Atangana–Baleanu fractional integral of order β of a real function $f(t)$	Definition 4.2
$^{abc}\mathfrak{D}^{\beta}_{+}f(t)$	The Caputo Atangana–Baleanu fractional derivative of order β of a fuzzy-valued function $f(t)$	Definition 4.3
$^{ab}\mathcal{I}^{\beta}_{+}f(t)$	The Riemann–Liouville Atangana–Baleanu fractional integral of order β of a fuzzy-valued function $f(t)$	Definition 4.4

(i) If f is gH-differentiable in type 1 then

$$\tilde{\mathscr{L}}\left\{{}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s) = \frac{\Phi(\beta)}{(1-\beta)s^{\beta}+\beta}\left[s^{\beta}\tilde{\mathscr{L}}\left\{f(t)\right\}(s) \ominus s^{\beta-1}f(0)\right].$$

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(ii) If f is gH-differentiable in type 2 then

$$\tilde{\mathscr{L}}\left\{^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s) = \frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta}\left[s^{\beta-1}f(0)\ominus s^{\beta}\tilde{\mathscr{L}}\left\{f(t)\right\}(s)\right].$$

Proof. For each $\alpha \in [0, 1]$, the α -cuts of $\tilde{\mathscr{L}} \left\{ {}^{abc} \mathfrak{D}^{\beta}_{+} f(t) \right\} (s)$ is given in following compact form

$$\left[\tilde{\mathscr{L}}\left\{^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s)\right]^{\alpha} = \left[\mathscr{L}\left\{\left(^{abc}\mathsf{D}^{\beta}_{+}f\right)^{-}_{\alpha}(t)\right\}(s), \mathscr{L}\left\{\left(^{abc}\mathsf{D}^{\beta}_{+}f\right)^{+}_{\alpha}(t)\right\}(s)\right].$$

(i) If f is gH-differentiable in type 1 then according Definition 3.2 and Definition 4.3, it is true that

$$\begin{bmatrix} abc \mathfrak{D}^{\beta}_{+} f(t) \end{bmatrix}^{\alpha} = \begin{bmatrix} abc \mathbf{D}^{\beta}_{+} f^{-}_{\alpha}(t), abc \mathbf{D}^{\beta}_{+} f^{+}_{\alpha}(t) \end{bmatrix}.$$

Hence, we directly deduce that

$$\left[\tilde{\mathscr{L}}\left\{^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s)\right]^{\alpha} = \left[\mathscr{L}\left\{^{abc}\mathsf{D}^{\beta}_{+}f^{-}_{\alpha}(t)\right\}(s), \mathscr{L}\left\{^{abc}\mathsf{D}^{\beta}_{+}f^{+}_{\alpha}(t)\right\}(s)\right].$$

Next, by applying Laplace transform (3), we receive

$$\begin{aligned} \mathscr{L}\left\{^{abc}\mathrm{D}^{\beta}_{+}f^{-}_{\alpha}(t)\right\}(s) &= \frac{\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \Big(s^{\beta}\mathscr{L}\left\{f^{-}_{\alpha}(t)\right\}(s) - s^{\beta-1}f^{-}_{\alpha}(0)\Big),\\ \mathscr{L}\left\{^{abc}\mathrm{D}^{\beta}_{+}f^{+}_{\alpha}(t)\right\}(s) &= \frac{\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \Big(s^{\beta}\mathscr{L}\left\{f^{+}_{\alpha}(t)\right\}(s) - s^{\beta-1}f^{+}_{\alpha}(0)\Big). \end{aligned}$$

Thus, it implies that the below equality holds for all $\alpha \in [0, 1]$

$$\begin{bmatrix} \mathscr{L}\left\{ {}^{abc}\mathsf{D}_{+}^{\beta}f_{\alpha}^{-}(t)\right\}(s), \mathscr{L}\left\{ {}^{abc}\mathsf{D}_{+}^{\beta}f_{\alpha}^{+}(t)\right\}(s) \end{bmatrix} \\ = \frac{\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \begin{bmatrix} s^{\beta}\mathscr{L}\left\{ f_{\alpha}^{-}(t)\right\}(s) - s^{\beta-1}f_{\alpha}^{-}(0), s^{\beta}\mathscr{L}\left\{ f_{\alpha}^{+}(t)\right\}(s) - s^{\beta-1}f_{\alpha}^{+}(0) \end{bmatrix}.$$

On the other hand, since the parametric form $\left[s^{\beta} \mathscr{L}\left\{f_{\alpha}^{-}(t)\right\}(s) - s^{\beta-1}f_{\alpha}^{-}(0), s^{\beta} \mathscr{L}\left\{f_{\alpha}^{+}(t)\right\}(s) - s^{\beta-1}f_{\alpha}^{+}(0)\right]$ holds for all $\alpha \in [0, 1]$ then we can write

$$\left[s^{\beta}\mathscr{L}\left\{f_{\alpha}^{-}(t)\right\}(s) - s^{\beta-1}f_{\alpha}^{-}(0), s^{\beta}\mathscr{L}\left\{f_{\alpha}^{+}(t)\right\}(s) - s^{\beta-1}f_{\alpha}^{+}(0)\right] = \left[s^{\beta}\tilde{\mathscr{L}}\left\{f(t)\right\}(s) \ominus s^{\beta-1}f(0)\right]^{\alpha}.$$

e, we receive $\left[\tilde{\mathscr{L}}\left\{abc\mathfrak{D}_{+}^{\beta}f(t)\right\}(s)\right]^{\alpha} = \frac{\Phi(\beta)}{(t-1)^{\alpha}}\left[s^{\beta}\tilde{\mathscr{L}}\left\{f(t)\right\}(s) \ominus s^{\beta-1}f(0)\right]^{\alpha}$ for all $\alpha \in [0, 1]$

Hence, we receive $\left[\hat{\mathscr{L}}\left\{a^{bc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s)\right]^{r} = \frac{\varphi(\beta)}{(1-\beta)s^{\beta}+\beta}\left[s^{\beta}\hat{\mathscr{L}}\left\{f(t)\right\}(s)\ominus s^{\beta-1}f(0)\right]^{r}$ (ii) Using the assumption that f is gH-differentiable in type 2, we have

$$\begin{bmatrix} abc \mathfrak{D}^{\beta}_{+}f(t) \end{bmatrix}^{\alpha} = \begin{bmatrix} abc D^{\beta}_{+}f^{+}_{\alpha}(t), abc D^{\beta}_{+}f^{-}_{\alpha}(t) \end{bmatrix}.$$

As a corollary of the Laplace transform (3) and by similar arguments as in Case (i), it is true that

$$\begin{split} \left[\tilde{\mathscr{L}} \left\{ {}^{abc} \mathfrak{D}^{\beta}_{+} f(t) \right\} (s) \right]^{\alpha} &= \left[\frac{\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta} \mathscr{L} \left\{ f^{+}_{\alpha}(t) \right\} (s) - s^{\beta-1} f^{+}_{\alpha}(0) \right), \\ &\qquad \frac{\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta} \mathscr{L} \left\{ f^{-}_{\alpha}(t) \right\} (s) - s^{\beta-1} f^{-}_{\alpha}(0) \right) \right] \\ &= \left[\frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta-1} f^{-}_{\alpha}(0) - s^{\beta} \mathscr{L} \left\{ f^{-}_{\alpha}(t) \right\} (s) \right), \\ &\qquad \frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta-1} f^{+}_{\alpha}(0) - s^{\beta} \mathscr{L} \left\{ f^{+}_{\alpha}(t) \right\} (s) \right) \right], \end{split}$$

for all $\alpha \in [0, 1]$, which implies that the H-difference $\frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta-1}f(0)\ominus s^{\beta}\tilde{\mathscr{L}}\left\{f_{\alpha}^{+}(t)\right\}(s)\right)$ exist and hence, we can write

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$$\begin{bmatrix} \frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta-1}f(0)\ominus s^{\beta}\tilde{\mathscr{L}}\left\{f_{\alpha}^{+}(t)\right\}(s)\right)\end{bmatrix}^{\alpha} = \\ \begin{bmatrix} \frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta-1}f_{\alpha}^{-}(0)-s^{\beta}\mathscr{L}\left\{f_{\alpha}^{-}(t)\right\}(s)\right), \frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta} \left(s^{\beta-1}f_{\alpha}^{+}(0)-s^{\beta}\mathscr{L}\left\{f_{\alpha}^{+}(t)\right\}(s)\right)\end{bmatrix}$$

Thus, it follows that if f is gH-differentiable in type 2 then

$$\left[\tilde{\mathscr{L}}\left\{{}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s)\right]^{\alpha} = \left[\frac{(-1)\Phi(\beta)}{(1-\beta)s^{\beta}+\beta}\left(s^{\beta-1}f(0)\ominus s^{\beta}\tilde{\mathscr{L}}\left\{f^{+}_{\alpha}(t)\right\}(s)\right)\right]^{\alpha} \quad \text{for all } \alpha \in [0,1],$$

which completes our proof. \Box

Proposition 7.2. Assume that $f : [0, \infty) \to \mathcal{E}$ is a continuous fuzzy-valued function. Then, we have

$$\tilde{\mathscr{L}}\left\{\int_{0}^{t} \mathbb{E}_{\beta}\left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta}\right] f(\tau) d\tau\right\}(s) = \frac{s^{\beta-1}}{s^{\beta} + \frac{\beta}{1-\beta}}\tilde{\mathscr{L}}\left\{f(t)\right\}(s).$$

Proof. For each t > 0, denote $g(t) = \mathbb{E}_{\beta} \left[-\beta \frac{t^{\beta}}{1-\beta} \right]$. Then, the convolution of f(t) and g(t) is given by

$$(f*g)(t) = \int_0^t f(\tau)g(t-\tau)d\tau = \int_0^t \mathbb{E}_\beta \left[-\beta \frac{(t-\tau)^\beta}{1-\beta} \right] f(\tau)d\tau.$$

By using the Laplace transform of fuzzy convolution in [5], we directly obtain

$$\tilde{\mathscr{L}}\left\{\int_{0}^{t} \mathbb{E}_{\beta}\left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta}\right] f(\tau) d\tau\right\}(s) = \tilde{\mathscr{L}}\left\{\mathbb{E}_{\beta}\left[-\beta \frac{t^{\beta}}{1-\beta}\right]\right\}(s)\tilde{\mathscr{L}}\left\{f(t)\right\}(s).$$

From [50], it is true that $\tilde{\mathscr{L}}\left\{\mathbb{E}_{\beta}\left[-\beta\frac{t^{\beta}}{1-\beta}\right]\right\}(s) = \frac{s^{\beta-1}}{s^{\beta} + \frac{\beta}{1-\beta}}$. Hence, the proof is completed. \Box

Remark 7.2. Based on Proposition 7.2, we can also imply the conclusion of Proposition 7.1. Indeed, for each $f \in C^1([0, b], \mathscr{E})$ and for all $t \in [0, b]$, we have

$${}^{abc}\mathfrak{D}^{\beta}_{+}f(t) := \frac{\Phi(\beta)}{1-\beta} \int_{0}^{t} \mathbb{E}_{\beta} \left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{gH}(\tau) d\tau$$

Then, by applying fuzzy Laplace transform for both sides of above formula, it yields

$$\begin{split} \tilde{\mathscr{L}}\left\{ {}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s) &= \tilde{\mathscr{L}}\left\{ \frac{\Phi(\beta)}{1-\beta} \int\limits_{0}^{t} \mathbb{E}_{\beta}\left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{gH}(\tau) d\tau \right\}(s) \\ &= \frac{\Phi(\beta)}{1-\beta} \tilde{\mathscr{L}}\left\{ \int\limits_{0}^{t} \mathbb{E}_{\beta}\left[-\beta \frac{(t-\tau)^{\beta}}{1-\beta} \right] f'_{gH}(\tau) d\tau \right\}(s). \end{split}$$

Next, by using Proposition 7.2 and the Laplace transform for gH-derivatives in [5], we obtain

$$\tilde{\mathscr{L}}\left\{{}^{abc}\mathfrak{D}^{\beta}_{+}f(t)\right\}(s) = \frac{\Phi(\beta)}{1-\beta}\frac{s^{\beta-1}}{s^{\beta}+\frac{\beta}{1-\beta}}\tilde{\mathscr{L}}\left\{f'_{gH}(t)\right\}(s)$$

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$$= \begin{cases} \frac{\Phi(\beta)}{1-\beta} \frac{s^{\beta} \tilde{\mathscr{L}}\{f(t)\}(s) \ominus s^{\beta-1} f(0)}{s^{\beta} + \frac{\beta}{1-\beta}} & \text{if } f \text{ is gH-differentiable in type 1} \\ \frac{(-1)\Phi(\beta)}{1-\beta} \frac{s^{\beta-1} f(0) \ominus s^{\beta} \tilde{\mathscr{L}}\{f(t)\}(s)}{s^{\beta} + \frac{\beta}{1-\beta}} & \text{if } f \text{ is gH-differentiable in type 2.} \end{cases}$$

Proposition 7.3. For each $\alpha \in (0, 1)$ and $0 < y \le x$, we have $x^{\alpha} - y^{\alpha} \le (x - y)^{\alpha}$.

Proof. For each $\alpha \in (0, 1)$ and $0 < y \le x$, consider a real-valued function $h(t) = (t - y)^{\alpha} - t^{\alpha}$ on the interval $[y, \infty)$. Since the function $h'(t) = \alpha(t - y)^{\alpha - 1} - \alpha t^{\alpha - 1} \ge 0$ for all $t \ge y$, it implies that the function h(t) is increasing on $[y, \infty)$. Hence, we obtain

$$h(x) = (x - y)^{\alpha} - x^{\alpha} \ge h(y) = -y^{\alpha},$$

or equivalently, $(x - y)^{\alpha} \ge x^{\alpha} - y^{\alpha}$. The proof is completed. \Box

8. Conclusions

In this paper, our aim is to investigate a mathematical SIQR model for propagation of viruses in WSNs with the rate of change in sense of Atangana–Baleanu Caputo fuzzy fractional gH-differentiability. To achieve this goal, we introduce a new concept of fractional derivative with non-local and non-singular kernel of fuzzy-valued functions. Here, the non-local kernel is built by the Mittag-Leffler function and the proposed fractional derivative is based upon the Caputo sense. Moreover, some related analysis properties of the proposed derivative and integral are also discussed. Next, the new derivative is applied to model the propagation of viruses in WSNs with fuzzy data and parameters. A theoretical result about the existence and uniqueness of mild solution of the FIVP is shown. In addition, the attack behavior of virus is then analytically solved via fuzzy Laplace transform and numerically solved via a proposed numerical method. In further research, it is planned to verify the applicability of the proposed fractional derivative in different epidemic models in WSNs and some related problem such as stability, stabilizability or control problem.

Declaration of competing interest

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

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Research paper

The dynamical behaviors of fractional-order SE₁E₂IQR epidemic model for malware propagation on Wireless Sensor Network



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ABSTRACT

In order to investigate the effectiveness of guarantine strategy and the heterogeneity of scale-free network on epidemic spreading, this paper focuses on the investigation of a new fractional epidemiology model, namely fractional SE₁E₂IQR epidemic model. Our proposed model introduces an isolation class (Q) and an exposure class with two distinct compartments E_1 (Type 1-exposed) and E_2 (Type 2-exposed). The dynamics of the network-based fractional-order SE1E2IQR epidemic model are studied from the viewpoint of stability analysis and bifurcation. Firstly, by using the next-generation method, we derive the basic reproductive ratio \mathcal{R}_0 of the proposed epidemic model, which plays an important role in determining not only the unique existence of epidemic equilibrium point \mathbf{E}_{*} but also the locally asymptotically stability of malware-free equilibrium point **E**₀. However, the paper points out that the condition $\mathcal{R}_0 < 1$ is not sufficient to eliminate the malware from the network. In addition, the direction of bifurcation at $\Re_0 = 1$ is also presented. Furthermore, by graphical simulations and computations, we can evaluate the importance of parameters in the basic reproductive ratio \mathcal{R}_0 and show that the quarantine treatment plays a key role in controlling the epidemic disease.

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1. Introduction

Recently, since many systems in nature and society can be described by using complex network models, there have been various considerable works studying the structures and dynamics of complex networks with interdisciplinary applications such as traffic control, environmental and habitat monitoring, communication systems, or biological systems, etc. A scale-free network is an important classical complex network, that can better describe many real-world systems such as social networks [1], the World Wide Web [2], wireless sensor networks [3], security network [4] or the Internet [5]. This work investigates Wireless Sensor Networks (WSNs) from the viewpoint of complex network theory in order to discuss the treatments to protect the network from the malicious object's attack and prolong the lifetime of the network. Note that sensor nodes are battery-operated, located in remote, complex terrain and it is obvious that the battery replacement is impractical. Moreover, each operation of WSNs requires thousands of sensor nodes that will be deployed, a WSN-specific parameter such as the energy loss should be considered. Therefore, when describing the network structure of WSNs, some recent works took into account the energy-aware problem such as Jian et al. [3] and Zhu et al. [6]. In

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this work, based on the Barabási Albert scale-free network, we proposed using a special kind of scale-free network, namely Energy-Aware Barabási Albert (EABA) scale-free network to characterize the network properties of WSNs. Because sensor nodes are energy-constrained devices, they generally have weak defense capabilities and become attractive targets for malware such as worms, viruses or trojans, etc. In addition, since sensor network uses wireless communication for information exchange, hackers and advertisers can easily exploit the flaws of systems and target a particular sensor node through which to introduce malware programs in WSNs. Next, this infected node starts to propagate in the network gradually from that point to its adjacent nodes. There are many negative influences of malware attacks on WSNs such as quickly exhausting the energy of sensor nodes, slowing down the operation in sensor networks or growing network traffic, etc. To protect the security and integrity of WSNs against malware attacks, we first need to clearly understand the dynamic characteristics of the malware and their infection mechanism. Some researchers found that the propagation pattern of epidemic disease in biological models and social models is quite similar to the propagation in the complex network generated by malware programs. And mathematical modeling has become a powerful tool for not only analyzing the propagation, evaluating the various prevention and control programs but also identifying the important data that need to be collected to make forecasts. Especially, mathematical modeling of epidemic dynamics on the complex network has recently attracted an increasing amount of attention from many mathematicians and researchers. Unlike classical epidemic models which assume the same rate of disease-causing contacts, epidemic models with network-based take into account the different degree of nodes, i.e., the heterogeneity contact rates, and complex topology of potential contact. There are many realistic examples of mathematical epidemic models in complex networks. Some literature can be found in [7-12].

It is well-known that fractional derivatives and fractional integrals, which appeared early in the 19th century, have the non-local property, i.e., these derivatives and integrals can present both the past information and distributed effect of any physical systems. This proves the great ability of fractional derivatives and integrals to represent complex realworld phenomena more accurately and efficiently than ordinary calculus. During a long history of development, numerous studies have proved the considerable advantages of fractional calculus with various disciplines of the real world. In recent years, besides the rapid popularization of fractional calculus, a lot of detailed studies on fractional dynamical systems and related problems have been carried out by many researchers and have achieved noticeable results in various fields of basic sciences and engineering due to some main advantages of fractional calculus such as memory properties of fractional derivatives or better simulations in fractal materials or viscoelasticity environments, see [7,8,13–18]. Due to the fact that malware takes advantage of the process of signal transmission between sensor nodes to inject malware code in the network and signal transmissions are known as memory and hereditary processes that often have the significant dependence on the flexibility of the environment, the history of functions or the texture and characteristic properties of the material, that are difficult to describe correctly by mean of integer order differential systems. In addition, fractional calculus is also an important tool for the study of some hereditary properties of compartments in epidemic models. In this work, based on the advantages of fractional calculus in the modeling of epidemic disease transmission on complex networks, we generalize classic epidemic models by replacing the integer-order derivative on the left side with fractional derivatives in Caputo sense. Then, the obtained model can produce very good estimation, as well as interesting equations from a mathematical point of view. However, we must face the natural question that does the change in the order of derivatives automatically establish consistent models w.r.t. the model's parameters? Interesting work was published in [19] proved that this cannot happen in general. Recently, there has been a lot of literature studying the fractional epidemiology theory and its applications in computer networks. For example, see Dubey et al. [20], Dong et al. [7], Graef et al. [8], Hassouna et al. [21], Mishra et al. [11,22] and Naim et al. [23].

Due to the fact that target areas of real-world applications in the WSNs always have complex terrains and irregular climates, the information transmission's speed in WSNs is profoundly influenced by geographical and climatic factors. That is the reason why we should take into account the influence of external natural factors when studying the infection of malware on the networks. On the other hand, since natural phenomena in reality always contain vagueness and uncertainty, it is obvious that we must accept the presence of uncertainties in our proposed models. Additionally, in the wireless sensor network, different network clusters will perform different sensing, measuring, and collecting tasks, and hence, the information's propagation speed is obviously uneven, which is also directly affected by the infection of malicious codes in the network. This factor is often expressed in the sense of a node's number of state changes and often cannot be measured precisely but expressed through language variables. Moreover, the infectivity of a susceptible node also depends on the density of infectious nodes in its neighbors, that is, not every susceptible node that contacts an infectious node, will become immediately an infectious node. In fact, in order to express the term of the density of nodes in the network, one cannot use exact values, but they are usually expressed through linguistic variables such as high, moderate, low. The appearance of uncertain factors in our considered model suggests introducing the use of fuzzy set theory. Initiated by Zadeh [24] in the early 1960s, fuzzy set theory has gained a lot of significant achievements in a wide range of every aspect of scientific areas, see [24,25]. Recently, there have been lots of computing tools developed to make use of fuzzy set theory in fuzzy control, where the experience of humans is valid. Fuzzy control is based on fuzzy sets, fuzzy logic, and fuzzy inference, which plays a key role in many real-world applied problems, see [25,26]. In practice, researchers often consider the uncertainty in the form of fuzzy sets. Thus, fuzzy logic and fuzzy set theory become a powerful tools for expressing inaccurate facts and making our studies agree with real-life situations. In the epidemiology theory, there have some interesting literature that applies fuzzy logic and fuzzy sets to study epidemic models. For instance, Dong et al. [7,13] applied fuzzy analysis and fuzzy fractional differential equations to study the fuzzy fractional SEIR epidemic models, fuzzy fractional SIQR epidemic model and discuss some interesting related problems such as solvability, optimal control, numerical solutions. The modified SIR epidemic model with fuzzy transmission rate and fuzzy control based on fuzzy sets were studied in [27–29]. However, to the best of our knowledge, there has a little work on the fuzzy epidemic model on heterogeneous complex networks, see [30] for instance, in which the network structure directly affects the malware infection. This means due to the heterogeneity of complex networks, the level of being infected and transmission rate in different network clusters are un-similar. This is an important motivation for the classification of three classes of infected node: Type 1- Exposed, Type 2-Exposed and Infectious in this work. Here, the fuzzy logic is applied to take into account the network's heterogeneity and estimate the rate of infection.

For the convenience of the reader, we briefly summarize the structure of this paper as follows: In Section 2, we review some recent relevant developments and publications in the study of the mathematical epidemic model on the complex network to motivate the research. After that, the main contributions of this paper are briefly summarized in Section 3, while Section 4 is devoted to formulating a fractional network-based SE_1E_2IQR epidemic model with a fuzzy rule-based transmission rate. In Section 5, we present a detailed study on the analysis of the proposed epidemic model consisting of the positiveness of solution, the existence of equilibrium points, the basic reproductive ratio \Re_0 , the stability analysis and the bifurcation analysis. In order to illustrate the correctness of theoretical results, Section 6 gives some numerical simulations and graphical representations. At last, the Conclusions in Section 7 and Appendix are given.

2. Related works

Recently, there have been a considerable number of studies in the epidemiological models on complex networks. In the following, we briefly review some literature related to this work:

- Graef et al. [8] proposed a fractional-order SIR epidemic model with demography to examine the user adoption and abandonment of online social networks, where adoption is analogous to infection, and abandonment is analogous to recovery. After that, they discussed the existence and uniqueness of non-negative solutions of the proposed model as well as the existence and stability of its equilibrium points by using the Jacobian matrix technique and the Lyapunov function method. In particular, a threshold R_0^{α} was established to prove that the user-free equilibrium \mathbf{E}_0 is locally asymptotically stable if $R_0^{\alpha} < 1$ and the user-prevailing equilibrium \mathbf{E}_* is globally asymptotically stable if $R_0^{\alpha} > 1$. The theoretical results were then demonstrated by a case study of fitting the considered model to some Instagram user data. However, it is a fact that in reality, the network of Instagram users is not well-mixed and it should be taken into consideration the heterogeneity of the network for a better description.
- The paper [31] of Huang et al. introduced a network-based SIQRS epidemic model with demographics and vaccination to investigate the epidemic disease on complex heterogeneous networks. After obtaining the basic reproduction number R_0 , the permanence of disease and the globally asymptotic stability of disease-free equilibrium are analytically proved. In addition, the unique endemic equilibrium is shown to be globally attractive by using a monotone iterative method. In another work, Huo et al. [9] proposed an epidemiological model with three compartments to study the disease widespread on a scale-free network. This work introduced a network-based SIRS epidemic model with infection age and relapse. A striking result of this work is the use of fluctuation lemma and Lyapunov functional method to prove the globally asymptotic stability of endemic equilibrium point corresponding to basic reproductive ratio R_0 . Li et al. [32] also proposed a three compartmental epidemic model to study the malware spreading in a complex heterogeneous network by introducing a network-based SIRS epidemic model with birth and death rates. This work proved that the dynamics of the network are completely dependent on the basic reproductive ratio R_0 . In particular, the disease-free equilibrium is globally attractive if $R_0 < 1$ and unstable if otherwise. Moreover, the unique disease-free equilibrium is globally asymptotically stable if $R_0 > 1$. Finally, numerical simulations were given to demonstrate theoretical results. In conclusion, we can see that all three papers took into consideration the degree distribution of network nodes, which makes their studies agree with the property of real-world networks.
- Li and Yousef [33] studied a network-based SIR epidemic model with a saturated treatment function, that plays an important significance in characterizing the real-world situation that the number of patients that needs to be treated may exceed the treatment capacity. For this aim, a threshold value R_0 , which plays an important significance in the stability of a disease-free equilibrium, is obtained. Next, the author investigated the bifurcation at $R_0 = 1$ and established a necessary condition for the bifurcation directions at $R_0 = 1$. The bifurcation direction (backward or forward) and the stability of equilibria were then discussed to determine whether the disease is eliminated on the network. A novelty of this work is the use of a saturated treatment function instead of a linear treatment function, which can be applied for our considered model in future work.
- In order to study the effect of anti-virus treatments on epidemic spreading, an SIS model with limited treatment capacity on adaptive networks was introduced in [34]. Firstly, the author derived the existence condition of backward bifurcation or forward bifurcation at the disease-free equilibrium. Then, they discussed the effect of the bifurcation direction occurring at the disease-free equilibrium on the bi-stability of endemic equilibria and the elimination of epidemic disease of the model. The obtained results are interesting and can be extended to the case of a network-based model.

- Recently, Mishra et al. have applied mathematical modeling to study some classes of epidemic diseases. In the paper [11], Mishra et al. formulated the worm's attack on wireless sensor networks by formulating an epidemic model, namely the SEIRS-V model. Moreover, this work also considered the effectiveness of vaccination for the treatment of epidemic diseases on the network. Two main results of this work are the computation of basic reproductive ratio *R*₀ and the globally asymptotic stability of equilibrium points. Another study of Mishra et al. was presented in [22]. Indeed, the work [22] introduced an epidemic model based on the biological predator–prey model, in which three compartments: Susceptible, Infectious, and Recovered are predators while the Terminally infected compartment plays the predator role. After that, equilibrium points of the considered model were calculated and applied to investigate the stability analysis of equilibrium points. However, despite of studying the epidemic model in complex networks, the heterogeneity of complex networks has not been taken into account in both [11,22].
- In recent years, there have been some studies on the investigation of mathematical epidemic models with fuzziness factors. For instance, in the work [7], Dong et al. considered a modified SIR model with quarantine state to model the worm propagation in Wireless sensor network with fuzzy parameters, namely fuzzy fractional SIQR epidemic model. Here, the proposed epidemic model was governed by a system of fuzzy fractional differential equations under gH-differentiability. Then, by using the theory of fuzzy dynamical systems and fuzzy analysis, the authors studied the solvability of the proposed epidemic model and then, discussed the effect of isolation with stifling the infection of worms. An optimal control problem for a fractional SEIR epidemic model with fuzzy parameters was proposed in [13]. Here, under the horizontal membership function approach and granular differentiability for fuzzy-valued function, the authors established a necessary condition for the optimality of the proposed optimal control problem. Moreover, the obtained theoretical results were then applied to study the widespread of COVID-19 pandemic. The epidemic models with fuzzy transmission can be found in [27-29], in which the work [28] formulated a simple SIS epidemic model with a linear treatment control. The novelty of this work is that both disease transmission rate and treatment function were considered as fuzzy numbers. Then, the concepts of the fuzzy expected value of infected individuals and fuzzy basic reproduction numbers were proposed and examined. Moreover, based on the malware load on the model, a threshold condition of the pathogen was given at which the SIS epidemic model undergoes a transcritical bifurcation. Nandi et al. [29] studied a fuzzy SIS epidemic model, where both the disease transmission rate and treatment function are considered in saturated forms and contained fuzziness. The fuzzy expected value of infected individuals and fuzzy basic reproduction numbers were determined and investigated to examine the nature of the proposed epidemic model. Moreover, a threshold condition of the pathogen was derived at which the epidemic model undergoes a backward bifurcation. However, the works [7,13,27– 29] only consider the heterogeneous property of epidemic models in sense of fuzziness of model parameters, but the network's heterogeneity has not been taken into consideration. In recent work, Hosseini and Zandvakili [30] investigated a mathematical SEIRS-C model to describe the rumor spreading on the social networks based on fuzzy logic-based. Then, this paper presented some initial results consisting of basic reproductive ratio \mathcal{R}_0 and local stability of disease-free equilibrium point.

3. The contributions of this work

Motivated by aforesaid, this work is devoted to presenting detailed results on the analysis of a network-based epidemic model on the scale-free network with fractional order derivative and fuzzy rule-based interaction constant. Unlike epidemic models introduced in the preceding works, our proposed epidemic model consists of a compartment of quarantine and two types of exposure compartments: Type 1-Exposed and Type 2-Exposed. Based on this assumption, we propose a six compartmental epidemic model, SE₁E₂IQR to evaluate both the effectiveness of quarantine strategy and the influence of underlying heterogeneity of complex networks to the malware infection. Here, the network's heterogeneity is expressed by the node's connectivity and node's state change, which are taken into consideration in the model by using fuzzy logic. This formulation is the novelty of our work, which helps the considered epidemic model better fit with real-world situations. The main achievements of our work can be highlighted as follows:

- (i) Propose a new fractional epidemic model with latent periods and quarantine in the form of mean-field reaction rate equations, namely fractional SE1E₂IQR epidemic model, for analyzing the malware widespread in heterogeneous networks. In addition, for a better description of the realistic properties of Wireless Sensor Network, we considered WSNs as an Energy-Aware Barabási Albert scale-free network, that takes into account the energy consumption of network's nodes. Especially, the number of potentially disease-causing interactions are determined by using fuzzy logic theory and the compartment of exposure states is divided into Type 1-Exposed state and Type 2-Exposed state to better describe the realistic situation of exposure.
- (ii) It is a fact that the possibility for malware transmission is not uniform on heterogeneous complex networks, i.e., the transmission possibility of nodes with different degrees is un-similar. Moreover, depending upon the characteristic property of the network, the node's state change also affects the transmission. That is the reason why in this work, the effects of node's degree and node's state change in malware propagation are taken into account by using an appropriate Mamdani multi-input single-output (MISO) fuzzy system with 9 fuzzy rules, which helps us estimate the fuzzy rule-based interaction constant M_q .



Fig. 1. The five compartments of the SEIQR epidemic model.

- (iii) Based on the advantages of fractional calculus in the modeling of epidemic models, we proposed a network-based epidemic model with fractional derivatives in Caputo sense. The difficulties in the proof's technique when dealing with the fractional epidemic models compared with the integer one can be overcome by applying the fractional comparison principle, fractional stability theory and some basic tools of fractional calculus.
- (iv) By applying the next-generation matrix method, the basic reproductive ratio \mathscr{R}_0 corresponding to the fractional SE₁E₂IQR epidemic model is calculated. In addition, this work showed that the degree-dependent rate of newly born nodes does not affect the basic reproductive ratio \mathscr{R}_0 . Next, this work discussed the key role of the quantity $\mathscr{R}_0 1$ in the investigation of the asymptotic stability of the malware-free equilibrium point \mathbf{E}_0 and the unique existence of epidemic equilibrium point \mathbf{E}_* .
- (v) The direct Lyapunov's method with a suitable Lyapunov function $\mathbf{V}(\mathbf{x}(t))$ is used to discussing the necessary condition for the globally asymptotic stability of the malware-free equilibrium point \mathbf{E}_0 . The obtained condition claims that the condition $\Re_0 < 1$ is not good enough to completely eliminate the disease in the network.
- (vi) It is undeniable that the effects of quarantine treatments play a vital role in stifling the widespread of epidemic diseases. However, there have been several works that considered the effectiveness of quarantine on the disease controlling on the complex heterogeneous networks, see [10,31]. By the formula (7), we find that the epidemic threshold \Re_0 significantly depends on not only the topology of complex networks but also quarantine rates ω_1, ω_2, c . The higher the values quarantine rates are, the smaller value the basic reproductive ratio \Re_0 gets, which means that the malware spreading is controlled.

4. The fractional SE₁E₂IQR epidemic model on wireless sensor network

In this section, our aim is to characterize the infection of malware programs in wireless sensor networks and investigate the effectiveness of quarantine strategy against widespread malware. For this aim, we assume that the total population of sensor nodes is finite and classify the network into six following potential compartments (see Fig. 1).

In many classical epidemic models, we often suppose that all individuals mix uniformly and the rates of disease-causing contacts of all individuals are the same. In the mathematical viewpoint, this homogeneity certainly makes the analysis and evaluation simpler and more tractable; however, this assumption contradicts reality. Indeed, in many network-based models such as Facebook, World Wide Web or Wireless sensor network, etc., that make use of complex network topology of potential contacts, it is a fact that the number of links (connections) of different nodes in the networks may be not similar and of course, the effect of malicious object's attack to these nodes are also not the same. Therefore,



Fig. 2. The flowchart of malware propagation among the six compartments: Susceptible (S), Type 1-Exposed (E₁), Type 2-Exposed (E₂), Infectious (I), Quarantine (Q), Recovered (R).

the heterogeneity and the difference in the importance of nodes need to be taken into account when modeling the mathematical epidemic models for the malware's attack in a scale-free network. To deal with the heterogeneity in the complex networks, we classify the total population of nodes into *n* groups based on the number of links a node has per unit time (i.e., the degree distribution of a node). This means that the nodes in the *k*th group have the same degree, say *k*, for $1 \le k \le n$, and they are assumed to be dynamically equivalent.

Let us denote $S_k(t)$, $E_{1,k}(t)$, $E_{2,k}(t)$, $I_k(t)$, $Q_k(t)$ and $R_k(t)$ by the densities of susceptible nodes, type 1-exposed nodes, type 2-exposed nodes, infectious nodes, quarantined nodes and recovered nodes with degree k at time t, respectively for k = 1, 2, ..., n. In addition, the function $N_k(t)$ stands for the number of nodes with degree k at time t. Then, we directly get that

$$S(t) = \sum_{k=1}^{p} \mathbb{P}(k)S_{k}(t), \qquad E_{1}(t) = \sum_{k=1}^{p} \mathbb{P}(k)E_{1,k}(t), \qquad E_{2}(t) = \sum_{k=1}^{p} \mathbb{P}(k)E_{2,k}(t),$$
$$I(t) = \sum_{k=1}^{p} \mathbb{P}(k)I_{k}(t), \qquad Q(t) = \sum_{k=1}^{p} \mathbb{P}(k)Q_{k}(t), \qquad R(t) = \sum_{k=1}^{p} \mathbb{P}(k)R_{k}(t),$$

are the global average densities of the six epidemic compartments, respectively, where $\mathbb{P}(k)$ is the probability that a randomly chosen node has degree *k*. The infection of malware program in the network can be described in the following flowchart.

It is a fact that the speed of information transmission on the heterogeneous complex networks is profoundly influenced by geographical and climatic factors, which means that when studying the infection of malware on the networks, we need to consider the influence of natural factors to the transmission processes. In addition, the possibility for malware transmission is not uniform for nodes in complex networks, i.e., the transmission possibility of nodes with a different degree is un-similar. Moreover, depending upon the characteristic property of the network, the node's state change also affects to the transmission. For instance, in a sensor network, different network clusters will perform different sensing, measuring, and collecting tasks, and hence, the information's propagation speed is obviously uneven, which is also directly affect the infection of malicious codes in the network (see Fig. 3). This factor is often expressed in the node's number of state changes and often cannot be measured precisely but expressed through language variables. Moreover, the infectivity of a susceptible node also depends on the density of infectious nodes in its neighbors, that is, not every susceptible node that contacts an infectious node, will immediately become an infectious node. Furthermore, it is easy to see that in order to express the term of density, one cannot use exact values that are usually expressed through linguistic variables such as high, moderate, low. In this work, we propose to use a linguistic variable q ($q \in \{high, moderate, low\}$) to represent the uncertainties occurring in the model and try to take these factors into account in our considered epidemic model.

In the following, we try to assign three linguistic terms "low", "moderate", "high" with fuzzy values corresponding to fuzzy rules and use the fuzzy inferences to take these rules into the proposed epidemic model in the form of the infection constant M_q . Let us consider three fuzzy numbers $A_1 = (0, 0, 0.2, 0.3)$, $A_2 = (0.3, 0.5, 0.7)$ and $A_3 = (0.7, 0.8, 1, 1)$ to represent for three linguistic terms "low", "moderate", "high", respectively. In addition, we use two triangular fuzzy numbers $B_1 = (0, 0.3, 0.6)$ and $B_2 = (0.4, 0.7, 1.0)$ to determine that the output of combined rules will belong to Type 1-Exposure state or Type 2-Exposure state. (see Fig. 4).

Let x denote for the density of infectious nodes, y denote for the rate of node's state changes and z denote for the state of each rule's output. Now, we propose a Mamdani MISO fuzzy system with nine rules corresponding to our proposed model:



Fig. 3. Applications of Wireless Sensor Network in sensing, collecting and transmitting data.



Fig. 4. The fuzzy sets for linguistic terms "low", "moderate", "high".

Rule 1: If *x* is "LOW" and *y* is "LOW" then *z* is Type 1-Exposure state.

Rule 2: If *x* is "LOW" and *y* is "MODERATE" then *z* is Type 1-Exposure state.

Rule 3: If *x* is "MODERATE" and *y* is "LOW" then *z* is Type 1-Exposure state.

Rule 4: If *x* is "MODERATE" and *y* is "MODERATE" then *z* is Type 2-Exposure state.

Rule 5: If *x* is "LOW" and *y* is "HIGH" then *z* is Type 2-Exposure state.

Rule 6: If *x* is "MODERATE" and *y* is "HIGH" then *z* is Type 2-Exposure state.

Rule 7: If *x* is "HIGH" and *y* is "LOW" then *z* is Type 2-Exposure state.

Rule 8: If *x* is "HIGH" and *y* is "MODERATE" then *z* is Type 2-Exposure state.

Rule 9: If *x* is "HIGH" and *y* is "HIGH" then *z* is Type 2-Exposure state.

Example 4.1. In the following, we assume that the density of infectious nodes $x_0 = 0.35$ and the rate of node's state change $y_0 = 0.65$ then the firing strength of these rules can be given by

$f_1 = 0$	$f_2 = 0.365 \land 0.57 = 0.365$	$f_3 = 0$
$f_4 = 0.37 \land 0.57 = 0.37$	$f_5 = 0.365 \land 0.665 = 0.365$	$f_6 = 0.37 \land 0.665 = 0.37$
$f_7 = 0$	$f_8 = 0$	$f_9 = 0.$



Fig. 5. The output of the fuzzy inference system.

The output of fuzzy inference system is obtained by using following operations

$$C(z) = \left[\bigvee_{i=1}^{4} (f_i \wedge B_1(z))\right] \vee \left[\bigvee_{i=5}^{9} (f_i \wedge B_2(z))\right].$$

Then, the output C(z) can be graphically expressed in Fig. 5. which can be characterized in the following formula

$$C(z) = \begin{cases} \frac{10}{3}z & \text{if } 0 \le z \le 0.1095\\ 0.365 & \text{if } 0.1095 < z \le 0.427\\ -5z + \frac{5}{2} & \text{if } 0.427 < z \le 0.46\\ \frac{10}{3}x - \frac{4}{3} & \text{if } 0.46 < z \le 0.511\\ 0.37 & \text{if } 0.511 < z \le 0.889\\ -\frac{10}{3}z + \frac{10}{3} & \text{if } 0.889 < z \le 1\\ 0 & \text{otherwise.} \end{cases}$$

Then, by using the center of gravity (COG) defuzzification, the constant M_q is given by

$$M_q = \frac{\int_0^1 z C(z) dz}{\int_0^1 C(z) dz} \approx 0.633.$$

According to the flowchart given in Fig. 2, the interactions among these six compartments can be described by using the following rules:

- (r1) The rate of newly born nodes with degree k is degree-dependent and given by $\Lambda(k)$.
- (r2) A sensor node may also log out the network at a natural rate μ .
- (r3) When the epidemic disease is permanent on the network, a susceptible node with degree k can be infected by its neighbor infectious nodes. However, this susceptible node will not immediately become infectious, but it goes into one of exposure states (E₁) or (E₂). It becomes a type 1-exposed sensor node with a rate $\sigma_1(k)$, while if the density of neighbor infectious nodes reaches the threshold (determined by fuzzy rules), it goes into the type 2-exposed state at a rate $\sigma_2(k)$.
- (r4) Each type 1-exposed node is assumed to go into type 2-exposure state (E_2) at a rate η if the number of type 1-exposed individual's infectious neighbor reaches the threshold determined by fuzzy rules.
- (r5) With the use of detection programs, exposed nodes are isolated from the network and moved to Quarantined state (Q) with the rate ω_1 and ω_2 , respectively.
- (r6) Each type 2-exposed node is assumed to become infectious at a rate ω_3 .
- (**r7**) The viral detection programs find out infectious source, and move them out of the network at a rate of *c*. These nodes are then changed into the quarantined state.

Some used	Some used parameters.				
Para.	Description	Unit			
$\Lambda(k)$	The degree-dependent rate of newly born nodes with degree k	PUT			
с	The rate of being isolated of nodes in infectious state	PUT			
μ	The natural death rate of nodes	PUT			
$\sigma_1(k)$	The degree-dependent infection rate from susceptible state to type 1-exposed nodes	PUT			
$\sigma_2(k)$	The degree-dependent infection rate from susceptible state to type 2-exposed state	PUT			
ω_1	The isolated rate of type 1-exposed nodes	PUT			
ω_2	The isolated rate of type 2- exposed state	PUT			
ω_3	The rate for a type 2-exposed node becomes infectious	PUT			
θ	The rate of being susceptible of recovered nodes	PUT			
r_1	The recovery rate of infectious nodes	PUT			
r_2	The recovery rate of quarantined nodes	PUT			
η	The transition rate from the type 1-exposed state to the type 2-exposed state	PUT			

PUT: per unit time.

- (r8) Each node in Quarantined state (Q) can be temporarily immune and get recovered at a rate r_2 .
- (r9) With the actions of anti-malware programs, each infectious node gets recovered with the rates r_1 . In addition, each recovered sensor node can become susceptible towards the possible attack of malware at a rate θ .

For simplicity, we summarize the used parameters and their descriptions in Table 1.

Now, we formulate a mathematical model that describes the malware propagation between six compartments (S), (E₁), (E₂), (I), (Q), and (R) in a Wireless sensor network along with the data transmission. In this work, we take into account the non-local property with memory effects of data diffusion and aim to accurately represent this characteristic in the rate of change of the proposed model. Thus, we suggest using the Caputo fractional-order derivative to establish the differential model for malware propagation. Based on the above hypotheses and notations, the SE₁E₂IQR epidemic model can be described by the following dynamical mean-field reaction rate equations:

$$\begin{cases} {}_{0}^{\mathcal{O}}\mathfrak{D}_{t}^{k}S_{k}(t) &= \Lambda(k) - (\sigma_{1}(k) + \sigma_{2}(k))S_{k}(t)\Theta(t) - \mu S_{k}(t) + \theta R_{k}(t) \\ {}_{0}^{\mathcal{O}}\mathfrak{D}_{t}^{k}E_{1,k}(t) &= \sigma_{1}(k)S_{k}(t)\Theta(t) - (\eta + \omega_{1} + \mu)E_{1,k}(t) \\ {}_{0}^{\mathcal{O}}\mathfrak{D}_{t}^{k}E_{2,k}(t) &= \sigma_{2}(k)S_{k}(t)\Theta(t) - (\mu + \omega_{2} + \omega_{3})E_{2,k}(t) + \eta E_{1,k}(t) \\ {}_{0}^{\mathcal{O}}\mathfrak{D}_{t}^{k}I_{k}(t) &= \omega_{3}E_{2,k}(t) - (\mu + c + r_{1})I_{k}(t) \\ {}_{0}^{\mathcal{O}}\mathfrak{D}_{t}^{k}Q_{k}(t) &= \omega_{1}E_{1,k}(t) + \omega_{2}E_{2,k}(t) + cI_{k}(t) - (r_{2} + \mu)Q_{k}(t) \\ {}_{0}^{\mathcal{O}}\mathfrak{D}_{t}^{k}R_{k}(t) &= r_{1}I_{k}(t) + r_{2}Q_{k}(t) - (\mu + \theta)R_{k}(t), \end{cases}$$
(1)

subject to the initial conditions

$$S_k(0) = S_k^0, \ E_{1,k}(0) = E_{1,k}^0, \ E_{2,k}(0) = E_{2,k}^0, \ I_k(0) = I_k^0, \ Q_k(0) = Q_k^0, \ R_k(0) = R_k^0,$$
(2)

where $\sigma_1(k)$, $\sigma_2(k)$ are the degree-dependent infection rates given by $\sigma_1(k) = \sigma_1 k$, $\sigma_2(k) = \sigma_2 k$, respectively and the other parameters are assumed to be positive. In addition, we assume that the initial number of nodes with degree k is given by

$$N_k(0) = S_k(0) + E_{1,k}(0) + E_{2,k}(0) + I_k(0) + Q_k(0) + R_k(0) = \frac{\Lambda(k)}{\mu}.$$
(3)

By summing up six fractional differential equations of the system (1), we obtain

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\left(S_{k}+E_{1,k}+E_{2,k}+I_{k}+Q_{k}+R_{k}\right)(t)=\Lambda(k)-\mu\left(S_{k}+E_{1,k}+E_{2,k}+I_{k}+Q_{k}+R_{k}\right)(t)$$

or equivalently,

$${}^C_0 \mathfrak{D}^{\beta}_t N_k(t) = \Lambda(k) - \mu N_k(t).$$
(4)

By taking the Laplace transform for the fractional differential Eq. (4), we get

$$s^{\beta} \mathscr{L}\{N_k(t)\}(s) - s^{\beta-1}N_k(0) = \frac{\Lambda(k)}{s} - \mu \mathscr{L}\{N_k(t)\}(s).$$

Table 2

Some	notations	and	abbreviatio	r

Notation	Description	Notation	Description
MFE	Malware-free equilibrium	α_4	$\mu + \theta$
EE	Epidemic equilibrium	α_5	$r_1 + c + \mu$
α_1	$\omega_1 + \mu + \eta$	$\alpha_{6,k}$	$\eta \sigma_1(k) + \alpha_1 \sigma_2(k)$
α ₂	$\omega_2 + \omega_3 + \mu$	$\alpha_{7,k}$	$\alpha_2 \alpha_5 \sigma_1(k) \omega_1 + \alpha_5 \alpha_{6,k} \omega_2 + c \alpha_3 \alpha_{6,k} \omega_3$
α ₃	$r_2 + \mu$	b_k	$\frac{\Lambda(k)}{\mu}$

which follows that $\mathscr{L}\{N_k(t)\}(s) = \frac{s^{\beta-1}}{s^{\beta} + \mu}N_k(0) + \frac{\Lambda(k)s^{\beta-(1+\beta)}}{s^{\beta} + \mu}$. Then, by taking the inverse Laplace transform and the initial condition (3), we receive

$$N_k(t) = \frac{\Lambda(k)}{\mu} \mathbb{E}_{\beta}(-\mu t^{\beta}) + \Lambda(k) t^{\beta} \mathbb{E}_{\beta,\beta+1}(-\mu t^{\beta})$$

By applying the transformation $\mathbb{E}_{\beta_1,\beta_2}(z) = z\mathbb{E}_{\beta_1,\beta_1+\beta_2}(z) + \frac{1}{\Gamma(\beta_2)}$ for $\beta_1 = \beta$, $\beta_2 = 1$ and $z = -\mu t^{\beta}$, we directly get that

$$N_k(t) = \frac{\Lambda(k)}{\mu} \mathbb{E}_{\beta}(-\mu t^{\beta}) + \frac{\Lambda(k)}{\mu} \left[1 - \mathbb{E}_{\beta,1}(-\mu t^{\beta}) \right] = \frac{\Lambda(k)}{\mu} := b_k,$$

which means that the total number of nodes with degree *k* is only degree-dependent.

The function $\Theta(t)$ is the probability that a given link points to an infectious individual and has the following general form

$$\Theta(t) = M_q \sum_{i=1}^n \frac{1}{i} \nu(i) \mathbb{P}(i|k) \frac{I_i(t)}{N_i(t)}$$

where the components of $\Theta(t)$ are explained as follows:

- The parameter M_q is the output of multi-input single-output (MISO) fuzzy system that is inferred from fuzzy rules for linguistic variable $q \in \{\text{high, moderate, low}\}$. The detailed description of M_q will be specified later.
- The function v(i) represents for the average number of links from which an infectious node with degree *i* will propagate the malware program to other nodes. It is called the infectivity of the node with degree *i* and assumed to be $v(k) \le k$ for each k = 1, 2, ..., n.
- The fraction $\frac{1}{i}$ represents the probability that one of the degree *i*-infectious neighbor of a given node connect to this node at a time step.
- The notation $\mathbb{P}(i|k)$ is the well-known conditional probability that a node with degree *k* links to a node with degree *i*. In this work, we consider the probability $\mathbb{P}(i|k)$ as follows:

$$\mathbb{P}\left(i|k\right) = \frac{i\mathbb{P}(i)}{\langle k \rangle}$$

Here, the term $\langle k \rangle = \sum_{k=1}^{n} k \mathbb{P}(k)$ is the average degree within the network.

In summary, the function $\Theta(t)$ is given as follows:

$$\Theta(t) = \frac{M_q}{\langle k \rangle} \sum_{i=1}^n \frac{\nu(i)}{b_i} \mathbb{P}(i) I_i(t).$$

5. The mathematical analysis of the fractional SE₁E₂IQR epidemic model

For simplicity in representation, we introduce a table of notations and abbreviations (see Table 2).

5.1. The positiveness and boundedness of the solution to the proposed model

For simplicity, we use the following state vector forms

 $\mathbf{x}^{k}(t) = \begin{bmatrix} E_{1,k}(t) & E_{2,k}(t) & I_{k}(t) & S_{k}(t) & Q_{k}(t) & R_{k}(t) \end{bmatrix}^{\top} \quad \text{for } k = 1, 2, \dots, n,$ $\mathbf{x}(t) = \begin{bmatrix} \mathbf{x}^{1}(t) & \mathbf{x}^{2}(t) & \cdots & \mathbf{x}^{n}(t) \end{bmatrix}.$ In addition, we denote the right-hand side of (1) by $f(\mathbf{x}^{k}(t))$, that is,

$$f(\mathbf{x}^{k}(t)) = \begin{bmatrix} f_{1}(\mathbf{x}^{k}(t)) \\ f_{2}(\mathbf{x}^{k}(t)) \\ f_{3}(\mathbf{x}^{k}(t)) \\ f_{4}(\mathbf{x}^{k}(t)) \\ f_{5}(\mathbf{x}^{k}(t)) \\ f_{5}(\mathbf{x}^{k}(t)) \\ f_{6}(\mathbf{x}^{k}(t)) \end{bmatrix} = \begin{bmatrix} \Lambda(k) - (\sigma_{1}(k) + \sigma_{2}(k)) S_{k}(t)\Theta(t) - \mu S_{k}(t) + \theta R_{k}(t) \\ \sigma_{1}(k)S_{k}(t)\Theta(t) - (\mu + \omega_{2} + \omega_{3}) E_{2,k}(t) + \eta E_{1,k}(t) \\ \omega_{3}E_{2,k}(t) - (\mu + c + r_{1})I_{k}(t) \\ \omega_{1}E_{1,k}(t) + \omega_{2}E_{2,k}(t) + cI_{k}(t) - (r_{2} + \mu)Q_{k}(t) \\ r_{1}I_{k}(t) + r_{2}Q_{k}(t) - (\mu + \theta)R_{k}(t) \end{bmatrix}$$

for each k = 1, 2, ..., n. Thus, the vector-valued function $\mathscr{F}(\mathbf{x}(t))$ is defined by

$$\mathscr{F}(\mathbf{x}(t)) = \begin{bmatrix} f(\mathbf{x}^{1}(t)) & f(\mathbf{x}^{2}(t)) & \cdots & f(\mathbf{x}^{n}(t)) \end{bmatrix}.$$

Now, we begin this section with the unique existence of positive solution and invariance results for the fractional differential system (1).

Theorem 5.1. If the initial condition $\mathbf{x}^{k}(0) \ge 0$ for all $k = \overline{1, n}$ then the fractional $SE_{1}E_{2}IQR$ epidemic model (1) with the initial condition (2) always has a unique non-negative solution $\mathbf{x}^{k}(t)$ and the function $\Theta(t) > 0$ for all $k = \overline{1, n}$ and t > 0. Moreover, the compact set

$$\Sigma^{+} = \left\{ \mathbf{x}(t) \in \mathbb{R}^{6n}_{+} : S_k + E_{1,k} + E_{2,k} + I_k + Q_k + R_k = b_k, \ k = \overline{1, n} \right\}.$$

is a positively invariant set of the epidemic model.

Proof. The proof of this theorem is divided into the following steps:

(Uniqueness and existence). For each $k = \overline{1, n}$, the fractional SE₁E₂IQR epidemic model (1) can rewritten in the following compact form

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\mathbf{x}^{k}(t) = f(\mathbf{x}^{k}(t)),$$

with the initial condition $\mathbf{x}^{k}(0) = \mathbf{x}_{0}^{k}$. The Jacobian matrix of the function $f(\mathbf{x}^{k}(t))$ is given by

	Γo	0	$\frac{-(\sigma_1(k)+\sigma_2(k))M_qv(k)\mathbb{P}(k)}{b_k\langle k\rangle}S_k(t)$	$-(\sigma_1(k) + \sigma_2(k))\Theta(t)$	0	θ	
af(wk)	$-\alpha_1$	0	$\frac{-\sigma_1(k)M_qv(k)\mathbb{P}(k)}{b_k\langle k\rangle}S_k(t)$	$-\sigma_1(k)\Theta(t)$	0	0	
$\frac{\partial f(\mathbf{x}^{k})}{\partial \mathbf{x}^{k}} =$	η	$-\alpha_2$	$\frac{-\sigma_2(k)M_q\nu(k)\mathbb{P}(k)}{b_k\langle k\rangle}S_k(t)$	$-\sigma_2(k)\Theta(t)$	0	0	.
	0	ω_3	$-\alpha_5$	0	0	0	
	ω_1	ω_2	-c	0	$-\alpha_3$	0	
	0	0	<i>r</i> ₁	0	r_2	$-\alpha_4$	

We can see that the Jacobian matrix $\frac{\partial f(\mathbf{x}^k)}{\partial \mathbf{x}^k}$ is a continuous function on \mathbb{R}^6_+ and hence, according to Remark 1.2.1 in [35], it implies that $f(\mathbf{x}^k(t))$ is a locally Lipschitz vector-valued function on \mathbb{R}^6_+ . Finally, by applying Theorem 1.3.1 and Theorem 1.4.1 of [35], we can conclude that Cauchy problem (1) - (2) has a unique solution for every $\mathbf{x}^k_0 \ge 0$.

(Positiveness). The assumption $\mathbf{x}^k(0) \ge 0$ implies that $S_k^0, E_{1,k}^0, E_{2,k}^0, I_k^0, Q_k^0$ and R_k^0 are all non-negative for each $k = \overline{1, n}$. Now, we will prove that for every $\mathbf{x}_0^k \ge 0$, the unique solution $\mathbf{x}(t)$ of Cauchy problem (1)–(2) is non-negative. For this aim, we consider the following cases:

Case 1: Assume that the functions $S_k(t)$ is positive on \mathbb{R}_+ . Then, we consider

According to Example 4.9 in [36], the general solution of the above fractional differential equations are

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$$\begin{cases} E_{1,k}(t) = E_{1,k}^{0} \mathbb{E}_{\beta} \left(-\alpha_{1} t^{\beta} \right) + \int_{0}^{t} \frac{\mathbb{E}_{\beta,\beta} \left(-\alpha_{1} (t-\tau)^{\beta} \right)}{(t-\tau)^{1-\beta}} \sigma_{1}(k) S_{k}(\tau) \Theta(\tau) d\tau \\ E_{2,k}(t) = E_{2,k}^{0} \mathbb{E}_{\beta} \left(-\alpha_{2} t^{\beta} \right) + \int_{0}^{t} \frac{\mathbb{E}_{\beta,\beta} \left(-\alpha_{2} (t-\tau)^{\beta} \right)}{(t-\tau)^{1-\beta}} \left[\sigma_{2}(k) S_{k}(\tau) \Theta(\tau) + \eta E_{1,k}(\tau) \right] d\tau \\ Q_{k}(t) = Q_{k}^{0} \mathbb{E}_{\beta} \left(-\alpha_{3} t^{\beta} \right) + \int_{0}^{t} \frac{\mathbb{E}_{\beta,\beta} \left(-\alpha_{3} (t-\tau)^{\beta} \right)}{(t-\tau)^{1-\beta}} \left[\omega_{1} E_{1,k}(\tau) + \omega_{2} E_{2,k}(\tau) + c I_{k}(\tau) \right] d\tau \\ R_{k}(t) = R_{k}^{0} \mathbb{E}_{\beta} \left(-\alpha_{4} t^{\beta} \right) + \int_{0}^{t} \frac{\mathbb{E}_{\beta,\beta} \left(-\alpha_{4} (t-\tau)^{\beta} \right)}{(t-\tau)^{1-\beta}} \left[r_{1} I_{k}(\tau) + r_{2} Q_{k}(\tau) \right] d\tau, \end{cases}$$
(5)

where $t \ge 0$ and $\mathbb{E}_{\beta,\beta}(\lambda t)$ is the well-known Mittag–Leffler function (see Chap. 1 of [36]). Now, we consider two following sub-cases:

Sub-case 1.1: Assume that the function $I_k(t)$ is non-negative on \mathbb{R}^+ for each $k = \overline{1, n}$, which means that the disease is persistent on the *k*th-group. Thus, the function

$$\Theta(t) = \frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\nu(k)}{b_k} \mathbb{P}(k) I_k(t)$$

is non-negative for all $t \ge 0$. Next, from the system (5), we directly get that $E_{1,k}(t), E_{2,k}(t), Q_k(t)$ and $R_k(t)$ are also non-negative functions for all $t \ge 0$.

In addition, due to the assumption that the disease is present on the network, there always exists at least one $k_0 \ge 1$ such that $I_{k_0}(0) > 0$ and $I_k(0) \ge 0$ for all $k = \overline{1, n}$. Thus, it implies that $\Theta(0) > 0$. From the fourth fractional differential equation of the system (1), we deduce that

$$\begin{split} {}^{C}_{0}\mathfrak{D}^{\beta}_{t}\Theta(t) &= \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\nu(k)}{b_{k}} \mathbb{P}(k)^{C}_{0}\mathfrak{D}^{\beta}_{t}I_{k}(t) = \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\nu(k)}{b_{k}} \mathbb{P}(k) \left[\omega_{3}E_{2,k}(t) - \alpha_{5}I_{k}(t) \right] \\ &\geq -\alpha_{5}\frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\nu(k)}{b_{k}} \mathbb{P}(k)I_{k}(t) = -\alpha_{5}\Theta(t). \end{split}$$

Then, the fractional comparison principle (Lemma 10, [37]) implies that $\Theta(t) \ge \Theta(0)\mathbb{E}_{\beta}(-\alpha_5 t^{\beta}) > 0$ for all t > 0. The proof is completed.

Sub-case 1.2: Assume that there exists a constant $t_1 > 0$ such that $I_k(t) > 0$ for all $t \in [0, t_1)$, $I_k(t_1) = 0$ and $I_k(t) < 0$ for $t > t_1$. Firstly, the presence of malware program on the network implies that the function $\Theta(t) > 0$ for all $t \in [0, t_1]$. Then, for each $t \in [0, t_1]$, the second differential equation of (1) implies that

$$E_{1,k}(t) = E_{1,k}^{0} \mathbb{E}_{\beta} \left(-\alpha_{1} t^{\beta} \right) + \int_{0}^{t} \frac{\mathbb{E}_{\beta,\beta} \left(-\alpha_{1} (t-\tau)^{\beta} \right)}{(t-\tau)^{1-\beta}} \sigma_{1}(k) S_{k}(\tau) \Theta(\tau) d\tau > 0$$

As a consequence, we directly get that

$$E_{2,k}(t) = E_{2,k}^0 \mathbb{E}_\beta \left(-\alpha_2 t^\beta \right) + \int_0^t \frac{\mathbb{E}_{\beta,\beta} \left(-\alpha_2 (t-\tau)^\beta \right)}{(t-\tau)^{1-\beta}} \left[\sigma_2(k) S_k(\tau) \Theta(\tau) + \eta E_{1,k}(\tau) \right] d\tau > 0$$

for all $t \in [0, t_1]$ and $k = \overline{1, n}$. Next, by substituting the inequality $E_{2,k}(t) > 0$ into the fourth fractional differential equation, we immediately receive

$$I_{k}(t) = I_{k}^{0} \mathbb{E}_{\beta} \left(-\alpha_{5} t^{\beta} \right) + \omega_{3} \int_{0}^{t} (t-\tau)^{\beta-1} \mathbb{E}_{\beta,\beta} \left(-\alpha_{5} (t-\tau)^{\beta} \right) E_{2,k}(\tau) d\tau > 0$$

for all $t \in [0, t_1]$, which contradicts to the assumption $I_k(t_1) = 0$. Hence, we implies that this sub-case cannot occur.

Case 2: Notice that the initial susceptible population $S_k(0) > 0$. Assume that there exists the first time $t_0 > 0$ such that $S_k(t_0) = 0$ and the function $S_k(t)$ is positive for all $t \in [0, t_0)$. Then, two following sub-cases can occur:

Sub-case 2.1: If the function $I_k(t)$ is positive on \mathbb{R}_+ then according to the solution formulas (5), it implies that the function $\Theta(t) > 0$ and the functions $E_{1,k}(t), E_{2,k}(t), Q_k(t)$ and $R_k(t)$ are all non-negative on the interval $[0, t_0]$ for each $k = \overline{1, n}$. Thus, for each $t \in [0, t_0]$, we have

$$C_0^C \mathfrak{D}_t^\beta S_k(t) = - \left[(\sigma_1(k) + \sigma_2(k)) \, \Theta(t) + \mu \right] S_k(t) + \Lambda(k) + \theta R_k(t)$$

$$\geq -M_1 S_k(t) + \Lambda(k) + \theta R_k(t),$$

where $\kappa_1 = \max_{[0,t_0]} \{ (\sigma_1(k) + \sigma_2(k)) \Theta(t) + \mu \}$. Thus, it yields

$$S_k(t) \geq S_k(0)\mathbb{E}_{\beta}(-K_1t^{\beta}) + \int_0^t (t-\tau)^{\beta-1}\mathbb{E}_{\beta,\beta}\left(-K_1(t-\tau)^{\beta}\right)\left[\Lambda(k) + \theta R_k(\tau)\right]d\tau > 0$$

for all $t \in [0, t_0]$, which contradicts to the assumption $S_k(t_0) = 0$. Hence, this case cannot occur.

Sub-case 2.2: Assume that there exists the first time $t_1 > 0$ such that $I_k(t) > 0$ for all $t \in [0, t_1)$, $I_k(t_1) = 0$ and $I_k(t) < 0$ for all $t > t_1$. Then, there are two possibilities as below:

- If $t_0 = \min\{t_0, t_1\}$ then it implies that $S_k(t) \ge 0$ and $I_k(t) > 0$ for all $t \in [0, t_0]$. From the system (5), we imply that the functions $E_{1,k}(t), E_{2,k}(t), Q_k(t)$ and $R_k(t)$ are non-negative for all $t \in [0, t_0]$. By doing similar arguments as in Sub-case 1.1, we deduce that $S_k(t_0) > 0$, that leads to the contradiction.
- If $t_1 = \min\{t_0, t_1\}$ then it implies that $S_k(t) > 0$ and $I_k(t) \ge 0$ for all $t \in [0, t_1]$. By doing similar arguments as in Sub-case 1.2, we can conclude that $I_k(t_1)$ is always positive, that leads to a contradiction and hence, this case cannot occur.

Therefore, we can conclude that the fractional $\underline{SE}_1\underline{E}_2IQR$ epidemic model (1) with the initial condition (2) always admits a unique non-negative solution $\mathbf{x}^k(t)$ for all $k = \overline{1, n}$ and the function $\Theta(t) > 0$ for t > 0.

(**Positively invariant**). Now, our aim is to prove that the set Σ^+ is a positively invariant set corresponding to the fractional differential model (1). It is well-known that the initial conditions (2) are assumed to satisfy Eq. (3) and hence, $\mathbf{x}^k(0) \in \Sigma^+$. Next, by summing up the six fractional differential equations of the fractional SE₁E₂IQR model (1), we directly obtain

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}N_{k}(t)=\Lambda(k)-\mu N_{k}(t),$$

which solves $N_k(t) = S_k(t) + E_{1,k}(t) + E_{2,k}(t) + I_k(t) + Q_k(t) + R_k(t) = b_k$ for all $k = \overline{1, n}$. Finally, by combining with the non-negativeness of the solution $\mathbf{x}(t)$, we deduce that the set Σ^+ is a positively invariant set to the proposed system. \Box

5.2. The existence of equilibrium points and basic reproductive ratio \mathscr{R}_0 of the proposed model

An important perspectives of the proposed epidemic model is to determine whether the network is able to survive under the attacking of malware. For this aim, we need to discuss the asymptotic stability of equilibrium points of the proposed epidemic model consisting of MFE point and EE point. Here, the MFE point and EE point can be determined by solving the following system of equations

$$\begin{aligned} \Lambda(k) &- (\sigma_1(k) + \sigma_2(k)) S_k(t) \Theta(t) - \mu S_k(t) + \theta R_k(t) = 0 \\ \sigma_1(k) S_k(t) \Theta(t) - (\eta + \omega_1 + \mu) E_{1,k}(t) = 0 \\ \sigma_2(k) S_k(t) \Theta(t) - (\mu + \omega_2 + \omega_3) E_{2,k}(t) + \eta E_{1,k}(t) = 0 \\ \omega_3 E_{2,k}(t) - (\mu + c + r_1) I_k(t) = 0 \\ \omega_1 E_{1,k}(t) + \omega_2 E_{2,k}(t) + c I_k(t) - (r_2 + \mu) Q_k(t) = 0 \\ r_1 I_k(t) + r_2 Q_k(t) - (\mu + \theta) R_k(t) = 0. \end{aligned}$$
(6)

Firstly, we give the existence result of the malware-free equilibrium point of the proposed epidemic model

Theorem 5.2. The fractional SE_1E_2IQR epidemic model (1) always admits an MFE point

$$\mathbf{E}_{0} = \underbrace{(0, 0, 0, b_{1}, 0, 0, \dots, 0, 0, 0, b_{n}, 0, 0)}_{6n}$$

Proof. We can directly check that $(0, 0, 0, b_k, 0, 0)$ satisfies the system (6) for all k = 1, 2, ..., n. Thus, it implies that the point

$$\mathbf{E}_0 = \underbrace{(0, 0, 0, b_1, 0, 0, \dots, 0, 0, 0, b_n, 0, 0)}_{6n}$$

is an equilibrium point of the proposed model (1), namely the malware-free equilibrium (MFE) point. \Box

Next, by applying the next-generation method, we can determine the basic reproductive ratio \mathscr{R}_0 of the fractional SE₁E₂IQR epidemic model (1). Indeed, the infection in wireless sensor network for the proposed model has following characteristic properties:

- There are only three infection causing compartments of the epidemic model (1), that are Type 1-Exposed (E₁), Type 2-Exposed (E₂) and Infectious (I).
- The propagation from the exposure compartment to the infectious compartment (I) or the transition between two exposure compartments are only the spread of an infected individual through the various compartments.

According to epidemiological theory, it follows that the gain term and loss term corresponding to the fractional SE₁E₂IQR epidemic model (1) is

- Gain terms: $\begin{bmatrix} \sigma_1(k)S_k(t)\Theta(t) & \sigma_2(k)S_k(t)\Theta(t) & 0 \end{bmatrix}^\top$. Loss terms: $\begin{bmatrix} \alpha_1E_{1,k} & -\eta E_{1,k}(t) + \alpha_2E_{2,k}(t) & -\omega_3E_{2,k}(t) + \alpha_5I_k(t) \end{bmatrix}^\top$.

Let \mathcal{O}_n and I_n be the $n \times n$ zero matrix and $n \times n$ identity matrix, respectively. Then, the basic reproductive ratio \mathscr{R}_0 is determined as follows:

Step 1: The rate matrix \mathcal{F} of new infection's appearance at \mathbf{E}_0 is $\mathcal{F} = \begin{bmatrix} \mathcal{O}_n & \mathcal{O}_n & \mathcal{O}_n \\ \mathcal{O}_n & \mathcal{O}_n & \mathcal{O}_n \\ \sigma_1 \mathcal{B} & \sigma_2 \mathcal{B} & \mathcal{O}_n \end{bmatrix}$, where \mathcal{B} is a block matrix given by

 $\mathcal{B} = \frac{M_q}{\langle k \rangle} \begin{bmatrix} b_1 \\ 2b_2 \\ \vdots \\ \vdots \\ nb \end{bmatrix} \begin{bmatrix} \frac{\nu(1)}{b_1} \mathbb{P}(1) & \frac{\nu(2)}{b_2} \mathbb{P}(2) & \cdots & \frac{\nu(n)}{b_n} \mathbb{P}(n) \end{bmatrix}.$

Step 2: The transition matrix \mathcal{V} of infected states is $\mathcal{V} = \begin{bmatrix} \alpha_1 \mathbf{I}_n & -\eta \mathbf{I}_n & \mathcal{O}_n \\ \mathcal{O}_n & \alpha_2 \mathbf{I}_n & -\omega_3 \mathbf{I}_n \\ \mathcal{O}_n & \mathcal{O}_n & \alpha_5 \mathbf{I}_n \end{bmatrix}$.

Step 3: The basic reproductive ratio \mathscr{R}_0 is the largest eigenvalue of the matrix \mathcal{FV}^{-1} given by

$$\mathcal{FV}^{-1} = \begin{bmatrix} \mathcal{O}_n & \mathcal{O}_n & \mathcal{O}_n \\ \mathcal{O}_n & \mathcal{O}_n & \mathcal{O}_n \\ \sigma_1 \mathcal{B} & \frac{\eta \sigma_1 + \alpha_1 \sigma_2}{\alpha_1 \alpha_2} \mathcal{B} & \frac{\omega_3 (\eta \sigma_1 + \alpha_1 \sigma_2)}{\alpha_1 \alpha_2 \alpha_5} \mathcal{B} \end{bmatrix}$$

Finally, the basic reproductive ratio \Re_0 of the fractional SE₁E₂IQR epidemic model (1) is given by

$$\mathscr{R}_{0} = \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\omega_{3} \nu(k) \mathbb{P}(k) (\eta \sigma_{1}(k) + \alpha_{1} \sigma_{2}(k))}{\alpha_{1} \alpha_{2} \alpha_{5}} = \frac{\omega_{3} M_{q} \langle \alpha_{6} \nu \rangle}{\alpha_{1} \alpha_{2} \alpha_{5} \langle k \rangle}, \tag{7}$$

where $\langle \alpha_6 \nu \rangle = \sum_{k=1}^n \nu(k) \mathbb{P}(k) (\eta \sigma_1(k) + \alpha_1 \sigma_2(k)).$

Remark 5.1. According to the formula (7), we can see that the basic reproductive ratio \mathscr{R}_0 has no relationship with the degree-dependent birth rate $\Lambda(k)$, which claims that the change of birth rate does not play any role in the widespread of disease. However, the number \mathscr{R}_0 is directly proportional to the value of the heterogeneous factor $\frac{\langle \alpha_6 \nu \rangle}{\langle k \rangle}$, which means that the network heterogeneity can make malware programs easier to spread in the network.

Theorem 5.3. Consider the fractional SE_1E_2IQR epidemic model (1) and the basic reproductive ratio defined by (7). If $\Re_0 > 1$ then there exists a unique epidemic equilibrium point

$$\mathbf{E}_{*} = \left\{ \left(E_{1,k}^{*}, E_{2,k}^{*}, I_{k}^{*}, S_{k}^{*}, Q_{k}^{*}, R_{k}^{*} \right) \right\}_{k=1}^{n} \\ = \left(E_{1,1}^{*}, E_{2,1}^{*}, I_{1}^{*}, S_{1}^{*}, Q_{1}^{*}, R_{1}^{*}, \dots, E_{1,n}^{*}, E_{2,n}^{*}, I_{n}^{*}, S_{n}^{*}, Q_{n}^{*}, R_{n}^{*} \right),$$

where

$$S_{k}^{*} = \frac{\alpha_{1}\alpha_{2}\alpha_{5}}{\omega_{3}\alpha_{6,k}\Theta^{*}}I_{k}^{*}, \qquad E_{1,k}^{*} = \frac{\alpha_{2}\alpha_{5}\sigma_{1}(k)}{\omega_{3}\alpha_{6,k}}I_{k}^{*}, \qquad E_{2,k}^{*} = \frac{\alpha_{5}}{\omega_{3}}I_{k}^{*}, \qquad Q_{k}^{*} = \frac{\alpha_{7,k}}{\omega_{3}\alpha_{3}\alpha_{6,k}}I_{k}^{*}, \qquad R_{k}^{*} = \frac{r_{1}\alpha_{3}\alpha_{6,k}\omega_{3} + r_{2}\alpha_{7,k}}{\alpha_{3}\alpha_{4}\alpha_{6,k}\omega_{3}}I_{k}^{*}, \qquad \Theta^{*} = \frac{M_{q}}{\langle k \rangle}\sum_{i=1}^{n}\frac{\nu(i)}{b_{i}}\mathbb{P}(i)I_{i}^{*} \qquad (8)$$

$$I_{k}^{*} = \frac{b_{k}\alpha_{3}\alpha_{4}\alpha_{6,k}\omega_{3}\Theta^{*}}{\left[\alpha_{3}\alpha_{4}\alpha_{6,k}\omega_{3} + \alpha_{3}\alpha_{4}\alpha_{5}\alpha_{6,k} + \alpha_{4}\alpha_{7,k} + \alpha_{2}\alpha_{3}\alpha_{4}\alpha_{5}\sigma_{1}(k) + r_{1}\alpha_{3}\alpha_{6,k}\omega_{3} + r_{2}\alpha_{7,k}\right]\Theta^{*} + \alpha_{1}\alpha_{2}\alpha_{3}\alpha_{4}\alpha_{5}}.$$

Proof. In order to evaluate the epidemic equilibrium point $\mathbf{E}_* = \{(E_{1,k}^*, E_{2,k}^*, I_k^*, S_k^*, Q_k^*, R_k^*)\}_{k=1}^n$, we need to impose the

right-hand side of the fractional system (1) to be equal to zero. Thus, we will prove that the tuple $(E_{1,k}^*, E_{2,k}^*, I_k^*, S_k^*, Q_k^*, R_k^*)$

Table 3

The	sensitivity	indices	of	model's	parameters.	
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No	Para.	Description	Sensitivity index
1	M_q	The fuzzy rule-based interaction constant	+1
2	$\langle k \rangle$	The average degree within the network	-1
3	С	The quarantine rate of Infectious nodes	-0.6538
4	ω_1	The quarantine rate of Type 1-exposed nodes	-0.1448
5	ω_2	The quarantine rate of Type 2-exposed nodes	-0.333
6	ω_3	The rate of being infectious of Type 2-exposed nodes	0.444
7	η	The transition rate from the type 1-exposed state to the type 2-exposed state	0.20276

satisfies the following system of equations

$$\begin{cases} S_k^* + E_{1,k}^* + E_{2,k}^* + I_k^* + Q_k^* + R_k^* = b_k \\ \sigma_1 S_k^* \Theta^* - \alpha_1 E_{1,k}^* = 0 \\ \sigma_2(k) S_k^* \Theta^* - \alpha_2 E_{2,k}^* + \eta E_{1,k}^* = 0 \\ \omega_3 E_{2,k}^* - \alpha_5 I_k^* = 0 \\ \omega_1 E_{1,k}^* + \omega_2 E_{2,k}^* + c I_k^* - \alpha_3 Q_k^* = 0 \\ r_1 I_k^* + r_2 Q_k^* - \alpha_4 R_k^* = 0, \end{cases}$$

for each k = 1, 2, ..., n, where the term Θ^* is given by $\Theta^* = \frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\nu(k)}{b_k} \mathbb{P}(k) I_k^*$ and the coefficients of (9) are given in Table 3. Then, from the fourth equation of (9), we immediately obtain $E_{2,k}^* = \frac{\alpha_5}{\omega_3} I_k^*$. Next, by combining the second and third equations of the system (9), we receive

$$E_{1,k}^* = \frac{\alpha_2 \alpha_5 \sigma_1(k)}{\left[\alpha_1 \sigma_2(k) + \eta \sigma_1(k)\right] \omega_3} I_k^* = \frac{\alpha_2 \alpha_5 \sigma_1(k)}{\alpha_{6,k} \omega_3} I_k^*.$$

Thus, by substituting $E_{1,k}^*$ into the second equation, it yields $S_k^* = \frac{\alpha_1}{\sigma_1(k)\Theta^*} E_{1,k}^* = \frac{\alpha_1\alpha_2\alpha_5}{\alpha_{6,k}\omega_3\Theta^*} I_k^*$. Two last equations of the system (9) imply that

$$\begin{aligned} Q_k^* &= \frac{\alpha_2 \alpha_5 \sigma_1(k) \omega_1 + \alpha_5 \alpha_{6,k} \omega_2 + c \alpha_3 \alpha_{6,k} \omega_3}{\alpha_3 \alpha_{6,k} \omega_3} I_k^* = \frac{\alpha_{7,k}}{\alpha_3 \alpha_{6,k} \omega_3} I_k^*, \\ R_k^* &= \frac{r_1 \alpha_3 \alpha_{6,k} \omega_3 + r_2 \alpha_{7,k}}{\alpha_3 \alpha_4 \alpha_{6,k} \omega_3} I_k^*. \end{aligned}$$

Finally, by substituting all terms S_k^* , $E_{1,k}^*$, $E_{2,k}^*$, I_k^* , Q_k^* and R_k^* into the first equation, we can solve the value of I_k^* as follows:

$$I_k^* = \frac{b_k \alpha_3 \alpha_4 \alpha_{6,k} \omega_3 \Theta^*}{\left[\alpha_3 \alpha_4 \alpha_{6,k} \omega_3 + \alpha_3 \alpha_4 \alpha_5 \alpha_{6,k} + \alpha_4 \alpha_{7,k} + \alpha_2 \alpha_3 \alpha_4 \alpha_5 \sigma_1(k) + r_1 \alpha_3 \alpha_{6,k} \omega_3 + r_2 \alpha_{7,k}\right] \Theta^* + \alpha_1 \alpha_2 \alpha_3 \alpha_4 \alpha_5}.$$
 (10)

For simplicity, we use the notation Θ instead of Θ^* and denote

$$\begin{split} A_k &= M_q \nu(k) \mathbb{P}(k) \, \alpha_3 \alpha_4 \alpha_{6,k} \omega_3 > 0, \\ \tilde{A}_{1,k} &= \alpha_3 \alpha_4 \alpha_{6,k} \omega_3 + \alpha_3 \alpha_4 \alpha_5 \alpha_{6,k} + \alpha_4 \alpha_{7,k} + \alpha_2 \alpha_3 \alpha_4 \alpha_5 \sigma_1(k) + r_1 \alpha_3 \alpha_{6,k} \omega_3 + r_2 \alpha_{7,k} > 0, \\ \tilde{A}_2 &= \alpha_1 \alpha_2 \alpha_3 \alpha_4 \alpha_5 > 0. \end{split}$$

Then, by using (10), we have a self-consistency equation $\Theta = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_k \Theta}{\tilde{A}_{1,k} \Theta + \tilde{A}_2}$, or equivalently,

$$\Theta - \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_k \Theta}{\tilde{A}_{1,k} \Theta + \tilde{A}_2} = 0.$$
(11)

It is clear that the function $\Theta \equiv 0$ is always a solution of the self-consistency Eq. (11). Now, our aim is to determine the condition under which the self-consistency Eq. (11) has a unique nontrivial solution Θ^* . For this aim, we define

$$F(\Theta) = 1 - \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_k}{\tilde{A}_{1,k}\Theta + \tilde{A}_2}, \qquad \Theta \in [0, 1].$$

(9)

Here, it should be noted that $\frac{A_k}{\tilde{A}_{1,k}} < \frac{M_q v(k) \mathbb{P}(k) \alpha_3 \alpha_4 \alpha_{6,k} \omega_3}{\alpha_3 \alpha_4 \alpha_{6,k} \omega_3} = M_q v(k) \mathbb{P}(k)$. Thus, we can see that $F(\Theta)$ is continuous on [0, 1] and

$$\mathsf{F}(1) = 1 - \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_k}{\tilde{A}_{1,k} + \tilde{A}_2} > 1 - \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_k}{\tilde{A}_{1,k}} > 1 - \frac{M_q}{\sum_{k=1}^{n} k \mathbb{P}(k)} \sum_{k=1}^{n} \nu(k) \mathbb{P}(k) > 0.$$

Moreover, for each $\Theta \in [0, 1]$, we have $\frac{dF(\Theta)}{d\Theta} = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_k \tilde{A}_{1,k}}{(\tilde{A}_{1,k}\Theta + \tilde{A}_2)^2} > 0$, which implies that the function $F(\Theta)$ is increasing on [0, 1]. Therefore, it follows from the Intermediate Value theorem that the equation $F(\Theta) = 0$ has a unique positive solution $\Theta^* \in (0, 1)$ if F(0) < 0. Hence,

$$\frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_{k}}{\tilde{A}_{2}} = \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\left[\eta \sigma_{1}(k) + \alpha_{1} \sigma_{2}(k)\right] \omega_{3}}{\alpha_{1} \alpha_{2} \alpha_{5}} \nu(k) \mathbb{P}(k) > 1,$$

which indicates that $\Re_0 > 1$. Finally, the positive solution Θ^* of the self-consistency Eq. (11) uniquely solves the epidemic equilibrium point **E**_{*} given by (8). \Box

5.3. The stability analysis of the proposed fractional epidemic model

In this section, we discuss the qualitative analysis of the proposed fractional epidemic model. Firstly, we consider the relation between the basic reproductive ratio \Re_0 and the locally asymptotic stability of the MFE point \mathbf{E}_0 :

Theorem 5.4. Consider the malware-free equilibrium point E_0 .

(i) If $\Re_0 > 1$ then the equilibrium \mathbf{E}_0 is unstable.

(ii) If $\Re_0 = 1$ then the equilibrium \mathbf{E}_0 is not locally asymptotically stable.

(iii) If $\Re_0 < 1$ and $\alpha_1 \alpha_2 + \alpha_1 \alpha_5 + \alpha_2 \alpha_5 \leq \frac{M_q \langle \sigma_2 \nu \rangle}{\langle k \rangle}$ then the equilibrium \mathbf{E}_0 is unstable.

(iv) If $\mathscr{R}_0 < 1$ and $\alpha_1 \alpha_2 + \alpha_1 \alpha_5 + \alpha_2 \alpha_5 > \frac{M_q \langle \sigma_2 \nu \rangle}{\langle k \rangle}$ then the equilibrium \mathbf{E}_0 is locally asymptotically stable.

Proof. In order to investigate the local asymptotic stability of the malware-free equilibrium \mathbf{E}_0 , we will apply the linearization method for the fractional SE₁E₂IQR epidemic model around \mathbf{E}_0 . For this aim, let us consider Jacobi matrix $D\mathscr{F}(\mathbf{E}_0)$ at the point \mathbf{E}_0 subjecting to the epidemic model (1) in following compact form

$$D\mathscr{F}(\mathbf{E}_{0}) = \begin{bmatrix} M_{11} & M_{12} & \cdots & M_{1n} \\ M_{21} & M_{22} & \cdots & M_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ M_{n1} & M_{n2} & \cdots & M_{nn} \end{bmatrix}_{6n \times 6n}$$

where for each $i, j = \overline{1, n}$, the 6 × 6-square matrices M_{ii}, M_{ij} are given by

According to the stability theory of dynamical systems, in order to prove the local asymptotic stability of the malware-free equilibrium \mathbf{E}_0 , we need to show that all eigenvalues of the Jacobi matrix $D\mathscr{P}(\mathbf{E}_0)$ have negative real parts. For this aim,



Fig. 6. The variation table for the equation $P_3(\tilde{\lambda}) = 0$.

we firstly construct the characteristic polynomial $P_{6n}(\tilde{\lambda})$ w.r.t. the Jacobi matrix $D\mathscr{F}(\mathbf{E}_0)$. Indeed, by using mathematical induction principle, we immediately obtain

$$\mathbf{P}_{6n}(\tilde{\lambda}) = (\tilde{\lambda} + \alpha_4)^n (\tilde{\lambda} + \alpha_3)^n (\tilde{\lambda} + \mu)^n (\tilde{\lambda} + \alpha_1)^{n-1} (\tilde{\lambda} + \alpha_2)^{n-1} (\tilde{\lambda} + \alpha_5)^{n-1} \mathbf{P}_3(\tilde{\lambda}),$$

in which the third-order polynomial $P_3(\tilde{\lambda})$ is given by

$$\begin{split} \mathbf{P}_{3}(\tilde{\lambda}) &= (\tilde{\lambda} + \alpha_{1})(\tilde{\lambda} + \alpha_{2})(\tilde{\lambda} + \alpha_{5}) - \frac{M_{q}\langle\sigma_{2}\nu\rangle}{\langle k\rangle}\tilde{\lambda} - \alpha_{1}\alpha_{2}\alpha_{5}\frac{\omega_{3}M_{q}\langle\alpha_{6}\nu\rangle}{\alpha_{1}\alpha_{2}\alpha_{5}\langle k\rangle} \\ &= \tilde{\lambda}^{3} + (\alpha_{1} + \alpha_{2} + \alpha_{5})\tilde{\lambda}^{2} + \left(\alpha_{1}\alpha_{2} + \alpha_{1}\alpha_{5} + \alpha_{2}\alpha_{5} - \frac{M_{q}\langle\sigma_{2}\nu\rangle}{\langle k\rangle}\right)\tilde{\lambda} + \alpha_{1}\alpha_{2}\alpha_{5}(1 - \mathscr{R}_{0}). \end{split}$$

It can be easily verified that the solution set of the characteristic equation $P_{6n}(\tilde{\lambda}) = 0$ consists of

- The negative solutions λ̃ = -α₄, λ̃ = -α₃, λ̃ = -μ with multiplicity *n*.
 The negative solutions λ̃ = -α₁, λ̃ = -α₂, λ̃ = -α₅ with multiplicity *n* − 1.

Therefore, we can conclude that the stability of malware-free equilibrium \mathbf{E}_0 completely depends on the sign of solution of the equation $P_3(\tilde{\lambda}) = 0$. For simplicity, let us denote

$$a_0 = \alpha_1 \alpha_2 \alpha_5 (1 - \mathscr{R}_0), \qquad a_1 = \alpha_1 \alpha_2 + \alpha_1 \alpha_5 + \alpha_2 \alpha_5 - \frac{M_q \langle \sigma_2 \nu \rangle}{\langle k \rangle}, \qquad a_2 = \alpha_1 + \alpha_2 + \alpha_5 > 0.$$

Now, our proof can be proceeded into the following cases:

Case 1: If $\mathscr{R}_0 > 1$ then it follows that the coefficient $a_0 < 0$ and hence, solutions of the equation $P_3(\tilde{\lambda}) = 0$ belongs to one of following possibilities: 3 positive roots, 1 positive root and 2 complex conjugate roots or 2 negative roots and one positive root. Thus, since the Jacobi matrix $D\mathscr{F}(\mathbf{E}_0)$ always admits at least one positive eigenvalue, the malware-free equilibrium \mathbf{E}_0 is unstable.

Case 2: If $\mathscr{R}_0 = 1$ then the equation $P_3(\lambda) = 0$ has at least one eigenvalue with zero real part and hence, the equilibrium \mathbf{E}_0 is not locally asymptotically stable.

Case 3: If $\mathscr{R}_0 < 1$ then it follows that the coefficient $a_0 > 0$ and hence, solutions of the equation $P_3(\tilde{\lambda}) = 0$ belongs to one of following possibilities: 3 negative roots, 1 negative root and 2 complex conjugate roots or 2 positive roots and 1 negative root. It should be noted that the equation $P'_3(\tilde{\lambda}) = 3\tilde{\lambda}^2 + 2a_2\tilde{\lambda} + a_1 = 0$ has the positive discriminant. Indeed, we have

$$\Delta = (\alpha_1 + \alpha_2 + \alpha_5)^2 - 3\left(\alpha_1\alpha_2 + \alpha_1\alpha_5 + \alpha_2\alpha_5 - \frac{M_q\langle\sigma_2\nu\rangle}{\langle k\rangle}\right)$$
$$= \frac{(\alpha_1 - \alpha_2)^2 + (\alpha_1 - \alpha_5)^2 + (\alpha_2 - \alpha_5)^2}{2} + \frac{3M_q\langle\sigma_2\nu\rangle}{\langle k\rangle} > 0.$$

Sub-case 3.1: If $a_1 = 0$ then the equation $P_3(\tilde{\lambda}) = 0$ has a zero solution $\tilde{\lambda} = 0$ with multiplicity 2, which follows that the malware-free equilibrium E_0 is unstable.

Sub-case 3.2: If $a_1 < 0$ then we directly get that the equation $P'_4(\tilde{\lambda}) = 3\tilde{\lambda}^2 + 2a_2\tilde{\lambda} + a_1 = 0$ has two distinct solutions, say $\tilde{\lambda}_1 < 0$ and $\tilde{\lambda}_2 > 0$. Next, we have the following variation table (see Fig. 6).

Since the fact that $P_3(0) = a_0 > 0$, Fig. 6 indicates that the equation $P_3(\tilde{\lambda}) = 0$ always have at least two roots with positive real part if $\Re_0 < 1$ and $a_1 < 0$. This follows that the malware-free equilibrium **E**₀ is unstable.

Sub-case 3.3: If $a_1 > 0$ then we directly get that the equation $P'_3(\tilde{\lambda}) = 3\tilde{\lambda}^2 + 2a_2\tilde{\lambda} + a_1 = 0$ has two distinct negative solutions, say $\tilde{\lambda}_1$ and $\tilde{\lambda}_2$. Next, we have the following variation table (see Fig. 7)



Fig. 7. The variation table for the equation $P_3(\tilde{\lambda}) = 0$.

Due to the fact that $P_3(0) = a_0 > 0$, Fig. 7 indicates that the equation $P_3(\tilde{\lambda}) = 0$ cannot have solution with non-negative real part if $\Re_0 < 1$. Therefore, we can conclude that if $\Re_0 < 1$ and $\alpha_1 \alpha_2 + \alpha_1 \alpha_5 + \alpha_2 \alpha_5 > \frac{M_q(\sigma_2 \nu)}{\langle k \rangle}$, all eigenvalues of Jacobi matrix $D\mathscr{F}(\mathbf{E}_0)$ have negative real parts, which means that the malware-free equilibrium \mathbf{E}_0 is locally asymptotically stable. \Box

Remark 5.2. As a consequence of Theorem 5.4, the condition $\Re_0 < 1$ is not sufficient enough for the local asymptotic stability of the malware-free equilibrium \mathbf{E}_0 .

In order to prove the next asymptotic stability results, we refer from Lemma 3.1 of [14] a necessary estimation related to fractional derivative:

Lemma 5.1 ([14]). Assume that $\mathbf{x} \in AC([0, \infty), \mathbb{R}^+)$ and $\alpha \in (0, 1]$. Then, for each $t \ge 0$ and $\mathbf{x}^* \in \mathbb{R}^+$, we have

$$\int_{0}^{C} \mathfrak{D}_{t}^{\beta} \left(\mathbf{x}(t) - \mathbf{x}^{*} - \mathbf{x}^{*} \ln(\frac{\mathbf{x}(t)}{\mathbf{x}^{*}}) \right) \leq \left(1 - \frac{\mathbf{x}^{*}}{\mathbf{x}(t)} \right)_{0}^{C} \mathfrak{D}_{t}^{\beta} \mathbf{x}(t)$$

Remark 5.3. Consider a real-valued function $\Psi : \mathbb{R}_+ \to \mathbb{R}_+$ defined by

$$\Psi(z) = z - z^* - z^* \ln\left(\frac{z}{z^*}\right), \qquad z, z^* \in \mathbb{R}_+.$$

Note that for all $z \ge 0$, the function $\Psi(z)$ is non-negative and attains its global minimum at $z = z^*$.

Next, we consider the globally asymptotic behavior of the MFE point E_0 , which is an important results in the theory of epidemiology.

Theorem 5.5. Denote

$$\tilde{\mathscr{R}}_{0} = \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \nu(k) \mathbb{P}(k) \frac{\omega_{3} \left(\sigma_{1}(k) + \sigma_{2}(k)\right)}{\alpha_{2} \alpha_{5}}$$

If $\tilde{\mathscr{R}}_0 < 1$ then the malware-free equilibrium point \mathbf{E}_0 of the fractional SE₁E₂IQR epidemic model (1) is globally asymptotically stable.

Proof. Let $\mathbf{x}(t) = \{(E_{1,k}(t), E_{2,k}(t), I_k(t), S_k(t), Q_k(t), R_k(t))\}_{k=1}^n$ be a non-negative solution of the fractional epidemic model (1). Now, we construct a functional $\mathbf{V}: \Sigma^+ \to \mathbb{R}$ as follows:

$$\mathbf{V}(\mathbf{x}(t)) = \frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\nu(k)}{b_k} \mathbb{P}(k) \left\{ \left[S_k(t) - b_k - b_k \ln\left(\frac{S_k(t)}{b_k}\right) \right] + E_{1,k}(t) + E_{2,k}(t) + I_k(t) \right\}.$$

Here, the above function $\mathbf{V}(\mathbf{x}(t))$ is non-negative definite with respect to the MFE state \mathbf{E}_0 . This function is called the Lyapunov function defined along the non-negative solution $\mathbf{x}(t)$ of the fractional system (1). Next, by taking Caputo fractional derivative in time of the function $\mathbf{V}(\mathbf{x}(t))$ along the solution $\mathbf{x}(t)$ and using Lemma 5.1, we immediately obtain

$$\begin{split} {}^{C}_{0}\mathfrak{D}^{\beta}_{t}\mathbf{V}(\mathbf{x}(t)) &= \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\nu(k)}{b_{k}} \mathbb{P}(k)^{C}_{0}\mathfrak{D}^{\beta}_{t} \left(S_{k}(t) - b_{k} - b_{k} \ln\left(\frac{S_{k}(t)}{b_{k}}\right) \right) \\ &+ \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\nu(k)}{b_{k}} \mathbb{P}(k) \left({}^{C}_{0}\mathfrak{D}^{\beta}_{t}E_{1,k}(t) + {}^{C}_{0}\mathfrak{D}^{\beta}_{t}E_{2,k}(t) + {}^{C}_{0}\mathfrak{D}^{\beta}_{t}I_{k}(t) \right) \\ &\leq \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\nu(k)}{b_{k}} \mathbb{P}(k) \left[\left(1 - \frac{b_{k}}{S_{k}(t)} \right)^{C}_{0}\mathfrak{D}^{\beta}_{t}S_{k}(t) + {}^{C}_{0}\mathfrak{D}^{\beta}_{t} \left(E_{1,k}(t) + E_{2,k}(t) + I_{k}(t) \right) \right] \end{split}$$

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where

$$\left(1 - \frac{b_k}{S_k(t)}\right)_0^c \mathfrak{D}_t^\beta S_k(t) = \left(1 - \frac{b_k}{S_k(t)}\right) [\Lambda(k) - (\sigma_1(k) + \sigma_2(k)) S_k(t)\Theta(t) - \mu S_k(t) + \theta R_k(t)]$$

$$= \Lambda(k) - (\sigma_1(k) + \sigma_2(k)) S_k(t)\Theta(t) - \mu S_k(t) + \theta R_k(t)$$

$$- \frac{b_k \Lambda(k)}{S_k(t)} + b_k (\sigma_1(k) + \sigma_2(k)) \Theta(t) + \mu b_k - \frac{b_k \theta R_k(t)}{S_k(t)}$$
(12)

and

By combining the estimations (12) and (13), we receive

$$\begin{split} \left(1 - \frac{b_k}{S_k(t)}\right)_0^C \mathfrak{D}_t^\beta S_k(t) + {}_0^C \mathfrak{D}_t^\beta \left(E_{1,k}(t) + E_{2,k}(t) + I_k(t)\right) \\ & \leq \left(\Lambda(k) - \mu S_k(t) - \frac{b_k \Lambda(k)}{S_k(t)} + \mu b_k\right) + \theta R_k(t) - \frac{b_k \theta R_k(t)}{S_k(t)} \\ & + b_k \left(\sigma_1(k) + \sigma_2(k)\right) \Theta(t) - (\mu + c + r_1) I_k(t). \end{split}$$

For each $t \ge 0$ and $\mathbf{x}(t) \in \Sigma^+$, we have

$$\Lambda(k) - \mu S_k(t) - \frac{b_k \Lambda(k)}{S_k(t)} + \mu b_k = \left(1 - \frac{b_k}{S_k(t)}\right) \left(\Lambda(k) - \mu S_k(t)\right) \le 0$$

$$\theta R_k(t) - \frac{b_k \theta R_k(t)}{S_k(t)} = \theta R_k(t) \left(1 - \frac{b_k}{S_k(t)}\right) \le 0.$$

As a consequence, we directly get that

$$\begin{split} & \int_{0}^{C} \mathfrak{D}_{t}^{\beta} \mathbf{V}(\mathbf{x}(t)) \leq \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \frac{\nu(k)}{b_{k}} \mathbb{P}(k) \left[b_{k} \left(\sigma_{1}(k) + \sigma_{2}(k) \right) \Theta(t) - \alpha_{5} I_{k}(t) \right] \\ &= \frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \nu(k) \mathbb{P}(k) \left(\sigma_{1}(k) + \sigma_{2}(k) \right) \Theta(t) - \alpha_{5} \Theta(t) \\ &= \alpha_{5} \Theta(t) \left[\frac{M_{q}}{\langle k \rangle} \sum_{k=1}^{n} \nu(k) \mathbb{P}(k) \frac{\omega_{3} \left(\sigma_{1}(k) + \sigma_{2}(k) \right)}{\alpha_{2} \alpha_{5}} \frac{\alpha_{2}}{\omega_{3}} - 1 \right] \\ &= \alpha_{5} \Theta(t) \left(\frac{\alpha_{2}}{\omega_{3}} \tilde{\mathscr{R}}_{0} - 1 \right). \end{split}$$

Thus, it follows that ${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\mathbf{V}(\mathbf{x}(t)) \leq 0$ if $\frac{\alpha_{2}}{\omega_{3}}\tilde{\mathscr{R}}_{0} - 1 \leq 0$, or equivalently,

$$\tilde{\mathscr{R}}_0 \leq \frac{\omega_3}{\alpha_2} = \frac{\omega_3}{\mu + \omega_2 + \omega_3} < 1.$$

In addition, we can also verify that ${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\mathbf{V}(\mathbf{x}(t)) = 0$ if and only if

$$S_k(t) = b_k$$
 and $E_{1,k}(t) = E_{2,k}(t) = I_k(t) = 0$, $k = 1, 2, ..., n$.

Hence, the largest invariant set of $\left\{ \mathbf{x}(t) \in \Sigma^+ : {}_0^C \mathfrak{D}_t^\beta \mathbf{V}(\mathbf{x}(t)) = 0 \right\}$ is $\{\mathbf{E}_0\}$. Finally, by applying LaSalle's invariance principle (see [38]), we can conclude that the malware-free equilibrium \mathbf{E}_0 is globally asymptotically stable if the condition $\tilde{\mathscr{R}}_0 < 1$ holds. \Box

Remark 5.4. Due to the fact that $\alpha_1 = \eta + \mu + \omega_1 > \eta$, it implies that

$$\frac{\omega_3\left(\sigma_1(k)+\sigma_2(k)\right)}{\alpha_2\alpha_5} > \frac{\omega_3\left(\eta\sigma_1(k)+\alpha_1\sigma_2(k)\right)}{\alpha_1\alpha_2\alpha_5}.$$

As a consequence, we can see that $\tilde{\mathscr{R}}_0 > \mathscr{R}_0$. According to Theorem 5.5, the malware-free equilibrium point \mathbf{E}_0 is globally asymptotically stable only when $\tilde{\mathscr{R}}_0 > 1$. Hence, we can conclude that the condition $\mathscr{R}_0 < 1$ is not sufficient to eliminate the disease on the network.

5.4. The bifurcation analysis

In the previous section, we claimed that if the basic reproductive ratio $\Re_0 \neq 1$ then the equilibrium points are either asymptotically stable or unstable. In this section, we aim to discuss the bifurcation phenomena of the proposed model occurring when $\Re_0 = 1$ or in the other words, we find a necessary condition to determine the direction of bifurcation of epidemic equilibrium curve at $\Re_0 = 1$. The theorem is stated as follows:

Theorem 5.6. The fractional SE_1E_2IQR epidemic model (1) always has a forward bifurcation at $\Re_0 = 1$ for all values of parameters.

Proof. In order to draw the bifurcation curve, we consider the graph of infectious compartments locally as a function of \mathscr{R}_0 . Then, the bifurcation direction is determined by the sign of the slope at the point $(\mathscr{R}_0, \Theta) = (1, 0)$. In particular, we have that the epidemic equilibrium curve bifurcates forward (or backward) if the derivative at the critical point $(\mathscr{R}_0, \Theta) = (1, 0)$.

is positive (or negative), respectively. Now, in order to determine the sign of the function $\frac{\partial \Theta}{\partial \mathscr{R}_0}$ at (1, 0), we will find this partial derivative by implicitly differentiating from the self-consistency Eq. (11).

Indeed, since the fact that at the epidemic equilibrium point, the function $I_k(t)$ is positive for all k = 1, 2, ..., n and $t \ge 0$, which follows that the function $\Theta(t)$ is also positive for all $t \ge 0$. According to the self-consistency Eq. (11), we deduce that the solution $F(\Theta) = 0$ must have at least one solution Θ^* , which solves the epidemic equilibrium point of the proposed model. Hence, we have the following equation

$$\frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{A_k}{\tilde{A}_{1,k} \Theta + \tilde{A}_2} = 1,$$

where according to Theorem 5.3, the parameters A_k , $\tilde{A}_{1,k}$ and \tilde{A}_2 are given by

$$\begin{aligned} A_k &= M_q \nu(k) \mathbb{P}\left(k\right) \alpha_3 \alpha_4 \alpha_{6,k} \omega_3 \\ \tilde{A}_2 &= \alpha_1 \alpha_2 \alpha_3 \alpha_4 \alpha_5 \end{aligned}$$

and

$$\begin{aligned} \mathsf{A}_{1,k} &= \alpha_3 \alpha_4 \alpha_{6,k} \omega_3 + \alpha_3 \alpha_4 \alpha_5 \alpha_{6,k} + \alpha_4 \alpha_{7,k} + \alpha_2 \alpha_3 \alpha_4 \alpha_5 \sigma_1(k) + r_1 \alpha_3 \alpha_{6,k} \omega_3 + r_2 \alpha_{7,k} \\ &= \omega_3 \left(\alpha_3 \alpha_4 \alpha_{6,k} + r_1 \alpha_3 \alpha_{6,k} \right) + \left(\alpha_3 \alpha_4 \alpha_5 \alpha_{6,k} + \alpha_4 \alpha_{7,k} + \alpha_2 \alpha_3 \alpha_4 \alpha_5 \sigma_1(k) + r_2 \alpha_{7,k} \right) \\ &= \omega_3 \alpha_{8,k} + \alpha_{9,k}. \end{aligned}$$

Next, by multiplying the term $\frac{M_q \langle \alpha_6 v \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle}$ to both numerator and denominator of $\frac{A_k}{\bar{A}_{1,k} \Theta + \bar{A}_2}$, we directly get that

$$\frac{M_q}{\langle k \rangle} \sum_{k=1}^{n} \frac{\alpha_3 \alpha_4 \alpha_{6,k} \nu(k) \mathbb{P}(k) \mathscr{R}_0}{\alpha_{8,k} \mathscr{R}_0 \Theta + \frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \left[\alpha_{9,k} \Theta + \tilde{A}_2 \right]} = 1$$
(14)

Here, it should be noted that $\mathscr{R}_0 = \frac{\omega_3 M_q \langle \alpha_6 v \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle}$ and the above equation is considered as the epidemic equilibrium curve in the (\mathscr{R}_0, Θ) positive quadrant. Then, in order to obtain the necessary and sufficient condition for the direction of bifurcation of the epidemic equilibrium curve, we take implicit differentiation Eq. (14) with respect to \mathscr{R}_0 and give the sign of the partial derivative $\frac{\partial \Theta}{\partial \mathscr{R}_0}$ at the point $(\mathscr{R}_0, \Theta) = (1, 0)$. Indeed, we have

$$\frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\alpha_3 \alpha_4 \alpha_{6,k} \nu(k) \mathbb{P}(k) \left(P_{1,k} - P_{2,k} \right)}{\left[\alpha_{8,k} \mathscr{R}_0 \Theta + \frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \left(\alpha_{9,k} \Theta + \tilde{A}_2 \right) \right]^2} = 0, \tag{15}$$

where the terms $P_{1,k}$ and $P_{2,k}$ is defined as follows:

$$P_{1,k} = \alpha_{8,k} \mathscr{R}_0 \Theta + \frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \left(\alpha_{9,k} \Theta + \tilde{A}_2 \right)$$
$$P_{2,k} = \mathscr{R}_0 \left[\alpha_{8,k} \Theta + \alpha_{8,k} \mathscr{R}_0 \frac{\partial \Theta}{\partial \mathscr{R}_0} + \alpha_{9,k} \frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \frac{\partial \Theta}{\partial \mathscr{R}_0} \right]$$

In the following, for simplicity, we denote $\frac{\partial \Theta}{\partial \mathscr{R}_0}\Big|_{(\mathscr{R}_0, \Theta)=(1, 0)}$ by $\frac{\partial \Theta}{\partial \mathscr{R}_0}\Big|_{(1, 0)}$ Then, at $\mathscr{R}_0 = 1$ and $\Theta = 0$, Eq. (15) becomes

$$\frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\alpha_3 \alpha_4 \alpha_{6,k} \nu(k) \mathbb{P}(k) \left[\frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \tilde{A}_2 - \alpha_{8,k} \frac{\partial \Theta}{\partial \mathscr{R}_0} |_{(1,0)} - \alpha_{9,k} \frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \frac{\partial \Theta}{\partial \mathscr{R}_0} |_{(1,0)} \right]}{\left(\frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \tilde{A}_2 \right)^2} = 0,$$

in which

$$\begin{split} \frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\alpha_3 \alpha_4 \alpha_{6,k} \nu(k) \mathbb{P}(k) \frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \tilde{A}_2}{\left(\frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \tilde{A}_2\right)^2} &= 1, \\ \frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\alpha_3 \alpha_4 \alpha_{6,k} \nu(k) \mathbb{P}(k) \alpha_{8,k} \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)}}{\left(\frac{M_q \langle \alpha_6 \nu \rangle}{\alpha_1 \alpha_2 \alpha_5 \langle k \rangle} \tilde{A}_2\right)^2} &= \sum_{k=1}^n \frac{\alpha_{6,k} \alpha_{8,k} \nu(k) \mathbb{P}(k) \langle k \rangle \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)}}{\alpha_3 \alpha_4 M_q \langle \alpha_6 \nu \rangle^2} \\ &= \frac{(\alpha_4 + r_1) \langle k \rangle}{\alpha_4 M_q \langle \alpha_6 \nu \rangle} \left(\sum_{k=1}^n \alpha_{6,k}^2 \nu(k) \mathbb{P}(k)\right) \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)} \\ &= \frac{(\alpha_4 + r_1) \langle k \rangle \langle \alpha_6^2 \nu \rangle}{\alpha_4 M_q \langle \alpha_6 \nu \rangle} \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)}. \end{split}$$

In addition, we recall that $\alpha_{9,k} = \alpha_3 \alpha_4 \alpha_5 (\alpha_2 \sigma_1(k) + \alpha_{6,k}) + (r_2 + \alpha_4) \alpha_{7,k}$ and denote

$$RHS = \frac{M_q}{\langle k \rangle} \sum_{k=1}^n \frac{\alpha_3 \alpha_4 \alpha_{6,k} \nu(k) \mathbb{P}(k) \alpha_{9,k} \frac{M_q(\alpha_6 \nu)}{\alpha_1 \alpha_2 \alpha_5(k)} \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)}}{\left(\frac{M_q(\alpha_6 \nu)}{\alpha_1 \alpha_2 \alpha_5(k)} \tilde{A}_2\right)^2}$$

$$= \sum_{k=1}^n \frac{\alpha_{6,k} \nu(k) \mathbb{P}(k) \sigma_1(k)}{\alpha_1 \langle \alpha_6 \nu \rangle} \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)} + \omega_1 (r_2 + \alpha_4) \sum_{k=1}^n \frac{\alpha_{6,k} \nu(k) \mathbb{P}(k) \sigma_1(k)}{\alpha_1 \alpha_3 \alpha_4 \langle \alpha_6 \nu \rangle} \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)}$$

$$+ \sum_{k=1}^n \frac{\alpha_{6,k}^2 \nu(k) \mathbb{P}(k)}{\alpha_1 \alpha_2 \langle \alpha_6 \nu \rangle} \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)} + \frac{(r_2 + \alpha_4)(\alpha_5 \omega_2 + c\alpha_3 \omega_3)}{\alpha_1 \alpha_2 \alpha_3 \alpha_4 \alpha_5} \sum_{k=1}^n \frac{\alpha_{6,k}^2 \nu(k) \mathbb{P}(k)}{\langle \alpha_6 \nu \rangle} \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)}$$

$$= \frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)} \left\{ \frac{\langle \alpha_6 \nu \sigma_1 \rangle}{\langle \alpha_6 \nu \rangle} \left[\frac{1}{\alpha_1} + \frac{\omega_1 (r_2 + \alpha_4)}{\alpha_1 \alpha_3 \alpha_4} \right] + \frac{\langle \alpha_6^2 \nu \rangle}{\langle \alpha_6 \nu \rangle} \left[\frac{1}{\alpha_1 \alpha_2} + \frac{(r_2 + \alpha_4)(\alpha_5 \omega_2 + c\alpha_3 \omega_3)}{\alpha_1 \alpha_2 \alpha_3 \alpha_4 \alpha_5} \right] \right\}.$$

Therefore, we immediately obtain

$$\frac{\partial \Theta}{\partial \mathscr{R}_{0}}|_{(1,0)}\left\{\frac{\langle \alpha_{6}^{2}\nu\rangle}{\langle \alpha_{6}\nu\rangle}\left[\frac{1}{\alpha_{1}\alpha_{2}}+\frac{(r_{2}+\alpha_{4})(\alpha_{5}\omega_{2}+c\alpha_{3}\omega_{3})}{\alpha_{1}\alpha_{2}\alpha_{3}\alpha_{4}\alpha_{5}}+\frac{(\alpha_{4}+r_{1})\langle k\rangle}{\alpha_{4}M_{q}}\right]+\frac{\langle \alpha_{6}\nu\sigma_{1}\rangle}{\langle \alpha_{6}\nu\rangle}\left[\frac{1}{\alpha_{1}}+\frac{\omega_{1}(r_{2}+\alpha_{4})}{\alpha_{1}\alpha_{3}\alpha_{4}}\right]\right\}=1.$$

Since the model's parameters are all positive, we can conclude that $\frac{\partial \Theta}{\partial \mathscr{R}_0}|_{(1,0)}$ is always positive for all values of parameters. Hence, the fractional SE₁E₂IQR epidemic model (1) always exhibits a forward bifurcation at $\mathscr{R}_0 = 1$. \Box

6. Simulation

This section perform some numerical simulations in terms of network structure and model's parameters to explain our theoretical results. Since Wireless Sensor Network is an Energy-Aware Barabási Albert scale-free network, two adjustable parameter λ_d and λ_e are assumed to be positive in order to demonstrate the importance of both node's degree and residual energy of node in the network. Moreover, we also assume that the connectivity among nodes dominates the network structure, i.e., $\lambda_d > \lambda_e$. Thus, we choose $\lambda_d = \frac{4}{5}$, $\lambda_e = \frac{1}{5}$. In addition, according to the formula (11) of [3], the degree distribution is $\mathbb{P}(k) = \frac{C}{(79.94k + 120)^{3.5}}$ and *C* is a constant such that $\sum_{k=1}^{n} \mathbb{P}(k) = 1$. In this section, we choose the number of groups n = 10, the infected rates $\sigma_1(k) = \sigma_1 k$, $\sigma_2(k) = \sigma_2 k$ and the infectivity $\nu(k) = k^m$ with $\sigma_1 = 0.35$, $\sigma_2 = 0.25$, m = 0.25. In the following, by using the series computing tool symsum in MatLab, we immediately get

In addition, we assume that the other parameters are chosen as follows: $\Lambda(k) = 4$, $\omega_1 = 0.1$, $\omega_2 = 0.12$, $\omega_3 = 0.2$, c = 0.17, $\mu = 0.04$, $r_1 = 0.05$, $\theta = 0.03$, $r_2 = 0.06$ and $\eta = 0.15$. Therefore, the basic reproductive ratio \Re_0 is given by

$$\mathscr{R}_0 = rac{\omega_3 M_q \langle lpha_6 \nu
angle}{lpha_1 lpha_2 lpha_5 \langle k
angle} pprox 1.1467 M_q.$$

Hence, we can conclude that if the fuzzy-rule based interaction constant M_q is large enough then the basic reproduction ratio \mathscr{R}_0 may exceed to 1, that proves the important role of the regulation of data transmitting process in the network when the network is infected.



Fig. 8. The sensitivity test results for significance of parameters.

6.1. The analysis sensitivity of the basic reproductive ratio \mathscr{R}_0

In an epidemic model, the sensitivity analysis studies how different uncertainty sources contribute to the model's overall uncertainty and the sensitivity indices then allow us to estimate the relative change of the basic reproductive ratio \mathscr{R}_0 when a parameter changes. We recall from [39] the following definition

Definition 6.1 (*[39]*). The normalized forward sensitivity index of a variable *u* that depends differentiably on a parameter *p* is defined by

$$\Upsilon_p^u = \frac{\partial u}{\partial p} \times \frac{p}{u}.$$

As a consequence, we now calculate the normalized sensitivity indices of the basic reproductive ratio \mathscr{R}_0 that measure the relative change of the ratio \mathscr{R}_0 w.r.t. the relative changes of other parameters. For this aim, we rewrite the basic reproductive ratio \mathscr{R}_0 as follows:

$$\mathscr{R}_{0} = \frac{\eta \omega_{3} M_{q} \langle \sigma_{1} \nu \rangle}{\alpha_{1} \alpha_{2} \alpha_{5} \langle k \rangle} + \frac{\omega_{3} M_{q} \langle \sigma_{2} \nu \rangle}{\alpha_{2} \alpha_{5} \langle k \rangle}$$

where $\langle \sigma_1 \nu \rangle = \sum_{k=1} \sigma_1(k) \nu(k) \mathbb{P}(k)$ and $\langle \sigma_2 \nu \rangle = \sum_{k=1} \sigma_2(k) \nu(k) \mathbb{P}(k)$. Then, we have

$$\begin{split} \Upsilon_{M_{q}}^{\mathscr{R}_{0}} &= 1, \qquad \Upsilon_{\langle k \rangle}^{\mathscr{R}_{0}} = -1, \qquad \Upsilon_{c}^{\mathscr{R}_{0}} = -\frac{c}{c+\mu+r_{1}}, \qquad \Upsilon_{\omega_{2}}^{\mathscr{R}_{0}} = -\frac{\omega_{2}}{\omega_{2}+\omega_{3}+\mu} \\ \Upsilon_{\omega_{1}}^{\mathscr{R}_{0}} &= -\frac{\omega_{1}\eta\langle\sigma_{1}\nu\rangle}{\alpha_{1}(\eta\langle\sigma_{1}\nu\rangle+\alpha_{1}\langle\sigma_{2}\nu\rangle)}, \qquad \Upsilon_{\omega_{3}}^{\mathscr{R}_{0}} = \frac{\omega_{2}+\mu}{\omega_{3}+\omega_{2}+\mu}, \qquad \Upsilon_{\eta}^{\mathscr{R}_{0}} = \frac{(\omega_{1}+\mu)\eta\langle\sigma_{1}\nu\rangle}{\alpha_{1}(\eta\langle\sigma_{1}\nu\rangle+\alpha_{1}\langle\sigma_{2}\nu\rangle)} \end{split}$$

Hence, the sensitivity test results of the proposed model's parameters can be summarized in the following table (Table 3) and Fig. 8, in which Table 3 shows the sensitivity indices of parameters based on the value of parameters proposed at the beginning of this section and Fig. 8, construct by using bar toolbox in Matlab, presents the graphical representations for Table 3.

The above table indicates that the basic reproductive ratio \mathcal{R}_0 is the least sensitive to the quarantine rate ω_1 , followed by the transition rate η . The fuzzy rule-based interaction constant M_q and the average degree $\langle k \rangle$ have the same sensitivity index. It can be also observed that the threshold \mathcal{R}_0 is more sensitive to the quarantine rate c than the one of ω_2 . The sensitivity of \mathcal{R}_0 to the infectious rate ω_3 is nearly $\frac{4}{9}$. By the data in Table 4 and Fig. 8, we can claim that if the rate of quarantine rate c is to increase by 10% then the value of \mathcal{R}_0 will decrease by 6.538%. Likewise, a 10% increase of the average degree will correspond to a 10% decrease of the threshold \mathcal{R}_0 while only a decrease of 1.448% of the quarantine rate ω_1 can increase the value of \mathcal{R}_0 by 10%. In order to decrease the value of \mathcal{R}_0 by 10%, the increases of 2.0276%, 4.44% and 10% corresponding to the parameters η , ω_3 and M_q are required.

On the other hand, Theorem 5.4 shows that the condition $\Re_0 < 1$ ensures the locally asymptotic stability of the MFE point **E**₀. In the following, by treating the basic reproductive ratio \Re_0 as a function of variables c, ω_1 and ω_2 , we give the effect of quarantine constants ω_1 , ω_2 , c in the change of \Re_0 with given other parameters and $M_q = 0.5$. The figures are plotted by using Matlab toolbox plot.

By using Matlab toolbox plot3d, we give Fig. 9 to represent the three-dimensional phrase portraits of the basic reproductive ratio \mathscr{R}_0 with effect of each pair of quarantine parameters. According to Fig. 9, we obtain the different changes of the basic reproductive ratio \mathscr{R}_0 with respect to quarantine parameters c, ω_1 and ω_2 . It experienced a dramatic decline in the change of \mathscr{R}_0 w.r.t. the parameter c, while the threshold \mathscr{R}_0 remains stable when the parameter ω_1 varies from 0 to 1. The basic reproductive ratio \mathscr{R}_0 markedly reduces when the parameter ω_2 tends to 1. Especially, it can be seen that the



Fig. 9. The effect of each quarantine parameter in the basic reproductive ratio \mathcal{R}_0 .



Fig. 10. The three-dimensional phrase portraits of the basic reproductive ratio \mathcal{R}_0 with effect of each pair of quarantine parameters.

isolation of exposed nodes is not enough for eliminating the disease in the network. Indeed, if we choose the parameter c = 0 and fix these other parameters then the basic reproductive ratio \mathscr{R}_0 is approximately 1.7, which means that the disease exists on the network. Thus, we derive the importance of the isolation of infectious sources from the network. In addition, the three-dimensional phrase portraits of the threshold \mathscr{R}_0 with quarantine parameters are also given in Fig. 10. From the below phrase portraits, we can conclude that the bigger the parameters are, the smaller the difference $\mathscr{R}_0 - 1$ is.

6.2. The dynamical behavior of the fractional SE_1E_2IQR epidemic model

In the following, based on the modified Adams–Bashforth–Moulton predictor–corrector method and Matlab program, we will present the graphical representations and show the effectiveness of the proposed epidemic model. In Fig. 11, we choose the fuzzy rule-based interaction parameter M_q such that $\Re_0 < 1$. It is shown that the malware-free equilibrium point \mathbf{E}_0 is globally asymptotically stable when $\Re_0 < 1$. Indeed, even for a high rate of infectious nodes at the beginning, the density of infectious nodes is dramatically decreasing while the density of susceptible nodes is sharply increasing after hitting the lowest point at about t = 5. The changes of other states, in general, are similar. In the beginning, they steadily go up and reach the peak. After that, all these states experience a slight decline and remain at the same level as times increases. In addition, Fig. 11 points out that the smaller the constant M_q gets, the higher density the susceptible nodes have, which means that the temporary reduction of connection when the network is attacked is one of the solutions to prevent the network from the widespread of malware programs. Fig. 12 considers the case $\Re_0 > 1$. In this case, we can see that despite a high rate of the susceptible population at the beginning, the density of susceptible nodes experiences a dramatic drop and reaches the lowest point at t = 20. After that, this state stays unchanged during the time. In addition, Fig. 12 shows that the epidemic disease is permanent on the network that allows the results of Theorem 5.4.

Furthermore, in order to evaluate the influences of node's degree and node's state change in stifling the infection, we give the changes of susceptible state and infectious state of the fractional SE₁E₂IQR epidemic model overtime at some different values of M_q , in which the dotted line represents for the result without using fuzzy logic and the solid lines represent for the results with some different M_q . According to Fig. 13, we can conclude that the bigger M_q gets, the higher density of infectious nodes and the lower density of susceptible nodes. Especially, the network-based epidemic model without using fuzzy logic experienced the highest density of infected nodes and the least density of susceptible nodes.



Fig. 11. The time series of S(t), $E_1(t)$, $E_2(t)$, I(t), Q(t) and R(t) with $\Re_0 < 1$.



Fig. 12. The time series of S(t), $E_1(t)$, $E_2(t)$, I(t), Q(t) and R(t) with $\Re_0 > 1$.



Fig. 13. The influence of fuzzy rule-based transmission parameter M_q to the densities of susceptible and infectious states.

compared with the one with fuzzy logic. Hence, we can conclude that the use of fuzzy logic can better describe the disease transmission on complex networks.



Fig. 14. The comparison of the node's densities in various states of some epidemic models.

In Fig. 14, we discuss the dynamic behavior of the proposed fractional SE_1E_2IQR epidemic model compared with three fractional epidemic models: SIR, SEIR, and SIQR epidemic models. We can see that in all given epidemic models, the densities of infectious nodes are decreasing, in which the infectious nodes in the SE_1E_2IQR model experience the most rapid decline compared with three other epidemic models. In the SIR epidemic model, the decrease of infectious nodes is lower than in other models due to the absence of quarantine treatments for infected individuals. Conversely, a noticeable reduction in the densities of infectious nodes can be seen in SIQR and SE_1E_2IQR epidemic models due to the use of quarantine compartment. Especially, in the SE_1E_2IQR epidemic model, the total population of infected nodes is classified into three classes: exposed state of type 1, exposed state of type 2 and infectious state. Next, by using appropriate treatments, we isolate sensor nodes containing malware from the network at rates ω_1 , ω_2 and c, respectively. Here, since not only infectious nodes in the network and that is also the reason why the density of infectious nodes in the SE_1E_2IQR model is lower than one in the other models. In conclusion, the biggest difference between the plot of SE_1E_2IQR epidemic model and the plots of other epidemic models is that the quarantine will increase the number of susceptible nodes and decrease the number of infectious nodes and infected but not infectious nodes at the end, which are consistent with reality.

Figs. 15 and 16 depict the dynamic for some cases of fractional order β of the fractional SE₁E₂IQR epidemic model (1) for $\Re_0 < 1$ and \Re_0 , respectively. We have observed that if we increase the value of parameter β , the type 1-exposed $E_1(t)$, the type 2-exposed $E_2(t)$ and infectious individuals I(t) goes down slightly. It should be noted that the increasing of fractional-order β will reduce the spread of malware in populations.

Figs. 17–19 show the effect of quarantine parameters c, ω_1 and ω_2 to the dynamics of fractional SE₁E₂IQR epidemic model. It is well-known that when the network is highly attacked by different types of malware programs, quarantine is one of the effective treatments besides immunization. Here, we can see that the isolation of infected states plays an essential role in stifling the number of infectious nodes and rising the numbers of susceptible and recovered nodes. Indeed, from the three below figures, as the quarantine rates are decreasing, the densities of susceptible nodes are moderately increasing while the density of infectious nodes experienced a noticeable decline. The obtained simulation results agree with real-world situations.

7. Conclusions

In order to illustrate the influence of heterogeneity in contact patterns and predict the effectiveness of quarantine strategy for preventing the malware spreading, we propose and analyze a network-based fractional SE_2E_2IQR epidemic model with a fuzzy infection on wireless sensor network, that is known as an EABA scale-free network. By using the next-generation method, we obtain an important threshold in epidemic control, namely the basic reproductive ratio \mathcal{R}_0 . After that, the epidemic dynamics of the proposed model are studied mathematically. Indeed, by the mean of Lyapunov's



Fig. 15. The effects of fractional-order derivative to the epidemic model with $\Re_0 < 1$.



Fig. 16. The effects of fractional-order derivative to the epidemic model with $\Re_0 > 1$.

indirect method, we show that the malware-free equilibrium point \mathbf{E}_0 is locally asymptotically stable if $\mathscr{R}_0 < 1$ and unstable if $\mathscr{R}_0 > 1$. Next, by using Lyapunov's direct method with an appropriate Lyapunov function $\mathbf{V}(\mathbf{x}(t))$, the globally asymptotic stability of the malware-free equilibrium point \mathbf{E}_0 is obtained. However, it is shown that it cannot eliminate the malware program in the network unless that the basic reproductive ratio is decreased below a lower level such that $\mathscr{R}_0 < \widetilde{\mathscr{R}}_0 < 1$. Moreover, in the case $\mathscr{R}_0 > 1$, we also point out the unique existence of the epidemic equilibrium point \mathbf{E}_* . In addition, at $\mathscr{R}_0 = 1$, the proposed fractional SE₂E₂IQR epidemic model always has a forward bifurcation. On the other hand, from the numerical simulation results, we can evaluate the importance of the model's parameters in \mathscr{R}_0 via sensitivity indices given in Section 6.1. Moreover, the effects of quarantine treatments and using fuzzy rule-based infection have been discussed.

In further research, it is planned to verify the applicability of the proposed fractional epidemic model on some particular heterogeneous complex networks such as Facebook, Instagram or Wireless sensor network, and some related qualitative



Fig. 17. The portrait phrase between Infectious state and other states with different values of c.

problems such as stabilizability or control problem. Moreover, the asymptotic behavior of endemic equilibrium \mathbf{E}_* is also an interesting question that has not been mentioned in this work and can be further considered. Furthermore, motivated by Li and Yousef [33], we intend to study the proposed problem with saturated treatment function instead of linear treatment for better fitting with reality scenario.

CRediT authorship contribution statement

Nguyen Phuong Dong: Software, Investigation, Writing – original draft, Writing – review & editing. **Hoang Viet Long:** Conceptualization, Methodology, Investigation, Writing – review & editing, Supervision. **Nguyen Thi Kim Son:** Conceptualization, Methodology, Investigation, Writing – review & editing, Supervision.



Fig. 18. The portrait phrase between Infectious state and other states with different values of ω_1 .

Declaration of competing interest

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

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Appendix

Scale-free and preferential attachment are two characteristic properties of various real-world networks such as the World Wide Web, information networks, sensor networks or networks of social users (see Fig. 20).

In Wireless Sensor Networks, it is known that sensor nodes are typically not rechargeable and hence, the residual energy is rather an important parameter and should be carefully considered in preferential attachment. It is a fact that when a new node joins in the network, it rationally wants to connect nodes with higher connections (i.e., higher degree), and for the long connection, a new node would also prefer those nodes with larger residual energy. In the following, we present briefly an introduction to the algorithms of Barabási Albert scale-free network and Energy-Aware Barabási Albert scale-free network (see Table 4).



Fig. 19. The portrait phrase between Infectious state and other states with different values of ω_2 .

Ei

where $\lambda_d + \lambda_e = 1$, $0 \le \lambda_d$, $\lambda_e \le 1$

are tunable parameters, p_i and E_i are the degree and residual energy of the node *i*, respectively.



Fig. 20. The scale-free network.

Table 4 The BA network mo	del and EABA network model.	
	The BA network model	The EABA network model
Initialization	The network contains an isolated set of N_0 nodes and assume that their links are chosen arbitrarily providing that each node has at least one link	The network contains an isolated set of N_0 nodes and assume that their links are chosen arbitrarily providing that each node has at least one link
Network growth	At each time-step, we add a new node with <i>m</i> links that connect the new node to <i>m</i> nodes of the network. After <i>t</i> time-steps, the BA network model generates a network with $N = t + N_0$ nodes and $N_0 + mt$ links.	At each time-step, we add a new node with <i>m</i> links that connect the new node to <i>m</i> nodes of the network. After <i>t</i> time-steps, the EABA network model generates a network with $N = t + N_0$ nodes and $N_0 + mt$ links.
Preferential attachment	The probability that a link of new node connects to an old node <i>i</i> is proportional to the degree p_i of the node <i>i</i> and is computed by $\mathbb{P}(i) = \frac{p_i}{\sum p_j}.$	A new node connects to <i>m</i> old nodes to form new edges with a probability $\mathbb{P}(i)$ proportional to both the degree and the residual energy of the node <i>i</i> $\mathbb{P}(i) = \frac{\lambda_d p_i}{\lambda_c E_i} + \frac{\lambda_c E_i}{\lambda_c E_i}$.

In addition, the probability that a node has k edges follows the

power-law distribution $\mathbb{P}(k)$.

Remark A.1. In the probability formula that a link of a new node connects to the old node *i*, there are two adjustable parameters λ_d and λ_e , which represent the connectivity degree and residual energy of the node *i*. Their values play an important role in the structure of the network. If $\lambda_d > \lambda_e$ then the inter-connectivity among nodes dominates the network structure, otherwise, the residual energy dominates the network structure. Especially, if $\lambda_d = 1$ and $\lambda_e = 0$ then the EABA network model becomes the well-known BA network model, while in the case $\lambda_d = 0$ and $\lambda_e = 1$, only residual energy decides the network structure and all sensor nodes are in balanced energy consumption state, that makes the lifetime of WSNs improved significantly.

Remark A.2. In EABA network model, the topology of network is dominated by not only the inter-connectivity p_i but also the residual energy of sensor node. Here, the residual energy of a sensor node *i* is determined by

$$E_i = E_0 - p_i \triangle E,$$

where *E* is the initial energy of node and $\triangle E$ is the loss energy used for establishing one link for each node. Thus, the total residual energy of all nodes at a time-step *t* can be rewritten as

$$\sum_{i} E_i = \sum_{i} (E_0 - p_i \triangle E) = (N_0 + t)E_0 - 2mt \triangle E,$$

where $N_0 + t$ is the number of sensor nodes at the time-step t and 2mt is the total degree.

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Original paper



The analysis of a fractional network-based epidemic model with saturated treatment function and fuzzy transmission

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Abstract

For understanding the influence of malware attacking on complex heterogeneous networks, this work studies a fractional network-based SIRS epidemic model with fuzzy transmission and saturated treatment function. Firstly, we apply the next-generation method to obtain the basic reproductive ratio \mathcal{R}_0 , that is an important threshold value in the investigation of asymptotic behavior of the proposed epidemic model. The obtained theoretical results indicates that the value \mathcal{R}_0 significantly depends on the topology structure of the underlying network and the malware load. In addition, we give a threshold value $\tilde{\mathcal{R}}_0 > \mathcal{R}_0$ that not only determines the existence of endemic equilibrium \mathbf{E}_* but also ensures the clean of malware programs on the network. At last, the sensitivity analysis of the threshold value \mathcal{R}_0 and some graphical simulations are presented to illustrate for the theoretical results.

Keywords: Fractional network-based epidemic model, fuzzy transmission, saturated treatment function, basic reproduction number, malware-free equilibrium, endemic equilibrium, asymptotic stability.

1 Introduction

Recently, many researchers have used mathematical modeling based on complex networks to study the spreading of malicious objects in various populations. This approach is known as an effective tool, that helps us to better understand the mechanism of epidemic diseases, to predict the evolution and influence of those diseases on the networks and decide whether they are epidemic or non-epidemic. It is well-known that the nature of epidemic models is the compartmental model, that is, the whole population is divided into some compartments and each compartment contains a number of individuals that share the same epidemiological state. In classical model, when the whole population is small and wellmixed, the rate of disease-causing contacts is often supposed to be equal. This assumption makes the model's evaluation more simply and tractable. However, it is un-realistic when the population is sufficiently large. Indeed, in many kinds of complex networks such as the Internet, Facebook, Instagram social networks, sensor network and biological chain network, etc., the connectability of different nodes on the networks is certainly un-similar and of course, the infections of malware programs to these nodes are also not the same. Therefore, there is a need to take into consideration the contact heterogeneity of complex networks when mathematically modeling epidemic models of malware program on the networks. Recently, various epidemic models with network-based settings have been analyzed for better understanding the dynamical behavior of epidemic diseases. Indeed, the paper [23] is known as a meaning pioneer work in this topic. This paper presented a study on network-based SIS epidemic model on scale-free network and carried out a detailed study with both analytical and numerical results of the proposed model. The most important contribution is the finding of a threshold value for which the epidemic is absent and the corresponding dynamical behavior. In the paper [12], Huo et al. proposed a three-compartmental epidemic model with susceptible, infected and recovered states to

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describe the virus infection on scale-free network. Firstly, the basic reproduction number \mathcal{R}_0 was evaluated to study some characteristic properties of the proposed model. After that, by establishing an appropriate Lyapunov function, the authors proved the importance of the number \mathcal{R}_0 in the study of asymptotic behavior of endemic equilibrium. In an other work, in order to better describe the realistic scenario when the number of infected individuals may exceed the treatment capacity, Li and Yousef [13] introduced the saturated treatment function in their work. After formulating a network-based SIRS epidemic model with saturation, the paper $[\square 3]$ calculated the basic reproduction number \mathcal{R}_0 and applied it to investigate asymptotic stability of equilibria, the backward bifurcation at $\mathcal{R}_0 = 1$. A novelty of this work is the use of saturated treatment function instead of linear treatment function, that can be applied for our considered model in future work. In the paper [II], Li et. al. introduced a SIRS epidemic model to describe the virus propagation on heterogeneous network. This works proved that the presence or absence of the disease on network completely depends on the value of basic reproduction number \mathcal{R}_0 , i.e., the virus-free equilibrium is globally asymptotically stable if $\mathcal{R}_0 < 1$, while if $\mathcal{R}_0 > 1$ then it is unstable. Next, the work [22] introduced an SIS model with limited treatment capacity on adaptive networks in order to study the effect of anti-virus treatments on epidemic spreading. Firstly, the author derived the existence condition of backward bifurcation or forward bifurcation at the disease-free equilibrium. Then, they discussed the effect of bifurcation direction occurring at the disease-free equilibrium to the bi-stability of endemic equilibria and the elimination of epidemic disease of the model. The obtained results are novely and interesting, however, the proposed model will be a better description for real-world network if it is extended to the case of network-based setting. Moreover, complex heterogeneous epidemic models are also applied for studying the information diffusion on the networks. For example, the rumor propagation on scale-free network was also studied by Zan et. al. [22], in which the authors formulated an SICR epidemic model and discussed the asymptotic stability of the model's equilibrium points. The contribution of this work is the introduction of a new compartment of counterattack to stifle the rumor propagation. In a recent work, Hosseini and Zandvakili 🛄 proposed a mathematical SEIRS-C model to describe the rumor spreading on social network. The highlighted contribution of this paper is the introduction of a new compartment (C) to study the effects of counter-attack factor in the rumor control. In addition, the use of fuzzy logic to express the transmission rate is also a novelty of this paper. After establishing the network-based SEIRS-C epidemic model, this paper presented the procedure to evaluate the basic reproductive ratio \mathcal{R}_0 and discussed the local asymptotic stability of disease-free equilibrium point of the proposed model.

Fractional differentiation and fractional integration, or fractional calculus in general, are considered as the effective tools for characterizing the behaviors of a large category of complex dynamical systems that the systems with integer order cannot be applied. With a long history of development, numerous studies have proved that fractional calculus has a considerable advantages and superiority when modeling many non-local phenomena, the processes with memory and hereditary properties or the motions in viscoelasticity environments. Beside the rapid popularization of fractional calculus, the study of fractional dynamical systems has been paid lots of attentions by researchers and achieved a lot of noticeable results in various fields of basic sciences and engineering such as electrical circuits, fluid dynamics, biological models, and so forth. On the other hand, with the introduction of Lyapunov function method for fractional differential systems (see [12]), the stability analysis of fractional differential systems has also attracted a lot of attentions. Due to the better ability in modeling and data-fitting, fractional calculus has been also applied to study the fractional epidemiology theory and applications. Note that most of studies in fractional epidemic models described the disease transmission by using fractional differential systems in Caputo sense. Then, it was proved that the obtained epidemic model can provide a better estimation for infection processes, as well as obtain the interesting differential equations from a mathematical viewpoint. However, we must face to a natural question that does the change in the order of derivative automatically establish consistent models w.r.t. parameters? Fortunately, the authors of [2] proved that this cannot happen in general. However, to the best of our knowledge, there have only a few studies on network-based epidemic models with fractional-order and related problems. For instance, Graef et. al. [9] proposed a fractional-order SIR epidemic model with demography to examine the user adoption and abandonment of online social networks, where adoption is analogous to infection, and abandonment is analogous to recovery. After that, they discussed the existence and uniqueness of non-negative solutions of the proposed model as well as the existence and stability of its equilibrium points by using the Jacobian matrix technique and the Lyapunov function method. In particular, a threshold R_0^{α} was established to prove that the user-free equilibrium \mathbf{E}_0 is locally asymptotically stable if $R_0^{\alpha} < 1$ and the user-prevailing equilibrium \mathbf{E}_* is globally asymptotically stable if $R_0^{\alpha} > 1$. The theoretical results were then demonstrated by a case study of fitting the considered model to some Instagram user data. However, it is a fact that in reality, the network of Instagram users is not well-mixed and it should be taken into consideration the heterogeneity of the network for better description. In the literature [13, 16, 2], the authors proposed to study different network-based epidemic models with fractional derivative. One of common characteristics of these models are the use of linear treatment function/linear immunization function, however, in reality, since each population or network has its maximal capacity for the treatment of a disease, the treatment function is often proportional to the number of the infected individuals when the capacity

of treatment is not reached, and otherwise, takes the maximal saturated level. Therefore, in this work, we propose to use a treatment function of saturated form for better description of the saturation phenomena.

Since the nature of almost natural phenomena is vagueness and uncertainty, the mathematical modeling of real-world epidemic diseases must always accept the presence of uncertainties. However, to our best knowledge, there have been very few studies considering the environmental uncertainty in any epidemic model. It is well-known in many biological models that the epidemic disease occurs only if the viral load reaches a certain threshold and obviously, the concept of viral amount is quite difficult to express by exact or certain value. This leads to the use of fuzzy set theory initiated by Zadeh [31] to get the better modeling of epidemic diseases in realistic situations. In the recent decades, fuzzy set theory has achieved a lot of significant results in the theory and application of fractional differential equations, see [6, 6, 8, 9, 10]. Despite of the tremendous potential in the modeling of epidemic models, the uses of fuzzy sets in epidemiology theory are not frequent. Some noticeable applications of fuzzy sets in epidemic models can be found in Dong et. al. [6, 6], Mahato et. al. [23], Mondal et. al. [23], Nandi et. al. [27].

Motivated by aforesaid, this work is devoted to study a fractional network-based epidemic model with three compartments: Susceptible (S), Infectious (I) and Recovered (R) with fuzzy transmission and the use of saturated treatment function. The main contributions of this work can be highlighted as follows:

- (i) Based on SIRS epidemic model, we formulate a new epidemic model with fractional-order derivative in the form of mean-field reaction rate equations, namely fractional network-based SIRS epidemic model, for describing and analyzing the spread of malicious objects on scale-free network. Especially, the proposed model considers a non-linear saturated treatment function for the better fitting with real-world situations. Indeed, in many realworld networks, there is often a maximal capacity for the treatment or immunization of an epidemic disease and moreover, when the number of infected cases take the maximal saturated level, this certainly leads to the situation that there are a number of infected being delayed for treatment. Hence, the assumption that the treatment rate is proportional to the number of infected individuals in some classical models seems less realistic. Therefore, based on the approach in [20, 63], this work proposes to use a nonlinear treatment function.
- (ii) Due to the fact that the disease infection often occurs only if the amount of malware program on the network exceeds a certain threshold value and reaches a saturation level at a finite malware load, we propose to use fuzzy membership function to represent the transmission rate σ_k , in which the infection happens only if the malware load on the network reaches a threshold value. Moreover, this work also discusses the effect of node's degree on the value of transmission rate.
- (iii) Based on the next-generation matrix method, we analytically compute the basic reproduction number \mathcal{R}_0 , that is an important threshold value in epidemiology theory. However, this work also indicates that the proposed epidemic model can't reach the endemic equilibrium state if the basic reproduction number $\tilde{\mathcal{R}}_0 < 1$. In addition, it is also proved that the existence and uniqueness of endemic equilibrium \mathbf{E}_* depends on not only the basic reproduction number \mathcal{R}_0 but also the other parameters due to the effect of saturated treatment function.
- (iv) By using the linearization method and the mathematical induction principle, we give a criteria for the local asymptotic stability or un-stability of malware-free equilibrium \mathbf{E}_0 that are related to the basic reproduction number \mathcal{R}_0 . Next, by applying the direct Lyapunov functional method with an appropriate Lyapunov function, we can conclude that the attractivity of the equilibrium \mathbf{E}_0 depends upon a threshold value $\tilde{\mathcal{R}}_0 > \mathcal{R}_0$, which proves that the condition $\mathcal{R}_0 < 1$ is not sufficient for eliminating the epidemic disease.

2 Model formulation

In this paper, we characterize the complex heterogeneous network by using Barabási-Albert scale-free network [I] to get better description for the heterogeneity of malware program 's propagation on the complex network. The structure of Barabási-Albert scale-free network can be briefly summarized as follows:

- At the initialization, the scale-free network has a small number of fully connected vertices with N_0 nodes;
- A new node with *m* links is added to the complex network after each time-step and linked to an old node *i* with a probability $\mathbb{P}(k_i) = \frac{k_i}{\sum_i k_j}$, where the parameter k_i is the degree (connectivity) of the *i*th-node.

• When the complex network attains the scale-free stationary state, it can be seen that $\mathbb{P}(k) = ck^{-3}$ is the power-law probability distribution such that a node has k connected links, where c is a parameter such that

$$\sum_{k} ck^{-3} = 1$$

2.1 The fuzzy transmission

In this work, assume that one infectious individual always comes to the contact of maximum one susceptible individual so that the degree-dependent transmission rate of the k^{th} -group $\sigma_k = \sigma k \leq k$. In addition, in order to describe the heterogeneity on the complex network, we propose to represent the transmission rate σ as a function of the available malware program. In particular, this parameter is proposed to describe through the following fuzzy set:

$$\sigma(\tau) = \begin{cases} 0 & \text{if } \tau \leq \tau_m \\ \sigma \frac{\tau - \tau_m}{\tau_0 - \tau_m} & \text{if } \tau_m < \tau \leq \tau_0 \\ \sigma & \text{if } \tau_0 < \tau \leq \tau_M. \end{cases}$$

Here, we can see that there always exists a lower threshold τ_m for the malware propagation, that is, the disease infection occurs only if the amount of malware program on the network must exceed τ_m , otherwise, the chance of transmission is negligible. In addition, the value of τ_m would depend upon both environmental characteristics and nature of malware program, that is reasonable for the choice of fuzzy membership function for transmission rate. Next, there has an upper threshold of malware load, say τ_0 , beyond which the transmission rate reaches the maximum value $\sigma(\tau) = 1$. From τ_m to τ_0 , the transmission rate is assumed to vary linearly. Furthermore, assume that the malware load has an upper bound, say τ_M . Moreover, since the nature of realistic phenomena is uncertainty, it is not natural to represent exactly the model's parameters by crisp values. For instance, in order to express the concept "amount of malware program", the use of linguistic variables seems to be more suitable. Thus, this work assumes that the malware load on the network can be classified into three classes and use linguistic terms, namely "LOW (\mathcal{A}_l) ", "MEDIUM (\mathcal{A}_m) " and "HIGH (\mathcal{A}_h) ", to characterize for each class. Additionally, in each classification, based on the threshold values τ_m, τ_0, τ_M , the malware load is expressed by using fuzzy numbers (see Figure **B**). This approach can be found in [**23**, **22**].



Figure 1: The membership function of fuzzy transmission rate σ and linguistic variables of malware load

2.2 The formulation of the fractional network-based SIRS epidemic model

In SIR epidemic model, we assume that each node can belong to one of three states: Susceptible state (S), Infectious state (S) and Recovered state (R). In order to taking into consideration the heterogeneity of scale-free networks, the whole population can't be well-mixed and the rate of disease-causing contacts must be varied depending upon the node's connectivity. Indeed, based on the number of connected links a node has per unit time, we classify the whole population into n groups and assume that nodes in a same group are dynamically equivalent. Denote $S_k(t)$, $I_k(t)$, and $R_k(t)$ by the densities of susceptible, infectious and recovered nodes with degree k at time t, respectively for $k = 1, 2, \ldots, n$ and denote $N_k(t)$ by the total number of nodes with degree k at time t. The flowchart of malware propagation of the SIRS epidemic model in the k^{th} -group is given in Figure 2.



Figure 2: The flowchart of malware propagation among compartments: Susceptible (S), Infectious (I), Recovered (R)

In several decades, fractional dynamical systems have proved their importance in real-world modeling due to the effective memory function of fractional derivatives, that has been widely used to model many non-local physical phenomena such as electric flows in signal propagation or processes in the porous media. Motivated by aforesaid, this work is devoted to study a network-based epidemic model governed by the following fractional mean-field reaction rate equations:

$$\begin{cases} {}_{0}^{C} \mathfrak{D}_{t}^{\beta} S_{k}(t) = \Lambda - \sigma_{k}(\tau) S_{k}(t) \Theta(t) - \mu S_{k}(t) + \omega R_{k}(t) \\ {}_{0}^{C} \mathfrak{D}_{t}^{\beta} I_{k}(t) = \sigma_{k}(\tau) S_{k}(t) \Theta(t) - \mu I_{k}(t) - \frac{r I_{k}(t)}{1 + \gamma \Theta(t)} \\ {}_{0}^{C} \mathfrak{D}_{t}^{\beta} R_{k}(t) = \frac{r I_{k}(t)}{1 + \gamma \Theta(t)} - (\mu + \omega) R_{k}(t), \end{cases}$$
(1)

with the initial conditions

$$S_k(0) = S_k^0, \qquad I_k(0) = I_k^0, \qquad R_k(0) = R_k^0,$$
(2)

in which the notation ${}_{0}^{C}\mathfrak{D}_{t}^{\beta}(\cdot)$ denotes for the Caputo fractional derivative of order $\beta \in (0,1]$ of state functions (see Definition \square in Appendix). The meanings of the model's parameters are given in Table 1:

No	Parameter	Description		
1	$\sigma_k(\tau)$	The degree-dependent fuzzy transmission rate		
2	r	The cure rate		
3	μ	The natural death rate		
4	Λ	The natural birth rate		
5	ω	The rate in which a recovered node turns into susceptible		

Table 1: The model's parameters

Furthermore, since the un-correlation of node's connectivity on the network is taken into consideration, the probability that a given link is connected to an infectious node can be expressed by the following function

$$\Theta(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k \mathbb{P}(k) I_k(t),$$

where $\langle k \rangle = \sum_{k=1}^{n} k \mathbb{P}(k)$ is known as the mean degree of the network. On the other hand, since the fact that an anti-malware program only attains a certain maximal treatment capacity for each epidemic disease, Zhang et. al. [33] introduced a pioneer work on the study of epidemic model with a staged treatment function $h(I) = \frac{rI}{1+\gamma I}$ compatible with the treatment capacity. This treatment function also shows its advantage in measuring the extent of the influence of the infected being delayed for treatment by using a parameter γ in treatment function. This makes our epidemic model seem more reasonable than the case using the linear function. In this paper, the terms $\frac{rI_k}{1+\gamma\Theta}$ represents for the recovery with treatment of the k^{th} -infectious group.

One can easily show that the solution of the fractional differential system (\square) with the initial condition (\square) is defined for all t > 0 and k = 1, 2, ..., n. From the view point of epidemiology, we only need to focus on the positiveness and the positively invariant set of solution. So we assume that

$$S_k(0) > 0, \quad I_k(0) \ge 0, \quad R_k(0) \ge 0, \quad k = 1, 2, \dots, n.$$

Denote

$$\mathbf{x}(t) = (S_1(t), I_1(t), R_1(t), \dots, S_n(t), I_n(t), R_n(t))^{\top},$$

$$\Sigma^+ = \left\{ \mathbf{x}(t) \in \mathbb{R}^{3n}_+ : S_k + I_k + R_k \le \frac{\Lambda}{\mu}, \ k = \overline{1, n} \right\}.$$

Due to the presence of epidemic disease on the network and by definition of the probability function $\Theta(t)$, we assume that $\Theta(t) > 0$ for each $t \ge 0$.

Lemma 2.1. Assume that $\mathbf{x}(t)$ is a solution of the fractional network-based SIRS epidemic model (\square) with the initial condition (\square) and $\mathbf{x}(0)$ belongs to Σ^+ . Then, for all t > 0, the solution $\mathbf{x}(t)$ belongs to Σ^+ .

Proof. By contrary, assume that for each $k = \overline{1, n}$, there is a $t_0 > 0$ such that $S_k(t) = 0$ at $t = t_0$, $S_k(t) > 0$ for all $0 \le t < t_0$ and $S_k(t) < 0$ for all $t_0 < t \le t_0 + \varepsilon_0$ with sufficiently small $\varepsilon_0 > 0$. Then, we consider two following cases:

Case 1: If $I_k(t) \ge 0$ for all $t \ge 0$, then we have

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}R_{k}(t) = \frac{rI_{k}(t)}{1+\gamma\Theta(t)} - (\omega+\mu)R_{k}(t) \ge -(\omega+\mu)R_{k}(t)$$

Then, by applying fractional comparison principle (Lemma 10, [L3]), it implies that the function

$$R_k(t) \ge R_k(0)\mathbb{E}_{\beta}\left(-(\omega+\mu)t^{\beta}\right) \ge 0,$$

for all $t \ge 0$. As a result, at $t = t_0$, we have ${}_0^C \mathfrak{D}_t^{\beta} S_k(t)|_{t=t_0} = \Lambda + \omega R_k(t_0) > 0$. By using Lemma \square for a = 0 and $t = t_0 + \varepsilon_0$, it implies that $S_k(t_0 + \varepsilon_0) = S_k(0) + \frac{\varepsilon_0^{\beta}}{\Gamma(\beta)} {}_0^C \mathfrak{D}_t^{\beta} S_k(t)|_{t=\xi}$, where $t_0 \le \xi \le t_0 + \varepsilon_0$ and $0 < \varepsilon_0 \ll 1$ is small enough such that ${}_0^C \mathfrak{D}_t^{\beta} S_k(t)|_{t=\xi} \ge 0$. This means $S_k(t_0 + \varepsilon_0) > 0$, which contradicts our assumption.

Case 2: If there exists a time $t_1 > 0$ such that $I_k(t) = 0$ at $t = t_1$, $I_k(t) > 0$ for all $t \in [0, t_1)$ and $I_k(t) < 0$ for all $t_1 < t \le t_1 + \varepsilon_1$ with sufficiently small $\varepsilon_1 > 0$, then our proof is proceeded in two following sub-cases: <u>Sub-case 1</u>: If $t_1 \ge t_0$ then by using similar arguments as in Case 1, we can prove that the functions $I_k(t), R_k(t)$ are all

non-negative on $[0, t_1]$ and $S_k(t_0 + \varepsilon_0) > 0$, which leads to a contradiction. Sub-case 2: If $t_1 < t_0$ then we have $S(t_1) > 0$ and $\Theta(t_1) > 0$. Moreover, at the time $t = t_1$, we receive

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}I_{k}(t)|_{t=t_{1}}=\sigma_{k}(\tau)S_{k}(t_{1})\Theta(t_{1})>0$$

Then, we can choose $0 < \varepsilon_1 \ll 1$ such that ${}_0^C \mathfrak{D}_t^\beta S_k(t)|_{t=\overline{\xi}} \ge 0$ with $\overline{\xi} \in [t_1, t_1 + \varepsilon_1]$. Next, by using Lemma \square for a = 0 and $t = t_1 + \varepsilon_1$, we obtain

$$I_k(t_1 + \varepsilon_1) = I_k(0) + \frac{\varepsilon_1^\beta}{\Gamma(\beta)} {}^C_0 \mathfrak{D}_t^\beta I_k(t)|_{t=\overline{\xi}}.$$

It implies that $I_k(t_1 + \varepsilon_1) > 0$, which contradicts our assumption. Therefore, we can conclude that $S_k(t) > 0$ is always positive for all $t \ge 0$. Finally, by doing similar arguments, we can also prove that the functions $I_k(t)$ and $R_k(t)$ are all non-negative for all $t \ge 0$ and $k = \overline{1, n}$.

Next, by using the second assumption, we have $N_k(0) = S_k(0) + I_k(0) + R_k(0) \le \frac{\Lambda}{\mu}$. By summing up all fractional differential equations of the system (**D**), we immediately obtain

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}N_{k}(t) = \Lambda - \mu N_{k}(t).$$

$$(3)$$

By applying Example 4.9 in [13], the general solution of the fractional differential equation (3) is given by

$$N_k(t) = N_k(0)\mathbb{E}_{\beta}(-\mu t^{\beta}) + \Lambda \int_0^t \frac{\mathbb{E}_{\beta,\beta}(-\mu(t-\tau)^{\beta})}{(t-\tau)^{1-\beta}}d\tau = N_k(0)\mathbb{E}_{\beta}(-\mu t^{\beta}) + \Lambda t^{\beta}\mathbb{E}_{\beta,\beta+1}(-\mu t^{\beta}).$$

Then, by choosing $\alpha_1 = \beta$, $\alpha_2 = 1$ and $x = -\mu t^{\beta}$, Lemma \square implies that

$$N_k(t) = N_k(0)\mathbb{E}_{\beta}(-\mu t^{\beta}) + \Lambda t^{\beta}\mathbb{E}_{\beta,\beta+1}(-\mu t^{\beta}) = N_k(0)\mathbb{E}_{\beta}(-\mu t^{\beta}) + \frac{\Lambda}{\mu}\left[1 - \mathbb{E}_{\beta}(-\mu t^{\beta})\right]$$
Since $\mathbf{x}(0) \in \Sigma^+$, it implies that $N_k(0) \leq \frac{\Lambda}{\mu}$ and it should be noted that $0 \leq \mathbb{E}_{\beta}(-\mu t^{\beta}) \leq 1$ for all $t \geq 0$. Thus, we have

$$N_k(t) \le \frac{\Lambda}{\mu} \mathbb{E}_{\beta}(-\mu t^{\beta}) + \frac{\Lambda}{\mu} \left[1 - \mathbb{E}_{\beta}(-\mu t^{\beta}) \right] = \frac{\Lambda}{\mu},$$

which means that Σ^+ is a positively invariant set for the fractional network-based epidemic model (**D**).

3 The basic reproduction number \mathcal{R}_0 and equilibrium points

3.1 The evaluation of basic reproduction number \mathcal{R}_0

It can be easily seen that the fractional network-based SIRS epidemic model (**D**) admits a malware-free equilibrium (MFE) $\mathbf{E}_0 = (\underbrace{\frac{\Lambda}{\mu}, 0, 0, \dots, \frac{\Lambda}{\mu}, 0, 0}_{3n})$. Now, our aim is to find a threshold value which plays a key role in not only the

unique existence of endemic equilibrium \mathbf{E}_* but also the local asymptotic behavior of the model (**D**). This value is called basic reproduction number and denoted by \mathcal{R}_0 . In epidemiology, the basic reproduction number \mathcal{R}_0 is the number of cases directly caused by an infected individual throughout its infectious period. The essential significance of \mathcal{R}_0 are determining if an infectious disease can spread in a population and determining the proportion of the population should be immunized through vaccination to eliminate the epidemic disease. Note that \mathcal{R}_0 is not a biological constant for a pathogen as it is also affected by other factors such as environmental conditions and the behavior of the infected population. In order to evaluate the basic reproduction number, we propose to apply the next-generation matrix method introduced by Diekmann et al. [**D**]. It should be noted that the infection causing compartment of the proposed model is the compartment (I). Therefore, by using the second equation of the system (**D**), we find out that the gain term and lost term for the epidemic model are as follows:

• The gain term is $\sigma_k(\tau)S_k(t)\Theta(t)$.

• The loss term is
$$\mu I_k(t) + \frac{rI_k(t)}{1 + \gamma \Theta(t)}$$

Then, the rate matrix \mathcal{F} of new infections appearance at the equilibrium \mathbf{E}_0 can be given by

$$\mathcal{F} = \frac{\sigma(\tau)\Lambda}{\mu\langle k\rangle} \begin{bmatrix} 1\mathbb{P}(1) & 2\mathbb{P}(2) & \cdots & n\mathbb{P}(n) \\ 2\mathbb{P}(1) & 2^{2}\mathbb{P}(2) & \cdots & 2n\mathbb{P}(n) \\ \vdots & \vdots & \ddots & \vdots \\ n\mathbb{P}(1) & 2n\mathbb{P}(2) & \cdots & n^{2}\mathbb{P}(n) \end{bmatrix} = \frac{\sigma(\tau)\Lambda}{\mu\langle k\rangle} \begin{bmatrix} 1\\ 2\\ \vdots\\ n \end{bmatrix} \begin{bmatrix} \mathbb{P}(1) & 2\mathbb{P}(2) & \cdots & n\mathbb{P}(n) \end{bmatrix},$$

and the transition matrix \mathcal{V} of infected states is $\mathcal{V} = (\mu + r)\mathbf{I}_n$, where \mathbf{I}_n is the $n \times n$ identity matrix. The basic reproduction number \mathcal{R}_0 is then the largest eigenvalue of the matrix \mathcal{FV}^{-1} given by

$$\frac{\sigma(\tau)\Lambda}{\mu(\mu+r)\langle k\rangle} \begin{bmatrix} 1\\ 2\\ \vdots\\ n \end{bmatrix} \begin{bmatrix} \mathbb{P}(1) & 2\mathbb{P}(2) & \cdots & n\mathbb{P}(n) \end{bmatrix}.$$

Therefore, we directly get that $\mathcal{R}_0 = \frac{\sigma(\tau)\Lambda\langle k^2 \rangle}{\mu(r+\mu)\langle k \rangle}$, where $\langle k^2 \rangle = \sum_{k=1}^n k^2 \mathbb{P}(k)$.

Remark 3.1. According to the formula of \mathcal{R}_0 , we can conclude that the threshold value \mathcal{R}_0 is directly proportional to the network structure's parameter $\frac{\langle k^2 \rangle}{\langle k \rangle}$. This means that the network's heterogeneity can directly affect to the malware widespread on the network.

3.2 The existence of an endemic equilibrium

The following theorem presents an interesting result on the existence and uniqueness of an endemic equilibrium (EE) of the network-based epidemic model (\square).

Theorem 3.2. Assume that $\Lambda \leq \mu \left(1 + \frac{r}{(\mu+\omega)(1+\gamma)}\right)$. Then, the following assertions are fulfilled:

- 1. If $\tilde{\mathcal{R}}_0 < 1$ then the fractional network-based SIRS epidemic model (\square) doesn't have any endemic equilibrium.
- 2. If $\mathcal{R}_0 > 1$ then the fractional network-based SIRS epidemic model (\square) admits at least one endemic equilibrium \mathbf{E}_* given by

$$\mathbf{E}_* = (S_1^*, I_1^*, R_1^*, \dots, S_n^*, I_n^*, R_n^*),$$

where

$$S_k^* = \frac{1}{\sigma_k(\tau)\Theta^*} \left(\mu + \frac{r}{1+\gamma\Theta^*} \right) I_k^*, \quad R_k^* = \frac{rI_k^*}{(\mu+\omega)(1+\gamma\Theta^*)}, \quad \Theta^* = \frac{1}{\langle k \rangle} \sum_{i=1}^n i \mathbb{P}(i)I_i^*,$$
$$I_k^* = \frac{\Lambda \sigma_k(\tau)\Theta^*}{\mu \left[\mu + \sigma_k(\tau)\Theta^* + \frac{r\sigma_k(\tau)\Theta^*}{(\mu+\omega)(1+\gamma\Theta^*)} + \frac{r}{1+\gamma\Theta^*} \right]}.$$

Moreover, if $\gamma < \frac{\sigma(\tau)}{\mu + \omega}$ then the endemic equilibrium \mathbf{E}_* of the network-based epidemic model (**D**) is unique.

Proof. Assume that $\mathbf{E}_* = (S_1^*, I_1^*, R_1^*, \dots, S_n^*, I_n^*, R_n^*)$ is an endemic equilibrium of the fractional network-based SIRS epidemic model (1). Then, for each $k = 1, 2, \dots, n$, the triple (S_k^*, I_k^*, R_k^*) satisfies the following system

$$\begin{cases} \sigma_k(\tau)S_k\Theta - \mu I_k - \frac{rI_k}{1 + \gamma\Theta} &= 0\\ \frac{rI_k}{1 + \gamma\Theta} - (\mu + \omega)R_k &= 0\\ S_k + I_k + R_k &= \frac{\Lambda}{\mu}, \end{cases}$$
(4)

where $\Theta = \frac{1}{\langle k \rangle} \sum_{i=1}^{n} i \mathbb{P}(i) I_i$. Next, by expressing the variables S_k , R_k in the two first equations of the system (**1**) in the terms of I_k , we immediately get

$$S_k^* = \frac{1}{\sigma_k(\tau)\Theta^*} \left(\mu + \frac{r}{1+\gamma\Theta^*}\right) I_k^*, \qquad \qquad R_k^* = \frac{r}{(\mu+\omega)(1+\gamma\Theta^*)} I_k^*.$$

After that, we substitute the expressions of S_k^* and R_k^* into the last equation of the system (1), we receive

$$\left[1 + \frac{1}{\sigma_k(\tau)\Theta^*} \left(\mu + \frac{r}{1 + \gamma\Theta^*}\right) + \frac{r}{(\mu + \omega)(1 + \gamma\Theta^*)}\right] I_k^* = \frac{\Lambda}{\mu}$$

$$\Lambda \sigma_k(\tau)\Theta^*$$

or equivalently, $I_k^* = \frac{\Lambda \sigma_k(\tau) \Theta^*}{\mu \left[\mu + \sigma_k(\tau) \Theta^* + \frac{r \sigma_k(\tau) \Theta^*}{(\mu + \omega)(1 + \gamma \Theta^*)} + \frac{r}{1 + \gamma \Theta^*} \right]}$. Next, by substituting I_k^* into the expression of the

function $\Theta(t)$, the equation $\Theta^* = \frac{1}{\langle k \rangle} \sum_{i=1}^n i \mathbb{P}(i) I_i^*$ becomes the following self-consistency equation

$$\Theta^* = \frac{1}{\langle k \rangle} \sum_{i=1}^n \frac{\Lambda \sigma(\tau) i^2 \mathbb{P}(i) \Theta^*}{\mu \left[\mu + \sigma_i(\tau) \Theta^* + \frac{r \sigma_i(\tau) \Theta^*}{(\mu + \omega)(1 + \gamma \Theta^*)} + \frac{r}{1 + \gamma \Theta^*} \right]}.$$
(5)

It should be noted that the self-consistency equation (**b**) always admits the trivial solution $\Theta \equiv 0$. Now, we aim to determine a sufficient condition for which the equation (**b**) has a solution $\Theta^* \in (0, 1)$. Firstly, we define

$$f(\Theta) = \frac{1}{\langle k \rangle} \sum_{i=1}^{n} \frac{\Lambda \sigma(\tau) i^2 \mathbb{P}(i)}{\mu \left[\mu + \sigma_i(\tau)\Theta + \frac{r\sigma_i(\tau)\Theta}{(\mu + \omega)(1 + \gamma\Theta)} + \frac{r}{1 + \gamma\Theta} \right]}$$

Here, we can see that

• The function $f(\Theta)$ is continuous on the closed interval [0, 1] and differentiable on the open interval (0, 1).

•
$$f(0) = \frac{\Lambda \sigma(\tau)}{\mu(r+\mu)\langle k \rangle} \sum_{k=1}^{n} k^2 \mathbb{P}(k) = \mathcal{R}_0.$$

• For each $\Theta \in [0,1]$, we have $f(\Theta) < \frac{1}{\langle k \rangle} \sum_{i=1}^n \frac{\Lambda \sigma(\tau) i^2 \mathbb{P}(i)}{\mu^2} = \tilde{\mathcal{R}}_0.$

• At $\Theta = 1$, we have

$$f(1) = \frac{1}{\langle k \rangle} \sum_{i=1}^{n} \frac{\Lambda \sigma(\tau) i^2 \mathbb{P}(i)}{\mu \left[\mu + \frac{r}{1+\gamma} + \sigma_i(\tau) \left(1 + \frac{r}{(\mu+\omega)(1+\gamma)} \right) \right]} < \frac{1}{\langle k \rangle} \sum_{i=1}^{n} \frac{\Lambda \sigma(\tau) i^2 \mathbb{P}(i)}{\mu \sigma_i(\tau) \left(1 + \frac{r}{(\mu+\omega)(1+\gamma)} \right)} = 1$$

Then, the non-trivial solution of the equation (\mathbf{S}) is the solution of the following equation

$$\frac{1}{\langle k \rangle} \sum_{i=1}^{n} \frac{\Lambda \sigma(\tau) i^2 \mathbb{P}(i)}{\mu \left[\mu + \sigma_i(\tau)\Theta + \frac{r\sigma_i(\tau)\Theta}{(\mu+\omega)(1+\gamma\Theta)} + \frac{r}{1+\gamma\Theta} \right]} = 1.$$
(6)

Note that if $\tilde{\mathcal{R}}_0 \leq 1$ then it implies that $f(\Theta) < \tilde{\mathcal{R}}_0 \leq 1$. As a result, there doesn't exist any value $\Theta \in [0, 1]$ such that the equation (**B**) holds, or equivalently, there doesn't exist any endemic equilibrium when $\tilde{\mathcal{R}}_0 \leq 1$. The first assertion of the theorem is completed.

By using the assumption $\mathcal{R}_0 > 1$, it directly follows that f(0) > 1. Therefore, by virtue of Intermediate Value theorem, the equation (**b**) has at least one solution $\Theta \in (0, 1)$, that is also the non-trivial solution of the equation (**b**). As a consequence, the solution $\Theta^* \in (0, 1)$ of the self-consistency equation (**b**) will solve the endemic equilibrium \mathbf{E}_* . In order to prove the uniqueness of the endemic equilibrium \mathbf{E}_* , let us compute

$$\frac{d}{d\Theta}f(\Theta) = \frac{d}{d\Theta}\left\{\sum_{k=1}^{n}\frac{A_k(1+\gamma\Theta)}{B_k(\Theta)}\right\} = \sum_{k=1}^{n}\frac{\gamma A_k B_k(\Theta) - A_k(1+\gamma\Theta)\frac{d}{d\Theta}B_k(\Theta)}{B_k^2(\Theta)},$$

where for simplicity in representation, we denote

$$A_k = \frac{\Lambda \sigma(\tau) k^2 \mathbb{P}(k)}{\mu \langle k \rangle}, \qquad \qquad B_k(\Theta) = (1 + \gamma \Theta)(\mu + \sigma_k(\tau)\Theta) + r + \frac{r \sigma_k(\tau)\Theta}{\mu + \omega}$$

By some fundamental computations, we obtain

$$\frac{d}{d\Theta}f(\Theta) = \sum_{k=1}^{n} \frac{r\gamma A_k - \frac{r\sigma_k(\tau)A_k}{\mu+\omega} - \sigma_k(\tau)A_k(1+\gamma\Theta)^2}{B_k^2(\Theta)} = \sum_{k=1}^{n} \frac{rA_k\left(\gamma - \frac{\sigma_k(\tau)}{\mu+\omega}\right) - \sigma_k(\tau)A_k(1+\gamma\Theta)^2}{B_k^2(\Theta)}.$$

Therefore, if $\gamma \leq \frac{\sigma(\tau)}{\mu + \omega}$ then the derivative $\frac{d}{d\Theta}f(\Theta) < 0$ for all $\Theta \in [0, 1]$ and hence, the equation (**B**) has a unique solution $\Theta \in (0, 1)$. The proof is completed.

4 The asymptotic behavior of malware-free equilibrium E_0

4.1 The local asymptotic stability

Theorem 4.1. The malware-free equilibrium \mathbf{E}_0 of the fractional network-based SIRS epidemic model (\square) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof. Based on the stability theory of fractional dynamical systems, the local asymptotic stability of the malware-free equilibrium \mathbf{E}_0 can be determined by finding the modulus of eigenvalue's arguments of Jacobi matrix $J(\mathbf{E}_0)$. Let us consider the Jacobi matrix at \mathbf{E}_0 of the epidemic model (II) in the following form

$$\mathbf{J}(\mathbf{E}_{0}) = \begin{bmatrix} \mathbf{J}_{11} & \mathbf{J}_{12} & \cdots & \mathbf{J}_{1n} \\ \mathbf{J}_{21} & \mathbf{J}_{22} & \cdots & \mathbf{J}_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{J}_{n1} & \mathbf{J}_{n2} & \cdots & \mathbf{J}_{nn} \end{bmatrix}_{3n \times 3n}^{*},$$

where J_{kk} , J_{kk} are 3×3 -square matrices given by

$$\mathbf{J}_{kk} = \begin{bmatrix} \frac{\sigma_k(\tau)k\Lambda\mathbb{P}(k)}{\mu\langle k \rangle} - (\mu + r) & 0 & 0\\ -\frac{\sigma_k(\tau)k\Lambda\mathbb{P}(k)}{\mu\langle k \rangle} & -\mu & \omega\\ r & 0 & -(\omega + \mu) \end{bmatrix}, \qquad \mathbf{J}_{ki} = \begin{bmatrix} \frac{\sigma_k(\tau)\Lambda i\mathbb{P}(i)}{\mu\langle k \rangle} & 0 & 0\\ -\frac{\sigma_k(\tau)\Lambda i\mathbb{P}(i)}{\mu\langle k \rangle} & 0 & 0\\ 0 & 0 & 0 \end{bmatrix} \qquad (k \neq i),$$

for each $k, j = \overline{1, n}$. Then, by applying the mathematical induction principle, the characteristic polynomial w.r.t. the Jacobi matrix $J(\mathbf{E}_0)$ can be given by

$$\mathcal{P}(\tilde{\lambda}) = (\tilde{\lambda} + \mu)^n (\tilde{\lambda} + \mu + \omega)^n (\tilde{\lambda} + \mu + r)^{n-1} \left(\tilde{\lambda} + (\mu + r) - \frac{1}{\mu \langle k \rangle} \sum_{k=1}^n \sigma_k(\tau) \Lambda k \mathbb{P}(k) \right)$$

According to Theorem 7.20 in [**G**], the malware-free equilibrium \mathbf{E}_0 is locally asymptotically stable if and only if all eigenvalues $\left\{\tilde{\lambda}_j\right\}_{j=\overline{1.3n}}$ of the Jacobi matrix $\mathbf{J}(\mathbf{E}_0)$ satisfy

$$\left|\arg\left(\tilde{\lambda}_{j}\right)\right| > \frac{\beta\pi}{2}, \qquad j = 1, 2, \dots, 3n.$$

It can easily verified that the characteristic equation $\mathcal{P}(\tilde{\lambda}) = 0$ has 3n - 1 negative solutions, namely $\tilde{\lambda} = -\mu$ with multiplicity n, $\tilde{\lambda} = -(\mu + \omega)$ with multiplicity n and $\tilde{\lambda} = -(\mu + r)$ with multiplicity n - 1. The last eigenvalue of the characteristic polynomial $\mathcal{P}(\tilde{\lambda})$ is

$$\tilde{\lambda} = -(\mu + r) + \frac{1}{\mu \langle k \rangle} \sum_{k=1}^{n} \sigma_k(\tau) \Lambda k \mathbb{P}(k) = (\mu + r) \left(\frac{\Lambda \sigma(\tau)}{\mu(\mu + r) \langle k \rangle} \sum_{k=1}^{n} k^2 \mathbb{P}(k) - 1 \right) = (\mu + r)(\mathcal{R}_0 - 1).$$

By using the assumption $\mathcal{R}_0 < 1$, we immediately deduce that eigenvalues of the Jacobi matrix $J(\mathbf{E}_0)$ are all negative and hence, their arguments $\arg\left(\tilde{\lambda}_j\right) = \pi$ for all $j = \overline{1, 3n}$. In addition, since $\beta \in (0, 1]$, we directly get that

$$\left| \arg \left(\tilde{\lambda}_j \right) \right| = \pi > \frac{\beta \pi}{2} \quad \text{for all } j = \overline{1, 3n}.$$

Therefore, by Theorem 7.20 in [**B**], we can conclude that the malware-free equilibrium \mathbf{E}_0 is locally asymptotically stable. Otherwise, if $\mathcal{R}_0 > 1$ then the eigenvalue $\tilde{\lambda} = (\mu + r)(\mathcal{R}_0 - 1)$ is real and strictly positive, i.e. it has zero argument, and hence, the malware-free equilibrium \mathbf{E}_0 is unstable.

Remark 4.2. The main approach of Theorem [..] is based on linearization method and stability criteria for fractional differential system in Theorem 7.20 in [I] related to modulus of eigenvalue's arguments. By applying linearization method, we get that the Jacobi matrix $J(E_0)$ is a square matrix of order 3n and then, the mathematical induction principle follows that the matrix $J(E_0)$ has 3n - 1 negative eigenvalues and the last eigenvalue depending on the sign of $\mathcal{R}_0 - 1$. Therefore, we can conclude that the basic reproduction number \mathcal{R}_0 plays a key role in the local asymptotic behavior of the network-based epidemic model [I]. At $\mathcal{R}_0 = 1$, since the eigenvalue $\tilde{\lambda} = (\mu + r)(\mathcal{R}_0 - 1)$ is zero then its argument is undefined. By using the remark after Theorem 2 in [I], we can conclude that the malware-free equilibrium E_0 is stable but not asymptotically stable.

4.2 The global asymptotic stability

In the following, we discuss the global asymptotic stability of the malware-free equilibrium for the network-based epidemic model (\blacksquare) . For this aim, we denote a threshold value

$$ilde{\mathcal{R}}_0 = rac{\Lambda \sigma(au) \langle k^2
angle}{\mu^2 \langle k
angle}.$$

Now, we will prove that $\tilde{\mathcal{R}}_0$ is the threshold value for which the malware-free equilibrium \mathbf{E}_0 is globally asymptotically stable. Indeed, we have

Theorem 4.3. If the parameter $\tilde{\mathcal{R}}_0$ satisfies $\tilde{\mathcal{R}}_0 < 1$ then the malware-free equilibrium \mathbf{E}_0 of the fractional networkbased SIRS epidemic model (**D**) is globally asymptotically stable.

Proof. Let $\mathbf{x}(t) = \{(S_k(t), I_k(t), R_k(t))\}_{k=1}^n$ be a solution of the fractional network-based SIRS epidemic model (**D**). For simplicity in representation, we denote

$$S_k := S_k(t), \qquad I_k := I_k(t),$$

$$R_k := R_k(t), \qquad \Theta := \Theta(t).$$

Now, we will apply the direct Lyapunov method to discuss the global asymptotic stability of the equilibrium \mathbf{E}_0 . In particular, we construct the Lyapunov function along the solution $\mathbf{x}(t)$ by a function $\mathbf{V}: \Sigma^+ \to \mathbb{R}$, given by

$$\mathbf{V}(\mathbf{x}(t)) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k \mathbb{P}(k) \left\{ S_k - \frac{\Lambda}{\mu} - \frac{\Lambda}{\mu} \ln\left(\frac{\mu S_k}{\Lambda}\right) + I_k + R_k \right\}$$
$$= \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k \mathbb{P}(k) \left\{ \Psi(S_k) + I_k(t) + R_k(t) \right\}.$$

According to Remark \square , we directly get that $\Psi(S_k) = S_k - \frac{\Lambda}{\mu} - \frac{\Lambda}{\mu} \ln\left(\frac{\mu S_k}{\Lambda}\right)$ is a non-negative function for all $S_k > 0$ and attains the global minimum at $S_k = \frac{\Lambda}{\mu}$. In addition, based on the non-negativeness of the solution $\mathbf{x}(t)$ stated in Lemma \square , it implies that the function $\mathbf{V}(\mathbf{x}(t))$ is non-negative definite with respect to malware-free equilibrium \mathbf{E}_0 . Next, by taking the fractional derivative in Caputo sense for the function $\mathbf{V}(\mathbf{x}(t))$ along $\mathbf{x}(t)$ and then, applying Lemma \square , we receive

where

$$\left(1 - \frac{\Lambda}{\mu S_k}\right)_0^C \mathfrak{D}_t^\beta S_k = 2\Lambda - \sigma_k(\tau) S_k \Theta - \mu S_k + \omega R_k - \frac{\Lambda^2}{\mu S_k} + \frac{\sigma_k(\tau) \Lambda \Theta}{\mu} - \frac{\omega \Lambda R_k}{\mu S_k}$$

$$= -\frac{\mu}{S_k} \left(\frac{\Lambda^2}{\mu^2} - 2S_k \frac{\Lambda}{\mu} + S_k^2\right) + \frac{\sigma_k(\tau) \Lambda \Theta}{\mu} - \sigma_k(\tau) S_k \Theta + \omega R_k \left(1 - \frac{\Lambda}{\mu S_k}\right),$$

$$(7)$$

and

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}I_{k} + {}_{0}^{C}\mathfrak{D}_{t}^{\beta}R_{k} = \sigma_{k}(\tau)S_{k}\Theta - \mu I_{k} - (\mu + \omega)R_{k}.$$
(8)

By combining two inequalities (\square) and (\square) , we receive

$$\left(1 - \frac{\Lambda}{\mu S_k}\right)_0^C \mathfrak{D}_t^\beta S_k + {}_0^C \mathfrak{D}_t^\beta I_k + {}_0^C \mathfrak{D}_t^\beta R_k \le -\frac{\mu}{S_k} \left(\frac{\Lambda}{\mu} - S_k\right)^2 + \frac{\sigma_k(\tau)\Lambda\Theta}{\mu} - \mu I_k + \omega R_k \left(1 - \frac{\omega + \mu}{\omega} - \frac{\Lambda}{\mu S_k}\right).$$

For each $t \ge 0$ and $\mathbf{x}(t) \in \Sigma^+$, note that $-\frac{\mu}{S_k} \left(\frac{\Lambda}{\mu} - S_k\right)^2 \le 0$ and $\omega R_k \left(1 - \frac{\omega + \mu}{\omega} - \frac{\Lambda}{\mu S_k}\right) \le 0$. Hence, we have

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\mathbf{V}(\mathbf{x}(t)) \leq \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k\mathbb{P}(k) \left[\frac{\sigma_{k}(\tau)\Lambda\Theta}{\mu} - \mu I_{k} \right]$$
$$= \mu\Theta \left[\frac{\sigma(\tau)}{\mu^{2}\langle k \rangle} \sum_{k=1}^{n} \Lambda k^{2}\mathbb{P}(k) - 1 \right]$$
$$= \mu\Theta(\tilde{\mathcal{R}}_{0} - 1).$$
(9)

Thus, it implies that if $\tilde{\mathcal{R}}_0 < 1$ then ${}_0^C \mathfrak{D}_t^\beta \mathbf{V}(\mathbf{x}(t)) < 0$. In addition, ${}_0^C \mathfrak{D}_t^\beta \mathbf{V}(\mathbf{x}(t)) = 0$ if and only if

$$S_k = \frac{\Lambda}{\mu}, \quad I_k = R_k = 0, \qquad k = 1, 2, \dots, n.$$

The largest invariant set of $\left\{ \mathbf{x}(t) \in \Sigma^+ : {}_0^C \mathfrak{D}_t^\beta \mathbf{V}(\mathbf{x}(t)) = 0 \right\}$ is the singleton set $\{\mathbf{E}_0\}$. Therefore, by using Lemma 4.6 in $[\square]$, the proof is completed.

Remark 4.4. The key tool to study the global asymptotic stability of the malware-free equilibrium \mathbf{E}_0 is the choice of an appropriate Lyapunov function $\mathbf{V}(\mathbf{x}(t))$. In general, the Lyapunov functions are often constructed in quadratic form or in a special form associated with dynamic of the proposed differential systems. In this theorem, the use of non-negative function $\Psi(S_k)$ plays an important role to associate the negative definite property of the Caputo fractional derivative ${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\mathbf{V}(\mathbf{x}(t))$ with the value of the threshold $\tilde{\mathcal{R}}_{0}$. Some preceding works, also used this type of Lyapunov function, can be found in [9, 13, 17, 19, 20].

Remark 4.5. By the inequality (**D**), we have

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\mathbf{V}(\mathbf{x}(t)) \leq (\mu+r)\Theta\left[\frac{\Lambda\sigma(\tau)}{\mu(\mu+r)\langle k\rangle}\sum_{k=1}^{n}k^{2}\mathbb{P}(k) - \frac{\mu}{\mu+r}\right] \leq (\mu+r)\Theta\left(\mathcal{R}_{0} - \frac{\mu}{\mu+r}\right).$$

This requires $\mathcal{R}_0 \leq \frac{\mu}{\mu+r} < 1$ to ensure ${}_0^C \mathfrak{D}_t^\beta \mathbf{V}(\mathbf{x}(t)) \leq 0$. Therefore, we can conclude that the condition $\mathcal{R}_0 < 1$ is not sufficient enough to eliminate the epidemic disease on network. that is the reason why we give a threshold value $\tilde{\mathcal{R}}_0 > \mathcal{R}_0$ to evaluate the global asymptotic stability of malware-free equilibrium \mathbf{E}_0 .

Applications $\mathbf{5}$

Hand-Foot-Mouth Disease (HFMD) is a common infectious disease for children, especially children are under 5 years old. From December 19th, 2020 to January 18th, 2021, Vietnam had 2, 901 cases of HFMD, that is 2.3 times higher than the same period last year, see [32]. In this section, we will apply the proposed fractional network-based SIRS epidemic model (**D**) for describing the dynamic of HFMD in the population of children below the age of 10. Here, since the HFMD is infected from a child to another through direct contacts and it is obvious that the contact between children in reality is not well-mixed, we will use Barabási-Albert scale-free network to describe the contact heterogeneity of children. We assume that the maximum contact of a child is at his school with n = 20 and the probability that a randomly child has degree k, i.e., he is in the contact with k other children, is given by $\mathbb{P}(k) = ck^{-3}$, where c is known as a balanced parameter such that $\sum_{k=1}^{20} ck^{-3} = 1$. Indeed, since $\sum_{k=1}^{20} \mathbb{P}(k) = 1$, it follows that the constant c = 0.8327 by using Matlab computation. Moreover, the parameters of network structure $\langle k \rangle$ and $\langle k^2 \rangle$ are computed by MatLab program as follows:

• The parameter $\langle k \rangle = \sum_{k=1}^{50} ck^{-2} \approx 1.3291$ is the average degree of the network, that is, on the average, each child

in the network will contact with 1.3291 other children.

• The parameter $\langle k^2 \rangle = \sum_{k=1}^{n} ck^{-1} \approx 2.9933$ is the second moment of the node degree that measures the fluctuation of the degree distribution.

Moreover, the used parameters of the epidemic model are given in Table 2. Here, all the parameter values are chosen hypothetically due to the unavailability of real-world data.

Parameter	Value	Parameter	Value
Λ	0.12	μ	0.05
σ	0.1	ω	0.06
r	0.8	γ	4

Table 2: The used parameters in the SIRS epidemic model

5.1 The influence of the fuzzy transmission rate to \mathcal{R}_0

Since the transmission rate $\sigma_k(\tau) = k\sigma(\tau)$ is represented as a function of viral load τ , the basic reproduction number \mathcal{R}_0 then can be known as a fuzzy number w.r.t. the viral load. Based on the analysis results presented in Section III and Section IV, the threshold value \mathcal{R}_0 has an essential role in the asymptotic behavior of the model. In the following, we will discuss the influence of viral load to the threshold value \mathcal{R}_0 and the viral infection. We assume that the amount of malware τ in the population has a linguistic meaning classified as "LOW", "MEDIUM" and "HIGH".

Case I. If the amount of viruses is "LOW", i.e., the triangular fuzzy number $\mathcal{A}_l = (\tau_c - \delta, \tau_c, \tau_c + \delta)$ satisfies $\tau_c + \delta < \tau_m$, then the transmission rate $\sigma_k(\tau) = 0$. In addition, it is clear that the basic reproduction number \mathcal{R}_0 then becomes zero, which means that the disease vanishes from the network, i.e., the malware-free equilibrium \mathbf{E}_0 is asymptotically stable. This case can be understood that the disease is not enough to cause the infection or the infected children are being isolated with the population, i.e., they have less importance on the network.

Case II. If the amount of viruses is "MEDIUM", i.e., the triangular fuzzy number $\mathcal{A}_m = (\tau_c - \delta, \tau_c, \tau_c + \delta)$ satisfies $\tau_c - \delta \geq \tau_m$ and $\tau_c + \delta < \tau_0$, then the transmission rate $\sigma_k(\tau)$ is considered a linear function w.r.t. the malware load τ . As a consequence, we also deduce that the basic reproduction number $\mathcal{R}_0 := \mathcal{R}_0(\tau)$, given by

$$\mathcal{R}_0(\tau) = \frac{\Lambda \sigma \langle k^2 \rangle}{\mu(r+\mu) \langle k \rangle} \frac{\tau - \tau_m}{\tau_0 - \tau_m},$$

is an increasing function w.r.t. the viral load τ . It leads to a fact that the higher viral load is, the bigger value the basic reproduction number \mathcal{R}_0 gets.

Case III. If the amount of viruses is "HIGH", i.e., the triangular fuzzy number $\mathcal{A}_h = (\tau_c - \delta, \tau_c, \tau_c + \delta)$ satisfies $\tau_c - \delta \leq \tau_0$, then the transmission rate $\sigma_k(\tau) = \sigma k$ is a constant function w.r.t the viral load τ . Therefore, the basic reproduction number \mathcal{R}_0 only depends on the model's parameters.

5.2 The sensitivity analysis of the threshold value \mathcal{R}_0

Now, we will discuss how different parameters contribute to the change of the threshold value \mathcal{R}_0 by evaluating the normalized sensitivity indices. According to Nakul et. al. [26], the sensitivity index of a quantity \mathbf{x} depending on a parameter λ can be determined by $\Upsilon_{\lambda}^{\mathbf{x}} = \frac{\partial \mathbf{x}}{\partial \lambda} \times \frac{\lambda}{\mathbf{x}}$. By the definition of the basic reproduction number \mathcal{R}_0 , this quantity depends on some model's parameters such as $r, \sigma(\tau), \mu, \Lambda$ and the parameter of network structure $\frac{\langle k^2 \rangle}{\langle k \rangle}$. Therefore, by direct computations, we obtain

$$\Upsilon^{\mathcal{R}_0}_{\sigma(\tau)} = 1, \qquad \Upsilon^{\mathcal{R}_0}_{\Lambda} = 1, \qquad \Upsilon^{\mathcal{R}_0}_{r} = -\frac{r}{\mu + r}, \qquad \Upsilon^{\mathcal{R}_0}_{\mu} = -\frac{(2\mu + r)}{\mu + r}, \qquad \Upsilon^{\mathcal{R}_0}_{\frac{\langle k^2 \rangle}{\langle k \rangle}} = 1.$$

Remark 5.1. We can see that the threshold value \mathcal{R}_0 is the most sensitive with the natural death rate μ . Furthermore, we can conclude that the increase of the cure rate r will reduce the value of \mathcal{R}_0 . In addition, the nodes with different degrees will get different influences to the value \mathcal{R}_0 . For the fuzzy transmission rate $\sigma(\tau)$, it will experience a 10% increase of the value \mathcal{R}_0 if we increase the parameter σ by a same percentage. Similarly, we can also conclude that the value of the basic reproduction number \mathcal{R}_0 increases with the increase of the structure parameter $\frac{\langle k^2 \rangle}{\langle k \rangle}$, which means that the HFMD could be controlled if the value $\frac{\langle k^2 \rangle}{\langle k \rangle}$ is decreasing, whereas the higher value of $\frac{\langle k^2 \rangle}{\langle k \rangle}$ could follow that more efforts must be done to eliminate the disease on the population, i.e. the controlling of the HFMD becomes more difficult if the parameter $\frac{\langle k^2 \rangle}{\langle k \rangle}$ is increasing. The results of sensitive test can be summarized in Table 3.

No	Parameter	Description	Sensitivity index			
1	$\sigma(\tau)$	The fuzzy transmission rate	+1			
2	r	The cure rate	$-\frac{16}{17}$			
3	μ	The natural death rate	$-\frac{18}{17}$			
4	Λ	The natural birth rate	+1			
5	$\frac{\langle k^2 \rangle}{\langle k \rangle}$	The parameter of network structure	+1			

Table 3: The sensitivity indices of model's parameters

In addition, for convenience, we present the sensitivity of parameters in Figure 2.



Figure 3: The sensitivity indices of the model's parameters

In the following, we discuss the change of the basic reproductive number \mathcal{R}_0 with respect to viral load τ . Let us choose the normalized values of threshold quantities τ_m, τ_0, τ_M by 0.25, 0.65, 1, respectively. Then, the transmission rate σ can be represented as a trapezoidal fuzzy number $\tilde{\sigma} = \sigma(0.25, 0.65, 1, 1)$. In order to dealt with the uncertainty in the network-based epidemic model (1), we will apply the granular approach for fuzzy numbers proposed by Mazandarani et al. [22] to represent the fuzzy transmission rate $\tilde{\sigma}$. The granular approach is developed from the idea of horizontal membership function of Piegat [30]. In this approach, we parametrize a fuzzy number u by using two indices α (level-sets index) and α_u (relative-distance-measure variable, see [22] for more detail) that measures the granule of information. In particular, for a fuzzy number u with respective level-sets $[u]^{\alpha} = [u_{\alpha}^-, u_{\alpha}^+], \alpha \in [0, 1]$, the granular representation of the fuzzy number u is given by

$$u^{gr}(\alpha, \alpha_u) = u_{\alpha}^{-} + \left[u_{\alpha}^{+} - u_{\alpha}^{-}\right] \alpha_u$$

in which $\alpha_u \in [0,1]$. As a consequence, the horizontal membership function (or gr-representation) of the trapezoidal fuzzy number $\tilde{\sigma}$ is given by $\tilde{\sigma}^{gr}(\alpha, \alpha_{\sigma}) = \sigma \left[0.25 + 0.4\alpha + (0.75 - 0.4\alpha)\alpha_{\sigma}\right]$. Then, the relative change of the basic reproduction number \mathcal{R}_0 is given in Figure **2**.



Figure 4: The relative change of the basic reproduction number \mathcal{R}_0 w.r.t. malware load: Fig. (a) $\sigma = 0.1$ and Fig. (b) $\sigma = 0.15$

Figure **1** (b) shows the importance of viral load in the change of \mathcal{R}_0 . If the amount of infectious source is increasing then the basic reproduction number \mathcal{R}_0 also increases from less than 1 to greater than 1. Hence, there has a noticeable change in the stability state of the proposed epidemic model when the viral load varies. Additionally, it experienced that the bifurcation occurs at some values τ . This phenomena will be studied in our next work. A similar result was discussed in [22].

6 Conclusions

This work studied a fractional network-based SIRS epidemic model with fuzzy transmission and saturated treatment function to discuss the malware attacking on the heterogeneous network. In reality, there may occur a scenario that the quantity of infected individuals who need to be treated may exceed the treatment capacity and reach a saturation level. Here, in order to better description for real-world situation, we introduce an epidemic model with a saturated treatment function instead of a linear treatment function. In addition, this work also use linguistic variables and fuzzy membership function to discuss the influence of malware load in the malware infection on the heterogeneous network. Based on the next-generation matrix, we analytically evaluate the basic reproduction number \mathcal{R}_0 , that is an important threshold value to investigate the asymptotic stability of malware-free equilibrium and the presence of endemic equilibrium on the network. We hope that this work will be the first stage to open up some further studies on the network-based epidemic model. In the next study, we are going to consider the optimal quarantine control problem for the network-based epidemic model (II) to evaluate the effect of quarantine treatment for controlling the epidemic disease. In addition, the bifurcation phenomena leading from a malware-free equilibrium to an endemic equilibrium is an important problem in the epidemiology theory. Since the proposed epidemic model considered the treatment function in nonlinear form, namely saturated treatment function, the basic reproduction number cannot describe the necessary disease elimination effort any more, i.e., a stable endemic equilibrium may co-exists with a stable malware-free equilibrium even if $\mathcal{R}_0 < 1$, which means that the backward bifurcation phenomena occurs. This is also an interesting topic we are going to discuss in the next study. On the other hand, the dynamic analysis for the endemic equilibrium \mathbf{E}_* hasn't been detailed discussed on this paper.

7 Appendix

In the following, we briefly recall a framework of fractional calculus, see [3, **L3**] for more details.

Definition 7.1. [3] For each $\beta > 0$ and $[a, b] \subset \mathbb{R}$, let a function $f : [a, b] \to \mathbb{R}$ such that $f \in L^1([a, b], \mathbb{R})$. Then, the Riemann-Liouville fractional integral operator of order β is defined by

$${}_a\mathfrak{I}^{\beta}_tf(t) = \frac{1}{\Gamma(\beta)}\int_a^t (t-s)^{\beta-1}f(s)ds, \quad t\in[a,b].$$

Definition 7.2. [3] Let $m := \lceil \beta \rceil$ be the smallest integer greater than or equal to β . The Caputo fractional derivative of order β of a function $f \in C^m(a, b)$ is defined by

$${}_{a}^{C}\mathfrak{D}_{t}^{\beta}f(t) = \frac{1}{\Gamma(m-\beta)}\int_{a}^{t}(t-s)^{m-\beta-1}f^{(m)}(s)ds.$$

In general, Caputo fractional derivative for a vector-valued function $f = (f_1, f_2, \dots, f_n)^\top$ is defined component-wise by

$${}_{a}^{C}\mathfrak{D}_{t}^{\beta}f(t) = \left({}_{a}^{C}\mathfrak{D}_{t}^{\beta}f_{1}(t), {}_{a}^{C}\mathfrak{D}_{t}^{\beta}f_{2}(t), \dots, {}_{a}^{C}\mathfrak{D}_{t}^{\beta}f_{n}(t)\right)$$

Consider the initial value problem for the following fractional differential equations (FDS)

$${}^{C}_{0}\mathfrak{D}^{\beta}_{t}\mathbf{x}(t) = A\mathbf{x}(t) + f(\mathbf{x}(t)) \qquad t > 0,$$
(10)

subject to the initial conditions

$$\mathbf{x}(0) = \mathbf{x}_0,\tag{11}$$

where $A \in \operatorname{Mat}_{n \times n}(\mathbb{R})$ and $f : \mathbb{R}^n \to \mathbb{R}^n$ is a continuously differentiable function and satisfies Lipschitz condition. According to Corollary 6.9 in [3], it implies the global unique existence of solutions of the initial value problem (\square). (\square). Next, let $\varphi : [0, \infty) \to \mathbb{R}^n$ be a solution of the initial value problem (\square)(\square). Now, we recall from Definition 7.2 in [3] the notions of stability and asymptotic stability of trivial solution of (\square).

Definition 7.3. [3] The trivial solution $\mathbf{x}^* \equiv \overline{0}$ of the FDS (\square) is said to be

• stable if for all $\varepsilon > 0$, there exists $\delta = \delta(\varepsilon) > 0$ such that the solution $\varphi(t, \mathbf{x}_0)$ of the initial value problem (III) (III) satisfies $\|\varphi(t, \mathbf{x}_0)\| < \varepsilon$ for all $t \ge 0$ whenever $\|\mathbf{x}_0\| < \delta$.

• asymptotically stable if it is stable and attractive, i.e., there is a constant $\gamma > 0$ such that $\lim_{t \to \infty} \|\varphi(t, \mathbf{x}_0)\| = 0$ whenever $\|\mathbf{x}_0\| < \gamma$.

Remark 7.4. The trivial solution $\mathbf{x}^* \equiv \overline{0}$ of the FDS (\square) is said to be globally asymptotically stable if its stability does not depend on the initial condition $\mathbf{x}_0 \in \mathbb{R}^n$.

Lemma 7.5. [3] For each $\beta_1, \beta_2 > 0$, we have $\mathbb{E}_{\alpha_1,\alpha_2}(x) = x\mathbb{E}_{\alpha_1,\alpha_1+\alpha_2}(x) - \frac{1}{\Gamma(\alpha_2)}$, where $\mathbb{E}_{\alpha_1,\alpha_2}(z)$ is the Mittag-Leffer functions of two parameters α_1 and α_2 (see [II]).

Lemma 7.6. [12] Let $\mathbf{x} : [0, \infty) \to \mathbb{R}^+$ be an absolutely continuous function on $[0, \infty)$ and $\beta \in (0, 1]$. Then, for each $\mathbf{x}^* \in \mathbb{R}^+$ and t > 0, the following inequality holds

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\left(\mathbf{x}(t)-\mathbf{x}^{*}-\mathbf{x}^{*}\ln(\frac{\mathbf{x}(t)}{\mathbf{x}^{*}})\right) \leq \left(1-\frac{\mathbf{x}^{*}}{\mathbf{x}(t)}\right){}_{0}^{C}\mathfrak{D}_{t}^{\beta}\mathbf{x}(t).$$

Remark 7.7. Let $\Psi : [0, \infty) \to \mathbb{R}$ be a function given by $\Psi(\mathbf{x}) = \mathbf{x} - \mathbf{x}^* - \mathbf{x}^* \ln\left(\frac{\mathbf{x}}{\mathbf{x}^*}\right)$. Then, it is true that the function $\Psi(\mathbf{x})$ is a non-negative function and attains the global minimum at the point $\mathbf{x} = \mathbf{x}^*$.

Lemma 7.8. [28] Assume that $\beta \in (0,1]$ and both the function Φ and its fractional derivative ${}_{0}^{C}\mathfrak{D}_{t}^{\beta}\Phi$ belong to the space C[a,b]. Then we have $\Phi(t) = \Phi(a) + \frac{1}{\Gamma(\beta)}{}_{a}^{C}\mathfrak{D}_{t}^{\beta}\Phi(\xi) (t-a)^{\beta}$, for $a \leq \xi \leq t$ and $t \in [a,b]$.

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Interconnected Takagi-Sugeno fuzzy system and fractional SIRS epidemic model for stabilization of Wireless Sensor Networks

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Abstract

This paper deals with the problems of studying the dynamical behavior of a controlled fractional network-based SIRS epidemic model and establishing an immunization policy based on interconnected fractional Takagi-Sugeno fuzzy systems. Firstly, we introduce a three-compartmental network-based epidemic model with saturated treatment function and fractional-order derivative. Next, we discuss some qualitative properties of the proposed network-based SIRS epidemic model such as the existence of positively invariant set, the backward bifurcation and the asymptotic behavior of solution. Especially, in order to study the asymptotic stability, we establish an epidemiological threshold value \mathfrak{R}_0 , namely basic reproductive ratio, which ensures not only the existence of at least one endemic equilibrium \mathbf{P}_* but also the local asymptotic stability of a malware-free equilibrium \mathbf{P}_0 . As a consequence of theoretical result, the malware-free equilibrium \mathbf{P}_0 is unstable when $\mathfrak{R}_0 > 1$ and hence, the rest of this paper is to address a stabilization problem for the proposed controlled fractional network-based epidemic model and present a novel stabilization criterion with parallel distributed compensation (PDC) controller related to linear matrix inequalities and positive definite matrices. Finally, we illustrate the obtained theoretical results by a computational example.

Keywords: A controlled fractional network-based SIRS epidemic model; Equilibrium points; Nonlinear treatment function; Asymptotic stability; Backward bifurcation; Fractional interconnected Takagi-Sugeno fuzzy system; Fuzzy state-feedback control.

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1. Introduction

It is a fact that, in the age of information technology, many real-life problems, such as electric power systems, nuclear reactors, aerospace systems, economic systems, and process control systems, etc., have become increasingly large in scope and complex in structure, that is the reason why the two past decades have witnessed a dramatic growth of research development on complex heterogeneous network. Now, it is still a promising research area and numerous significant results have continued to appear one after another in variety disciplines of sciences, engineering and real-world processes such as intelligent transportation systems, smart power grids, water and energy distribution, mobile robots, industrial processes, sensor network and computer networks, see, see [1, 5, 11, 18, 22, 29, 38, 41]. The structure of each complex heterogeneous network consists of a certain number of nodes connected by links or edges for data transmission, information gathering, processing and implementation. Nodes in network systems can exchange information through wired and wireless communication channels. Unfortunately, if some malicious objects such as viruses, worms or etc. are attached in transmitted data then these malicious objects may take advantage of data transmission process to be widespread on the network, attack and threaten the safety of network systems. For example, some computer viruses are capable of acquiring personal data from network users such as account passwords, causing serious damage to individuals and corporations. As a consequence, in order to against with this threat of contemporary information society, a lot of treatments have been done such as regularly upgrading the system, promoting monitoring, strengthening security layers,... in which the development of antimalware software is an important way to safeguard the information systems. However, no matter how good the

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measure, there are always its advantages and disadvantages. The problem comes from the fact that no anti-malware program can detect and clear all kinds of malicious objects. Moreover, accompanied by the dramatic development of various network systems, there are more and more types of malicious objects appearing and they seem to be more dangerous and harder to be detected. We must admit that when a new type of malware appears, it takes a while to have a software to handle this malware, i.e., there often has an inevitable lag from the appearance of a new malware to the introduction of anti-malware program targeting this malicious object. The danger here is that during this lag, malicious objects can widely spread in the network that results extreme influences to the safety, security and smooth operation of network systems. In the study and development of anti-malware programs, the first task is to accurately analyze the dynamic properties of malware programs as well as their infection mechanism and then, predict their evolutionary directions. For this aim, many scientists have contributed their work to mathematical modeling the malware program's infection on network systems based on differential or statistical models. This approach seems to be realistic because the rates of disease-causing contacts and the interactions among nodes on network systems are un-similar. And hence, the concept of a complex heterogeneous network is incorporated into epidemic models to describe the effect of contact heterogeneity of the network's nodes in a malware program's attack. In this work, our aim is to investigate the mechanism of malware propagation on complex heterogeneous network based on the use of mean-field reaction-rate differential equations. These systems are often known as network-based epidemic models and the last decade have experienced a noticeable development on the qualitative studies of malware propagation on complex heterogeneous networks governed by network-based epidemic models, see [5, 11, 12, 15, 17, 27] for therein.

Nowadays, fractional calculus and dynamical systems governed by fractional differential equations and fractional partial differential equations have attracted more and more attention by many researchers due to their great applications in various areas of science and engineering such as mechanics and control processes [39, 40], information networks [10, 11, 17], epidemiology [9, 16], economics [13, 44], viscoelastic materials [4, 32, 37], mathematical modeling [6, 25, 26, 28, 31] and etc. These studies have proved that fractional derivative has many advantages in comparison with integer-order derivative. In addition, one of the most simple examples, where the fractional calculus shows its importance, can be observed in the diffusion processes. It is proved that the diffusion process is obtained when the order of fractional derivative is in (0,1). Another impact of fractional derivative can be found in the stability analysis. Indeed, there exist differential equations that are not stable with integer-order derivative, but their fractional versions are stable with fractional derivatives (see [8]). Although the studies of both fractional dynamic systems and network-based epidemic models have an interesting significance and have gained a lot of achievements, there only exists a few works studied the network-based epidemic models with fractional-order derivative. Some of them can be found in [11, 12, 14, 17, 21, 33]. The study of stability and stabilization problem for various types of differential systems is an important branch of applications-oriented mathematics. Here, theory of stability, that has done so for well over 130 years since the 1892 work of A.M. Lyapunov, provides core techniques and effective tools for the analysis of dynamical behaviors of many mathematical model-based processes. If we regard the stability theory plays a key role in the study of dynamical control systems and their applications then the feedback stabilization problem is a core problem of mathematical control theory. Probably the first modern stabilization problem was done in an earlier study of J. Clerk Maxwell for the feedback control problem of a steam engine governor. Along with the popularization of fractional differential equations and fractional partial equations in modeling real-world processes, many qualitative problems related to fractional dynamical systems have been attracted by mathematicians and scientists, in which the stability theory and control problems are two of specified problems. Some recent works, that extensively study on these topics, can be found in [4, 6, 13, 25, 26, 34, 37, 39, 40]. However, it should be noted that the existing results are limited on linear models and the studies of fractional nonlinear differential systems, especially fractional network-based models, are in the first stage of the research and have just gained some initial theoretical results. For example, see [14, 16, 17, 21]. Indeed, due to the structural characteristics, such as high dimension, nonlinear, uncertain interconnections or induced delays [41], the stability analysis of networked-based models can be very challenging. It is well-known that networked-based models consist of several connected subsystems by linear or nonlinear couplings. In this context, the problem of handling nonlinear interconnections can be considered as one of the great challenges in both the feedback stabilization problem and design of controllers for networked-based models, since usually only linear interconnections are considered in some current works, see [5, 14, 19, 30, 48].

Takagi–Sugeno (TS) fuzzy models have been recognized as an effective way to represent nonlinear systems with an arbitrary precision by means of convex combination of local models, which in general are linear time-invariant differential equations [42]. By exploiting the convex structure of TS models, stability analysis and control design conditions can be formulated as convex optimization problems under linear matrix inequality (LMI) constraints [45]. It is well known that exact TS fuzzy models can be obtained to represent nonlinear systems by employing the sector non-linearity approach [43]. As a consequence, the number of local models (or fuzzy rules) exponentially grows with the amount of system nonlinearities related to the number of premise variables. In three past decades, it experienced a dramatic development of the studies on stability analysis and stabilization based on TS fuzzy models. The TS fuzzy model takes a lot of advantages of human knowledge and experience to handle real-world systems with fewer fuzzy rules than other types of fuzzy systems. Hence, it is natural idea to apply TS fuzzy model to deal with fractional-order models [28, 31] or network-based systems [1, 30].

Next, in order to highlight the novelty of this work, we present some comparisons with some other related works, in which we use some following abbreviations: Epidemic model (EM), Takagi-Sugeno fuzzy system (T-S), Fractional-order dynamic model (FO) and Network-based model (NS).

No	Authors/Year	EM	T-S	FO	NS	Keywords
1	Zhang et al. (2004), see [50]		x			Cost-guaranteed optimal control problem; Nonlinear time-delay dynamical system; Parallel distributed compensation (PDC) controller; Linear matrix inequality; Takagi-Sugeno fuzzy model
2	Lin et al. (2007), see [30]		x		x	Large-scale Takagi-Sugeno fuzzy systems; Parallel distributed compensation fuzzy controller; Linear matrix inequalities
3	Li et al. (2014), see [28]		x	x		Fractional order uncertain TS fuzzy model; Robust stability; PDC state feedback control; Fractional order Rössler system; Fractional order uncertain Lorenz system
4	A. Benzaouia and A.E. Hajjaji (2017), see [2]		x	x		Continuous-time fractional positive systems; Takagi-Sugeno fuzzy model; Stabilization; Linear programming; State feedback controller
5	Lu et al. (2020), see [33]	x		x	x	Inter-city networked coupling effects; Fractional-order; SEIHDR epidemic model;
6	Araújo et al. (2021) , see $[1]$		x		x	Distributed saturation control; Network nonlinear systems; Interconnected Takagi-Sugeno fuzzy model
7	H.J. Lee (2022), see [23]	x	x			SIR epidemic model; Positive Takagi-Sugeno fuzzy model; Static output-feedback control; $\mathscr{L}_{\infty} - \mathscr{L}_{\infty}$ disturbance attenuation
8	Dong et al. (2022), see [11]	x		x	x	Energy-Aware Barabási Albert scale-free network model; Fractional SE ₁ E ₂ IQR epidemic model; Equilibria; Globally asymptotic stability; Backward bifurcation
9	Dong et al. (2022), see [12]	x		x	x	Fractional network-based epidemic model; Fuzzy transmission saturated treatment function; Equilibria; Basic reproduction number; Asymptotic stability
10	The proposed work	x	x	x	x	Fractional network-based SIRS epidemic model with control; Equilibria; Nonlinear treatment function; Asymptotic stability; Backward bifurcation; Interconnected Takagi-Sugeno fuzzy system; Fuzzy PDC state-feedback control.

Table 1: THE COMPARISON OF THE PROPOSED MODEL WITH SOME RELATED WORKS

To the best of our knowledge, there isn't any work studied on the Takagi-Sugeno fuzzy system for fractional network-based epidemic model, which deals with both the complex heterogeneity of network system modeled the attack of malware programs on real-world networks and the non-local properties induced from the fractional differential model. It is well-known from Table 1 that with the outbreak of epidemiological studies on network systems in some recent years, it is natural to carry out a detailed research on control problems for fractional network-based epidemic models under Takagi-Sugeno fuzzy systems approach. Motivated by aforesaid, this work is devoted to study a fractional epidemiological model on complex with immunization, namely controlled fractional network-based SIRS epidemic model, and present some qualitative properties of the proposed epidemic model. For more clearly, we highlight the main contributions of this work as follows:

- (i) Study a controlled fractional network-based SIRS epidemic model with saturated treatment function to describe the dynamical behavior of malware program's attack on complex heterogeneous network under immunization control. Here, based on the theory of fractional differential equations and theory of complex network, we propose to study a large-scale differential systems including n fractional mean-field reaction rate equation of the form (4). Furthermore, in order to provide a better description for the scenario that epidemic disease due to malware programs blows up, we consider a nonlinear treatment function instead of linear one.
- (ii) Based on the mathematical formulation (4) of the k^{th} subsystem of the controlled fractional network-based SIRS epidemic model, we analyze the dynamical behaviors of the proposed epidemic model and summarize some important characteristic properties such as positivity, the existence of positively invariant set and asymptotic stability. Especially, a noticeable achievement of this work is the use of next-generation matrix method (see [7]) to evaluate the basic reproductive ratio \Re_0 , that is regarded as an important threshold value in the epidemiological theory. Furthermore, in order to study the effect of model's parameters to \Re_0 , we present some evaluations of the normalized sensitivity indices of parameters.
- (iii) It is well-known that the existence and dynamical behavior of the epidemic model's equilibrium points are cores of the epidemiological study. In this work, we show that the malware-free equilibrium \mathbf{P}_0 of the controlled fractional network-based SIRS epidemic model always exists for all input control \mathbf{u} . Especially, in the case $\mathbf{u} = 0$, we receive a well-known malware-free equilibrium introduced in [12]. Next, Theorem 4.1 points out that the existence of endemic equilibrium \mathbf{P}_* strongly depends on the basic reproductive \mathfrak{R}_0 , but this existence may be not unique.
- (iv) A detailed discussion on the asymptotic stability of the malware-free equilibrium \mathbf{P}_0 is presented in Theorem 4.2 and Theorem 4.3. While we only adjust the basic reproductive ratio \mathfrak{R}_0 less than unity to ensure the local asymptotic stability of the malware-free equilibrium \mathbf{P}_0 , the global asymptotic stability of \mathbf{P}_0 requires a larger threshold value $\mathfrak{R}_0 > \mathfrak{R}_0$ and this value must be less than unity. In this section, in order to explain the phenomena that there may have an endemic equilibrium co-existing with \mathbf{P}_0 even $\mathfrak{R}_0 < 1$, we discuss the condition of backward bifurcation of the proposed model.
- (v) It is an interesting question that how to control the dynamical behavior of the controlled fractional networkbased SIRS epidemic model or in an other word, how to stabilize the unstable malware-free equilibrium \mathbf{P}_0 of the proposed model, which arising from the fields of network models and physiological systems with memory effect. For this aim, we apply the interconnected Takagi-Sugeno fuzzy system approach to establish some sufficient conditions in the linear matrix inequality (LMIs) form for the stabilization of the equilibrium \mathbf{P}_0 . To the best of our knowledge, there isn't any work done with Takagi-Sugeno fuzzy system for network-based differential systems with fractional order derivative.

Finally, the structure of this paper is given as follows:

2. Preliminaries

In this section, we briefly recall from [8, 20, 47] some notions and auxiliary results on fractional dynamical systems and their stability theory, Takagi-Sugeno fuzzy system for network models. First of all, we summarize some notations and basic concepts that will be used throughout this paper.

2.1. Notations

- \mathbb{R}^n_+ denotes for the non-negative orthant of the *n*-dimensional real space \mathbb{R}^n ;
- \mathbb{I}_n is the identity matrix of order n and $\mathbf{0}$ is the zero matrix with appropriate dimensions;
- $A = [a_{ij}]_{m \times n}$ denotes for a real matrix with m rows and n columns and A^{\top} denotes for its transpose;
- A matrix $A = [a_{ij}]_{n \times n}$ is called *positive definite* if $x^{\top}Ax > 0$ for all $x \in \mathbb{R}^n \setminus \{\mathbf{0}\}$;
- ${}_{0}^{C}\mathscr{D}_{t}^{\beta}x(t)$ denotes for the Caputo fractional derivative of order β of a function x(t);
- $\mathbb{E}_{\beta_1,\beta_2}(x)$ denotes for the Mittag-Leffer functions of two parameters β_1 and β_2 ;
- $\arg(\lambda)$ and $|\arg(\lambda)|$ denote for the argument of a complex number λ and its corresponding modulus.

2.2. Fractional calculus

Definition 2.1 ([8]). Let $m := \lceil \beta \rceil$. Then, the Caputo fractional derivative of order β of a function $f \in C^m(0,T)$ is defined by

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}f(t) = \frac{1}{\Gamma(m-\beta)} \int_{0}^{t} (t-s)^{m-\beta-1} f^{(m)}(s) ds.$$

In particular, if f(t) is a continuously differentiable function on [0, T] then

$${}_0^C \mathscr{D}_t^\beta f(t) = \frac{1}{\Gamma(1-\beta)} \int_0^t (t-s)^{-\beta} f'(s) ds.$$

Especially, if $f = (f_1, f_2, \dots, f_n)^{\top}$ is a vector-valued function then the Caputo fractional derivative of f(t) is defined component-wise by ${}_0^C \mathscr{D}_t^{\beta} f(t) = \begin{pmatrix} C \mathscr{D}_t^{\beta} f_1(t), C \mathscr{D}_t^{\beta} f_2(t), \dots, C \mathscr{D}_t^{\beta} f_n(t) \end{pmatrix}^{\top}$.

Proposition 2.1 ([8]). For each $\beta_1, \beta_2 > 0$, we have $\mathbb{E}_{\beta_1,\beta_2}(x) = x\mathbb{E}_{\beta_1,\beta_1+\beta_2}(x) - \frac{1}{\Gamma(\beta_2)}$, where $\mathbb{E}_{\beta_1,\beta_2}(x)$ is the Mittag-Leffer functions of two parameters β_1 and β_2 (see [20] for more details).

Lemma 2.1 ([36]). Assume that $\beta \in (0,1]$ and both the function Φ and its fractional derivative ${}_{0}^{C}\mathscr{D}_{t}^{\beta}\Phi$ belong to the space C[a,b]. Then we have

$$\Phi(t) = \Phi(a) + \frac{1}{\Gamma(\beta)} {}^{C}_{a} \mathscr{D}^{\beta}_{t} \Phi(\xi) (t-a)^{\beta},$$

for $a \leq \xi \leq t$ and $t \in [a, b]$.

Consider an initial value problem (IVP) for the following fractional differential equations (FDS)

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{x}(t) = A\mathbf{x}(t) + f(\mathbf{x}(t)), \qquad t > 0$$
(1)

subject to the initial condition $\mathbf{x}(0) = \mathbf{x}_0$, where $A \in \operatorname{Mat}_{n \times n}(\mathbb{R})$ and $f : \mathbb{R}^n \to \mathbb{R}^n$ is a continuously differentiable function. Let $\varphi : [0, \infty) \to \mathbb{R}^n$ be a solution of the problem (IVP) and recall from Definition 7.2 in [8] the concepts of stability of trivial solution to the FDS (1).

Definition 2.2 ([8]). The trivial solution $\mathbf{x} \equiv \mathbf{0}$ of the FDS (1) is said to be

- stable if for all $\varepsilon > 0$, there exists $\delta = \delta(\varepsilon) > 0$ such that the solution $\varphi(t, \mathbf{x}_0)$ of (IVP) satisfies $\|\varphi(t, \mathbf{x}_0)\| < \varepsilon$ for all $t \ge 0$ whenever $\|\mathbf{x}_0\| < \delta$.
- asymptotically stable if it is stable and attractive, i.e., there exists a $\gamma > 0$ such that $\lim_{t \to \infty} \|\varphi(t, \mathbf{x}_0)\| = 0$ whenever $\|\mathbf{x}_0\| < \gamma$.

In the case $f(\mathbf{x}(t)) \equiv \mathbf{0}$, the stability of the obtained linear fractional differential system is equivalent to the stability of the trivial solution $\mathbf{x} \equiv \mathbf{0}$. Next, we present a necessary and sufficient criteria for the stability of linear fractional differential system

Theorem 2.1 ([8]). The linear fractional differential systems ${}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{x}(t) = A\mathbf{x}(t)$ is

- asymptotically stable if and only if $|\arg(\lambda_i)| > \frac{\beta\pi}{2}$ holds for all eigenvalues λ_i of the matrix A.
- stable if $|\arg(\lambda_i)| \ge \frac{\beta\pi}{2}$ holds for all eigenvalues λ_i of the matrix A and all critical eigenvalues, which satisfy the condition $|\arg(\lambda_i)| = \frac{\beta\pi}{2}$, have geometric multiplicity one.

Definition 2.3 ([26]). A continuous function $\gamma : [0, t) \to [0, \infty)$ is said to belong to \mathscr{K} -class if this function is strictly increasing and $\gamma(0) = 0$.

Next, we recall from [26] an extension of Lyapunov direct method for fractional dynamic systems

Theorem 2.2 ([26]). Let $\mathbf{x} \equiv \mathbf{0}$ be an equilibrium point of the fractional differential system (1). Assume that there exist a Lyapunov function $\mathbf{V}(t, \mathbf{x}(t))$ and some \mathscr{K} -class functions $\gamma_i(\cdot)$ (i = 1, 2, 3) such that

$$\gamma_1(\|\mathbf{x}(t)\|) \le \mathbf{V}(t, \mathbf{x}(t)) \le \gamma_2(\|\mathbf{x}(t)\|)$$

$${}_0^C \mathscr{D}_t^{\beta} \mathbf{V}(t, \mathbf{x}(t)) \le -\gamma_3(\|\mathbf{x}(t)\|).$$

Then, the equilibrium point $\mathbf{x} \equiv \mathbf{0}$ of the fractional differential system (1) is asymptotically stable.

2.3. The interconnected fractionalTakagi-Sugeno fuzzy systems

It is well-known that the qualitative knowledge of many real-world systems can be represented by nonlinear dynamic systems and the Takagi-Sugeno (T-S) fuzzy model is regarded as a very good representation for some certain classes of nonlinear dynamic systems, see [42]. In this work, we consider a continuous-time network-based nonlinear dynamic system which consists of N interconnected fractional nonlinear subsystems with observer:

$$\begin{cases} {}^{C}_{0}\mathscr{D}^{\beta}_{t}\mathbf{x}_{i}(t) &= \mathrm{H}\left(\mathbf{x}_{i}(t),\mathbf{u}_{i}(t)\right) + \sum_{j=1}^{N}\mathrm{H}_{ij}\left(\mathbf{x}_{i}(t),\mathbf{x}_{j}(t)\right) \\ \mathbf{y}_{i}(t) &= \overline{\mathrm{H}}\left(\mathbf{x}_{i}(t)\right), \end{cases}$$

for all i = 1, 2, ..., N, where H, \overline{H} and H_{ij} are smooth nonlinear functions. Assume that by using linearization method or sector nonlinearity method (see [24]), we obtain a large-scale fractional Takagi-Sugeno fuzzy system \mathbf{E} composed of N interconnected subsystems \mathbf{E}_i , and each subsystem \mathbf{E}_i is represented by the following fractional Takagi-Sugeno fuzzy system

"The rule \mathbf{E}_i^p : If $z_{i1}(t)$ is F_{i1}^p and $z_{i2}(t)$ is F_{i2}^p and ... and $z_{iq}(t)$ is F_{iq}^p then

$$\begin{cases} C \mathscr{D}_{t}^{\beta} \mathbf{x}_{i}(t) &= A_{i}^{p} \mathbf{x}_{i}(t) + B_{i}^{p} \mathbf{u}_{i}(t) + \sum_{j=1}^{N} \alpha_{ij}^{p} \mathbf{x}_{j}(t) \\ \mathbf{y}_{i}(t) &= D_{i}^{p} \mathbf{x}_{i}(t), " \end{cases}$$

for all $p = 1, 2, ..., r_i$, where \mathbf{E}_i^p is denoted for the p^{th} rule of the interconnected subsystem \mathbf{E}_i and other terms can be explained as follows:

- A_i^p , B_i^p and D_i^p are constant matrices with appropriate dimensions.
- $\mathbf{x}_i(t) \in \mathbb{R}^{n_i}$ and $\mathbf{u}_i(t) \in \mathbb{R}^{m_i}$ are the state vector and the input vector of the subsystem \mathbf{E}_i , respectively.
- The vector $\mathbf{z}_i(t) = \begin{bmatrix} z_{i1}(t) & z_{i2}(t) & \cdots & z_{iq}(t) \end{bmatrix}^\top$ is a measurable premise variable for the subsystem \mathbf{E}_i , which may be equal to $\mathbf{x}_i(t)$ or a function of $\mathbf{x}_i(t)$.
- The index r_i is the number of fuzzy rules in the subsystem \mathbf{E}_i .
- For each j = 1, 2, ..., q, F_{ij}^p are antecedent fuzzy sets of the rule p and $F_{ij}^p(z_{ij}(t))$ is the grade of membership function of $z_{ij}(t)$ in F_{ij}^p .
- The matrix α_{ij}^p is the interconnected matrix between the subsystem \mathbf{E}_i and \mathbf{E}_j in the rule p.

After that, by applying the standard fuzzy inference method, the subsystem \mathbf{E}_i is equivalent to

$$\begin{cases} {}^{C}_{0}\mathscr{D}^{\beta}_{t}\mathbf{x}_{i}(t) &= \sum_{p=1}^{r_{i}} w_{i}^{p}\left(\mathbf{z}_{i}(t)\right) \left\{ A_{i}^{p}\mathbf{x}_{i}(t) + B_{i}^{p}\mathbf{u}_{i}(t) + \sum_{j=1}^{N} \alpha_{ij}^{p}\mathbf{x}_{j}(t) \right\} \\ \mathbf{y}_{i}(t) &= \sum_{p=1}^{r_{i}} w_{i}^{p}\left(\mathbf{z}_{i}(t)\right) D_{i}^{p}\mathbf{x}_{i}(t), \end{cases}$$

$$(2)$$

where

$$w_i^p(\mathbf{z}_i(t)) = \frac{\varphi_i^p(\mathbf{z}_i(t))}{\sum_{p=1}^{r_i} \varphi_i^p(\mathbf{z}_i(t))}, \qquad \qquad \varphi_i^p(\mathbf{z}_i(t)) = \prod_{j=1}^q F_{ij}^p(z_{ij}(t)). \tag{3}$$

Note that $\varphi_i^p(\mathbf{z}_i(t)) \ge 0$ for each $p = 1, 2, \dots, r_i$ and $w_i^p(\mathbf{z}_i(t))$ satisfying $\sum_{p=1}^{r_i} w_i^p(\mathbf{z}_i(t)) = 1$ can be regarded as the

normalized weights of the IF-THEN rules for all i = 1, 2, ..., N (see [30] for more details).

Next, we recall a necessary lemma that are used for studying the asymptotic stability of interconnected fractionalTakagi-Sugeno fuzzy system:

Lemma 2.2 ([34], Lemma 4). Let $\mathbf{x}(t) \in \mathbb{R}^n$ be a continuously differentiable function and $\mathbf{P} \in Mat_{n \times n}(\mathbb{R})$ be a symmetric positive definite matrix. Then, for each $\beta \in (0, 1]$ and $t \ge 0$, the following inequality holds

$$\frac{1}{2} {}_{0}^{C} \mathscr{D}_{t}^{\beta} \left(\mathbf{x}^{\top}(t) \mathrm{P} \mathbf{x}(t) \right) \leq \mathbf{x}^{\top}(t) \mathrm{P} {}_{0}^{C} \mathscr{D}_{t}^{\beta} \mathbf{x}(t).$$

3. Model Formulation

In this work, we propose to use a three-compartment epidemic model, namely fractional network-based SIRS epidemic model, to study the effect of malware propagation on complex heterogeneous network. The SIRS epidemic model categorizes the whole populations of node into three compartments, which are briefly explained in Figure 1.



Figure 1: The description of a three-compartment epidemic model: Susceptible (S) - Infectious (I) - Recovered (R)

It is known that the traditional epidemic models often consider an over-simplified assumption that all individuals are uniformly mixed and the importance of each node in the malware spreading is similar, that is, each individual has the same probability of contact with an infected one. This makes considered models tractable but not realistic, especially, for epidemic diseases in large populations such as social network or information network. Hence, in the modeling of many real-world epidemic models, it is obviously necessary to take into consideration the effect of contact heterogeneity or node's degree when evaluating the rate of epidemic-causing contacts. Motivated by the work of Pastor-Satorras et al. [38], we classify the total population of nodes into n groups and in the k^{th} -group, we denote by $S_k(t)$, $I_k(t)$ and $R_k(t)$ the densities of susceptible, infectious and recovered nodes of degree k at time t, respectively. In addition, we assume that $N_k(t) = S_k(t) + I_k(t) + R_k(t)$ is the number of nodes with degree k at the time t. The flowchart of malware propagation on the k^{th} -group is given in Figure 2.



Figure 2: The transfer diagram of malware propagation among three compartments: Susceptible (S), Infectious (I) and Recovered (R) on the k^{th} -group

The state transition diagram in Figure 2 illustrates how nodes are shifted from one state to another state during the malware propagation process. In particular, the state change between these three states is governed by the following rules:

• Each node may die at a rate of μ when it depletes its battery power and a new node can join the network at a rate of Λ . The network's recruitment rate Λ into Susceptible state and the network's discard rate μ are assumed to be equal to ensure the balance and continuity of the network, that is, a closed system where nodes eliminated from the system are actively replaced by full-energy new nodes.

- When a susceptible node of degree k gets exposed to malware programs, it transits to Infectious state with a rate $\sigma_k \Phi(t)$, where the parameter σ_k is the transmission rate when susceptible nodes contact with the infection. In addition, under the protection of firewall and anti-virus programs, a susceptible node can be shifted to the state (R) at a time-dependent adjustable rate $\mathbf{u}_k(t)$ and then, we say that this node is immunized. In this work, we regard the immunization as a control strategy.
- In classical epidemic models, the treatment rate of infectious node is assumed to be proportional to the number of this nodes, that is, the treatment function is linear. However, it is a fact that for epidemic models on complex heterogeneous network, anti-virus programs have just a certain maximal capacity for the treatment of a malware infection. Motivated by the work of Li et al. [27], we use the treatment function of the form

$$\varphi(I_k) = \frac{rI_k}{1 + \gamma\Phi}$$

for the treatment of the k^{th} -group, in which r is the cure rate and the parameter γ is used to measure the extent of the effect of infected nodes being delayed for treatment. This treatment function seems more realistic than the linear ones.

• Nodes in the state (R) log out the network with a rate μ due to running out of battery power. On the other hand, because the immunization of anti-virus programs is just temporary, recovered nodes can lose immunity and become susceptible again at a rate of ω .

In perspective of the superiority of non-integer order derivative and in order to describe the complete memory effect of malware spreading processes on complex heterogeneous network, we apply the fractional calculus tool to study a network-based malware spreading model with fractional-order derivative. In particular, we characterize the mechanism of malware propagation on complex heterogeneous network in the k^{th} -group by the following fractional mean-field reaction rate equation:

$$\begin{cases} {}^{C}_{0}\mathscr{D}^{\beta}_{t}S_{k}(t) = \Lambda - \sigma_{k}\Phi(t)S_{k}(t) - (\mu + \mathbf{u}_{k}(t))S_{k}(t) + \omega R_{k}(t) \\ {}^{C}_{0}\mathscr{D}^{\beta}_{t}I_{k}(t) = \sigma_{k}\Phi(t)S_{k}(t) - \mu I_{k}(t) - \frac{rI_{k}(t)}{1 + \gamma\Phi(t)} \\ {}^{C}_{0}\mathscr{D}^{\beta}_{t}R_{k}(t) = \mathbf{u}_{k}(t)S_{k}(t) + \frac{rI_{k}(t)}{1 + \gamma\Phi(t)} - (\mu + \omega)R_{k}(t), \end{cases}$$

$$\tag{4}$$

subject to the initial conditions

$$S_k(0) = S_k^0 > 0, I_k(0) = I_k^0 \ge 0, R_k(0) = R_k^0 \ge 0. (5)$$

where ${}_{0}^{C}\mathscr{D}_{t}^{\beta}(\cdot)$ is the Caputo fractional derivative of order $\beta \in (0, 1]$ of the state functions, the transmission rate σ_{k} satisfies $\sigma_{k} = k\sigma$ and all other model's parameters are assumed to be positive. The function $\Phi(t)$ is the probability that a given link is connected to an infectious node and according to Huang et al. [15], this function is given by the following expression

$$\Phi(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \varphi(k) \mathbb{P}(k) I_k(t),$$

where $\mathbb{P}(k)$ is the probability that a randomly chosen node has degree k, $\varphi(k) = k$ is the spreading ability of a node with degree k and $\langle k \rangle = \sum_{k=1}^{n} k \mathbb{P}(k)$ denotes for the mean degree of the network.

4. The analysis of fractional network-based SIRS epidemic model

4.1. The positively invariant set

For simplicity in representation, we denote

$$\begin{split} \tilde{\mathbf{x}}_{k}(t) &= \begin{bmatrix} S_{k}(t) & I_{k}(t) & R_{k}(t) \end{bmatrix}^{\top} \\ \tilde{\mathbf{x}}(t) &= \begin{bmatrix} \tilde{\mathbf{x}}_{1}(t) & \tilde{\mathbf{x}}_{2}(t) & \cdots & \tilde{\mathbf{x}}_{n}(t) \end{bmatrix}^{\top} = \begin{bmatrix} S_{1}(t) & I_{1}(t) & R_{1}(t) & \cdots & S_{n}(t) & I_{n}(t) & R_{n}(t) \end{bmatrix}^{\top} \\ \Sigma^{+} &= \{ \tilde{\mathbf{x}}(t) \in \mathbb{R}^{3n}_{+} : S_{k}(t) + I_{k}(t) + R_{k}(t) = 1, \ k = \overline{1, n}, \ t \ge 0 \} \\ F_{k}(t, \tilde{\mathbf{x}}(t), \mathbf{u}(t)) &= \begin{bmatrix} \Lambda - \sigma_{k} \Phi(t) S_{k}(t) - (\mu + \mathbf{u}_{k}(t)) S_{k}(t) + \omega R_{k}(t) \\ \sigma_{k} \Phi(t) S_{k}(t) - \mu I_{k}(t) - \frac{r I_{k}(t)}{1 + \gamma \Phi(t)} \\ \mathbf{u}_{k}(t) S_{k}(t) + \frac{r I_{k}(t)}{1 + \gamma \Phi(t)} - (\mu + \omega) R_{k}(t) \end{bmatrix} (k = \overline{1, n}) \\ F(t, \tilde{\mathbf{x}}(t), \mathbf{u}(t)) &= \begin{bmatrix} F_{1}(t, \tilde{\mathbf{x}}(t), \mathbf{u}(t)) & F_{2}(t, \tilde{\mathbf{x}}(t), \mathbf{u}(t)) & \cdots & F_{n}(t, \tilde{\mathbf{x}}(t), \mathbf{u}(t)) \end{bmatrix}^{\top}. \end{split}$$

In addition, this work regards the input control $\mathbf{u}_k(t)$ as the rate of susceptible individuals being immunized per unit of time and define

$$\mathscr{U}_{ad} = \left\{ \mathbf{u}(\cdot) \in \left(L^1[0,T] \right)^n : 0 \le \mathbf{u}_k(t) \le b, \ k = \overline{1,n} \right\} \quad (0 < b < 1),$$

by the admissible control set consisting of Lebesgue measurable functions on the time interval [0, T]. Here, due to the fact that immunization of all entire susceptible individuals at one time is un-realistic, we assume that each input control $\mathbf{u}_k(t)$ is restricted by the condition $0 \leq \mathbf{u}_k(t) \leq b < 1$.

Next, due to the presence of malware programs on complex heterogeneous network and by definition of the probability function $\Phi(t)$, we assume that $\Phi(t) > 0$ for each $t \ge 0$. Thus, from the epidemiological viewpoint, we only need to focus on the existence of positive solution and positively invariant set of the fractional network-based SIRS epidemic model, which are given as follows:

Lemma 4.1. For each input control $u \in \mathscr{U}_{ad}$, Cauchy problem for the fractional network-based SIRS epidemic model always has exactly one positive solution $\tilde{\mathbf{x}}(t)$. In addition, if the initial condition satisfies $\tilde{\mathbf{x}}(0) \in \Sigma^+$ then for all t > 0, the solution $\tilde{\mathbf{x}}(t)$ belongs to Σ^+ .

Proof. Our proof is divided into following steps:

Step 1 (The existence and uniqueness): In order to prove the unique existence of the solution $\tilde{\mathbf{x}}(t)$, we will show that the initial value problem (4) - (5) has a unique solution $\tilde{\mathbf{x}}_k(t)$ for all $t \ge 0$. For this aim, we rewrite the fractional network-based dynamical system (4) as follows:

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}\begin{bmatrix}S_{k}(t)\\I_{k}(t)\\R_{k}(t)\end{bmatrix} = \begin{bmatrix}-\mu - \mathbf{u}_{k}(t) & 0 & \omega\\0 & -\mu & 0\\\mathbf{u}_{k}(t) & 0 & -(\mu + \omega)\end{bmatrix}\begin{bmatrix}S_{k}(t)\\I_{k}(t)\\R_{k}(t)\end{bmatrix} + \begin{bmatrix}\Lambda - \sigma_{k}\Phi(t)S_{k}(t)\\-\frac{rI_{k}(t)}{1+\gamma\Phi(t)}\\\frac{rI_{k}(t)}{1+\gamma\Phi(t)}\end{bmatrix}.$$

Since the input control $\mathbf{u}_k(t)$ is a bounded function for each $k = \overline{1, n}$, we can see that the matrix

$$A(\mathbf{u}_k) = \begin{bmatrix} -\mu - \mathbf{u}_k(t) & 0 & \omega \\ 0 & -\mu & 0 \\ \mathbf{u}_k(t) & 0 & -(\mu + \omega) \end{bmatrix}$$

is a bounded matrix function for all $t \ge 0$ and the norm $||A(\mathbf{u}_k)|| = \max\{\mu + 2b, \mu, \mu + 2\omega\} = \mu + 2\max\{b, \omega\}$. Additionally, we denote $\mathbf{f}(t, \tilde{\mathbf{x}}(t)) = (\mathbf{f}_1(t, \tilde{\mathbf{x}}(t)), \mathbf{f}_2(t, \tilde{\mathbf{x}}(t)), \dots, \mathbf{f}_n(t, \tilde{\mathbf{x}}(t)))^\top$ and for each $k = 1, 2, \dots, n$, we have

$$\mathbf{f}_k(t, \tilde{\mathbf{x}}(t)) = \begin{bmatrix} \Lambda - \sigma_k \Phi(t) S_k(t) & -\frac{r I_k(t)}{1 + \gamma \Phi(t)} & \frac{r I_k(t)}{1 + \gamma \Phi(t)} \end{bmatrix}^\top.$$

Note that for each $\tilde{\mathbf{x}}(t) \in \Sigma^+$ and $k = 1, 2, \ldots, n$, it yields

$$\begin{aligned} \|\mathbf{f}_{k}(t,\tilde{\mathbf{x}}(t))\| &\leq \Lambda + \sigma_{k} \left| \Phi(t) \right| \left| S_{k}(t) \right| + \frac{2r|I_{k}(t)|}{|1 + \gamma \Phi(t)|} \\ &\leq \Lambda + \sigma_{k} \left| S_{k}(t) \right| + 2r \left| I_{k}(t) \right| \\ &\leq \Lambda + \max\left\{ \sigma_{k}, 2r \right\} \left\| \tilde{\mathbf{x}}(t) \right\| \end{aligned}$$

and

$$\|\mathbf{f}(t,\tilde{\mathbf{x}}(t))\| \le n \left(\Lambda + \max_{k} \left\{\sigma_{k}, 2r\right\} \|\tilde{\mathbf{x}}(t)\|\right)$$

Let

$$a_{\mathbf{f}} = \max\left\{n\Lambda, n\left(\mu + 2\max\left\{b,\omega\right\} + \max_{k}\left\{\sigma_{k}, 2r\right\}\right)\right\} = n\left(\mu + 2\max\left\{b,\omega\right\} + \max_{k}\left\{\sigma_{k}, 2r\right\}\right) > 0.$$

Hence, for all $\tilde{\mathbf{x}}(t) \in \Sigma^+$, $\mathbf{u} \in \mathscr{U}_{ad}$ and t > 0, it is true that

$$\begin{aligned} \|\mathbf{F}(t, \tilde{\mathbf{x}}(t), \mathbf{u}(t))\| &\leq n \left[\Lambda + \left(\mu + 2 \max\left\{ b, \omega \right\} + \max_{k} \left\{ \sigma_{k}, 2r \right\} \right) \|\tilde{\mathbf{x}}(t)\| \right] \\ &\leq a_{\mathbf{f}} \left(1 + \|\tilde{\mathbf{x}}(t)\| \right). \end{aligned}$$

On the other hand, for each $\tilde{\mathbf{x}}(t), \overline{\mathbf{x}}(t) \in \Sigma^+$ and $t \ge 0$, we have

$$\begin{aligned} \|\mathbf{f}_{k}(t,\tilde{\mathbf{x}}(t)) - \mathbf{f}_{k}(t,\overline{\mathbf{x}}(t))\| &\leq \sigma_{k} \left| S_{k}(t)\Phi(t) - \overline{S}_{k}(t)\overline{\Phi}(t) \right| + 2r \left| \frac{I_{k}(t)}{1 + \gamma\Phi(t)} - \frac{\overline{I}_{k}(t)}{1 + \gamma\overline{\Phi}(t)} \right| \\ &\leq \sigma_{k} \left(1 + \frac{\sqrt{\langle k^{2} \rangle}}{\langle k \rangle} \right) \|\tilde{\mathbf{x}}(t) - \overline{\mathbf{x}}(t)\| + 2r \left(1 + \frac{2\gamma\sqrt{\langle k^{2} \rangle}}{\langle k \rangle} \right) \|\tilde{\mathbf{x}}(t) - \overline{\mathbf{x}}(t)\| \end{aligned}$$

Denote $L_{\mathbf{f}} = n\left(\mu + 2\max\left\{b,\omega\right\}\right) + \max_{k} \left[\sigma_{k}\left(1 + \frac{\sqrt{\langle k^{2} \rangle}}{\langle k \rangle}\right) + 2r\left(1 + \frac{2\gamma\sqrt{\langle k^{2} \rangle}}{\langle k \rangle}\right)\right] > 0$. Then, for each $\mathbf{u} \in \mathscr{U}_{ad}$, $\tilde{\mathbf{x}}(t), \overline{\mathbf{x}}(t) \in \Sigma^{+}$ and for all t > 0, we receive

$$\left\| \mathbf{F}(t, \tilde{\mathbf{x}}(t)) - \mathbf{F}(t, \overline{\mathbf{x}}(t)) \right\| \le L_{\mathbf{f}} \left\| \tilde{\mathbf{x}}(t) - \overline{\mathbf{x}}(t) \right\|,$$

which means that the function $F(t, \tilde{\mathbf{x}}(t))$ satisfies Lipschitz conditions w.r.t. the state variable $\tilde{\mathbf{x}}(t)$. Finally, by applying Corollary 6.9 in [8], we can conclude that Cauchy problem for the fractional network-based SIRS epidemic model always has exactly one positive solution $\tilde{\mathbf{x}}(t)$ defined for all $t \ge 0$.

Step 2 (The positiveness of solution): For this proof, we assume by contrary that for each $k = \overline{1, n}$, there exists a time $t_0 > 0$ such that

$$\begin{cases} S_k(t_0) = 0\\ S_k(t) > 0 & \text{for all } 0 \le t < t_0\\ S_k(t) < 0 & \text{for some } t > t_0. \end{cases}$$

Then, we consider two following cases:

Case 1: If the function $I_k(t)$ is non-negative for all $t \ge 0$ then we have

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}R_{k}(t) = \mathbf{u}_{k}(t)S_{k}(t) + \frac{rI_{k}(t)}{1+\gamma\Theta(t)} - (\omega+\mu)R_{k}(t) \ge -(\omega+\mu)R_{k}(t).$$

After that, by applying fractional comparison principle (Lemma 10, [25]), we receive

$$R_k(t) \ge R_k(0)\mathbb{E}_\beta\left(-(\omega+\mu)t^\beta\right) \ge 0 \quad \text{for all } t \in [0, t_0].$$

Next, by substituting these above results to the first differential equation of the system (4), we have

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}S_{k}(t)|_{t=t_{0}}=\Lambda+\omega R_{k}(t_{0})>0,$$

which means that there is a sufficiently small constant $\varepsilon_0 > 0$ such that $\frac{\varepsilon_0^{\beta}}{\Gamma(\beta)} {}^C_0 \mathfrak{D}_t^{\beta} S_k(t)|_{t=\xi} \ge 0$ for all $t \in (t_0, t_0 + \varepsilon_0)$. Finally, by using Lemma 2.1 for a = 0 and $t = t_0 + \varepsilon_0$, we receive

$$S_k(t_0 + \varepsilon_0) = S_k(0) + \frac{\varepsilon_0^\beta}{\Gamma(\beta)} {}^C_0 \mathfrak{D}_t^\beta S_k(t)|_{t=\xi} > 0,$$

which contradicts to our assumption.

Case 2: If there exists a time $t_1 > 0$ such that

$$\begin{cases} I_k(t) = 0 & \text{if } t = t_1 \\ I_k(t) > 0 & \text{if } t \in [0, t_1) \\ I_k(t) < 0 & \text{for some } t > t_1. \end{cases}$$

Then our proof is proceeded in two following sub-cases:

<u>Sub-case 1</u>: If $t_1 \ge t_0$ then by doing similar arguments as in Case 1, we can prove that the functions $I_k(t), R_k(t)$ are all non-negative on the interval $[0, t_0]$ and $S_k(t_0 + \varepsilon_0) > 0$, which leads to the contradiction.

<u>Sub-case 2</u>: If $t_1 < t_0$ then we have $S(t_1) > 0$ and $\Theta(t_1) > 0$. Moreover, at the time $t = t_1$, we have

$${}_{0}^{C}\mathfrak{D}_{t}^{\beta}I_{k}(t)|_{t=t_{1}} = \sigma_{k}(\tau)S_{k}(t_{1})\Theta(t_{1}) > 0.$$

Then, we can choose $0 < \varepsilon_1 \ll 1$ such that ${}_0^C \mathfrak{D}_t^\beta S_k(t)|_{t=\overline{\xi}} \ge 0$ with $\overline{\xi} \in [t_1, t_1 + \varepsilon_1]$. Next, by using Lemma 2.1 for a = 0 and $t = t_1 + \varepsilon_1$, we obtain

$$I_k(t_1 + \varepsilon_1) = I_k(0) + \frac{\varepsilon_1^{\beta}}{\Gamma(\beta)} {}^C_0 \mathfrak{D}_t^{\beta} I_k(t)|_{t=\overline{\xi}} > 0.$$

This leads to a contradiction with our assumption. Therefore, we can conclude that the function $S_k(t) > 0$ is always positive for all $t \ge 0$. As a consequence, by doing similar arguments, we can also prove that the functions $I_k(t)$ and $R_k(t)$ are all non-negative for all $t \ge 0$ and $k = \overline{1, n}$.

Step 3 (The positively invariant set): By summing up all fractional differential equations of the system (4), we receive the following fractional differential equation

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}N_{k}(t) = \Lambda - \mu N_{k}(t).$$

By applying the result of Example 4.9 in [20], we receive

$$N_k(t) = N_k(0)\mathbb{E}_{\beta}(-\mu t^{\beta}) + \Lambda t^{\beta}\mathbb{E}_{\beta,\beta+1}(-\mu t^{\beta}) = N_k(0)\mathbb{E}_{\beta}(-\mu t^{\beta}) + \frac{\Lambda}{\mu}\left[1 - \mathbb{E}_{\beta}(-\mu t^{\beta})\right].$$

Here, we apply Theorem 4.2. in [8] with $n_1 = \beta$, $n_2 = 1$ and $x = -\mu t^{\beta}$. Then, by the assumption that $\tilde{\mathbf{x}}(0) \in \Sigma^+$, we immediately obtain

$$N_k(t) = \frac{\Lambda}{\mu} \mathbb{E}_{\beta}(-\mu t^{\beta}) + \frac{\Lambda}{\mu} \left[1 - \mathbb{E}_{\beta}(-\mu t^{\beta}) \right] = \frac{\Lambda}{\mu} = 1,$$

since the recruitment rate and discard rate are assumed to be equal. This means the set Σ^+ is a positively invariant set for the fractional network-based SIRS epidemic model.

4.2. The basic reproductive ratio \mathfrak{R}_0 and the existences of equilibrium states

In the theory of epidemiology, one of essential problems in many preceding studies is to find equilibrium states of the proposed epidemic model including malware-free equilibrium \mathbf{P}_0 and endemic equilibrium \mathbf{P}_* . Beside this, we also focus on evaluating an important threshold value of the epidemiological theory, namely basic reproductive ratio \mathfrak{R}_0 . This value plays a key role in not only the existence of endemic equilibrium but also the asymptotic behavior of the model. In order to find the equilibrium states, we solve the following algebraic system

$$\begin{cases} \Lambda - \sigma_k \Phi S_k - (\mu + \mathbf{u}_k) S_k + \omega R_k &= 0\\ \sigma_k \Phi S_k - \mu I_k - \frac{r I_k}{1 + \gamma \Phi} &= 0\\ \mathbf{u}_k S_k + \frac{r I_k}{1 + \gamma \Phi} - (\mu + \omega) R_k &= 0. \end{cases}$$
(6)

If the absence of malware programs on the network happens, the fractional network-based SIRS epidemic model admits a unique malware-free equilibrium $\mathbf{P}_0 = \left(S_1^0, I_1^0, R_1^0, \dots, S_n^0, I_n^0, R_n^0\right)$ given by

$$\mathbf{P}_{0} = \underbrace{\left(\frac{\mu+\omega}{\mu+\omega+\mathbf{u}_{1}}, 0, \frac{\mathbf{u}_{1}}{\mu+\omega+\mathbf{u}_{1}}, \dots, \frac{\mu+\omega}{\mu+\omega+\mathbf{u}_{n}}, 0, \frac{\mathbf{u}_{n}}{\mu+\omega+\mathbf{u}_{n}}\right)}_{3n},$$

while if malware programs persist on complex heterogeneous network, the proposed epidemic model has at least one endemic equilibrium \mathbf{P}_* under some certain conditions related to the basic reproductive ratio \mathfrak{R}_0 . Next, in order to formulate the basic reproductive ratio \mathfrak{R}_0 , we will apply the next-generation matrix method proposed by Diekmann et al. [7] with the following facts:

- (i) There is only a unique compartment causing the malware spreading process on the network, that is the compartment of infectious nodes (I).
- (ii) The state change from the compartment (I) to other compartments is only regarded as the transition of infected individuals through the various compartments.

Therefore, according to next-generation matrix method, the gain term and loss term of the fractional networkbased SIRS epidemic model are defined by $\sigma_k \Phi(t) S_k(t)$ and $\mu I_k(t) + \frac{rI_k(t)}{1 + \gamma \Phi(t)}$, respectively. Next, for simplicity in representations and computations, we denote $a_k =$ for each $k = \overline{1, n}$. Denote \mathcal{F} and \mathcal{V} by the rate matrix of new infection's appearance and the transition matrix of infected compartments at \mathbf{P}_0 , respectively. Then, by some computations, we receive $\mathcal{V}^{-1} = \frac{1}{\mu + r} \mathbb{I}_n$ and \mathcal{F} is a square matrix of order n given by

$$\mathcal{F} = \frac{\sigma(\mu + \omega)}{\langle k \rangle} \begin{bmatrix} \frac{1}{\mu + \omega + \mathbf{u}_1} \\ \frac{2}{\mu + \omega + \mathbf{u}_2} \\ \vdots \\ \frac{n}{\mu + \omega + \mathbf{u}_n} \end{bmatrix} \begin{bmatrix} \mathbb{P}(1) & 2\mathbb{P}(2) & \cdots & n\mathbb{P}(n) \end{bmatrix}$$

According to the next-generation matrix method, the basic reproductive ratio \mathfrak{R}_0 is known as the largest eigenvalue of the matrix \mathcal{FV}^{-1} , that is,

$$\Re_{0} = \frac{\sigma(\mu+\omega)}{(\mu+r)\langle k\rangle} \sum_{k=1}^{n} \frac{k^{2} \mathbb{P}(k)}{(\mu+\omega+\mathbf{u}_{k})} = \frac{\sigma(\mu+\omega)\langle k^{2}\mathbf{u}\rangle}{(\mu+r)\langle k\rangle},\tag{7}$$

in which $\langle k^2 u \rangle = \sum_{k=1}^n \frac{k^2 \mathbb{P}(k)}{(\mu + \omega + \mathbf{u}_k)}.$

Remark 4.1. From the formula (7), we can conclude that

- The recruitment rate Λ doesn't have any effect to the value of basic reproductive ratio \mathfrak{R}_0 while according to the formula of \mathfrak{R}_0 , it will be experienced a considerable change in the value of basic reproductive ratio \mathfrak{R}_0 if we adjust the control treatment \mathbf{u}_k . In particular, the stronger control input we carry out, the smaller basic reproductive ratio we get.
- The basic reproductive ratio \Re_0 depends on not only the model's parameters but also the network structure, that means the network heterogeneity can make malware programs easier to spread on the network.

Indeed, in order to discuss how parameters affect to the basic reproductive ratio \mathfrak{R}_0 , we evaluate the normalized sensitivity indices of parameters to the threshold value \mathfrak{R}_0 by applying the method of Nakul et al. [35]. Here, for simplicity, we assume that the input control is constant, i.e., $\mathbf{u}_k = \overline{\mathbf{u}} := \text{const}$ for all $k = \overline{1, n}$. As a result, the basic reproductive ratio \mathfrak{R}_0 becomes

$$\Re_{0} = \frac{\sigma(\mu + \omega) \langle k^{2} \rangle}{(\mu + r) \left(\mu + \omega + \overline{\mathbf{u}}\right) \langle k \rangle}.$$

Then, the sensitivity index of a quantity \mathbf{X} depending on a parameter λ is determined by $\Upsilon_{\lambda}^{\mathbf{X}} = \frac{\partial \mathbf{X}}{\partial \lambda} \times \frac{\lambda}{\mathbf{X}}$. Then, we receive some following sensitivity indices

$$\begin{split} \Upsilon^{\mathfrak{R}_{0}}_{\sigma} &= 1, \qquad \Upsilon^{\mathfrak{R}_{0}}_{r} = 1, \qquad \Upsilon^{\mathfrak{R}_{0}}_{r} = -\frac{r}{\mu+r}, \qquad \Upsilon^{\mathfrak{R}_{0}}_{\overline{\mathbf{u}}} = -\frac{\overline{\mathbf{u}}}{\mu+\omega+\overline{\mathbf{u}}}, \\ \Upsilon^{\mathfrak{R}_{0}}_{\omega} &= \frac{\omega \overline{\mathbf{u}}}{(\mu+\omega)\left(\mu+\omega+\overline{\mathbf{u}}\right)}, \qquad \qquad \Upsilon^{\mathfrak{R}_{0}}_{\mu} = \frac{\mu}{\mu+\omega} \left(1 - \frac{\mu+\omega}{\mu+\omega+\overline{\mathbf{u}}} - \frac{\mu+\omega}{\mu+r}\right). \end{split}$$

Remark 4.2. Due to the sensitivity index of the basic reproductive ratio \mathfrak{R}_0 w.r.t the immunization control \mathbf{u}_k is negative, we can conclude that the threshold value \mathfrak{R}_0 will be controlled by increasing the control input. However, it can be easily checked that the modulus of both $\Upsilon_{\mathbf{u}_k}^{\mathfrak{R}_0}$ and $\Upsilon_r^{\mathfrak{R}_0}$ are less than the unity. This means that there is a need to combine various actions for the better controlling of malware spread. In addition, since σ is a parameter characterized for the malware spread due to infectious-contact, the basic reproductive ratio \mathfrak{R}_0 is strongly dependent on the transmission rate σ . Indeed, the sensitive index $\Upsilon_{\sigma}^{\mathfrak{R}_0}$ claims that how much percentage the parameter σ increases, the basic reproductive ratio experienced a increase of the same percentage. Furthermore, the network structure's parameter $\frac{\langle k^2 \rangle}{\langle k \rangle}$ is also one of the most sensitive parameters and its increase by 10% will leads to the decrease of \mathfrak{R}_0 with a same percentage, which means that the higher value of $\frac{\langle k^2 \rangle}{\langle k \rangle}$ could follow that more efforts must be done to eliminate malware programs on complex heterogeneous network.

Next, we investigate the existence of endemic equilibrium \mathbf{P}_* associated with the basic reproductive ratio \mathfrak{R}_0 . For this aim, we denote

$$\mathbf{P}_* = (S_1^*, I_1^*, R_1^*, \dots, S_n^*, I_n^*, R_n^*)$$

Then, the necessary condition for the existence of endemic equilibrium \mathbf{P}_* is given in following theorem:

Theorem 4.1. If the basic reproductive ratio $\Re_0 > 1$ then the fractional network-based SIRS epidemic model always has at least one endemic equilibrium $\mathbf{P}_* = (S_1^*, I_1^*, R_1^*, \dots, S_n^*, I_n^*, R_n^*)$ defined by

$$\begin{split} S_k^* &= \frac{1}{\sigma_k \Phi^*} \left(\mu + \frac{r}{1 + \gamma \Phi^*} \right) I_k^*, \\ R_k^* &= \frac{1}{\mu + \omega} \left[\frac{r}{1 + \gamma \Phi^*} + \frac{\mathbf{u}_k}{\sigma_k \Phi^*} \left(\mu + \frac{r}{1 + \gamma \Phi^*} \right) \right] I_k^*, \\ I_k^* &= \frac{\sigma_k \Phi^*}{\left\{ \mu + \frac{r}{1 + \gamma \Phi^*} + \sigma_k \Phi^* + \frac{\sigma_k \Phi^*}{\mu + \omega} \left[\frac{r}{1 + \gamma \Phi^*} + \frac{\mathbf{u}_k}{\sigma_k \Phi^*} \left(\mu + \frac{r}{1 + \gamma \Phi^*} \right) \right] \right\}. \end{split}$$

Proof. It is well-known that the endemic equilibrium $\mathbf{P}_* = (S_1^*, I_1^*, R_1^*, \dots, S_n^*, I_n^*, R_n^*)$ of the fractional networkbased SIRS epidemic model is a solution of the system (6). Thus, by substituting consecutively the variables S_k and R_k by I_k , we directly get that

$$S_{k} = \frac{1}{\sigma_{k}\Phi} \left(\mu + \frac{r}{1+\gamma\Phi}\right) I_{k}$$
$$R_{k} = \frac{1}{\mu+\omega} \left[\frac{r}{1+\gamma\Phi} + \frac{\mathbf{u}_{k}}{\sigma_{k}\Phi} \left(\mu + \frac{r}{1+\gamma\Phi}\right)\right] I_{k},$$

for each $k = \overline{1, n}$. Then, by summing up side by side of the system (6), we receive

$$\mu \left(S_k + I_k + R_k \right) = \Lambda,$$

and hence, we immediately obtain

$$I_k = \frac{\sigma_k \Phi}{\left\{\mu + \frac{r}{1 + \gamma \Phi} + \sigma_k \Phi + \frac{\sigma_k \Phi}{\mu + \omega} \left[\frac{r}{1 + \gamma \Phi} + \frac{\mathbf{u}_k}{\sigma_k \Phi} \left(\mu + \frac{r}{1 + \gamma \Phi}\right)\right]\right\}}$$

Next, by definition of the probability function $\Phi(t)$, we obtain the following self-consistency equation

$$\Phi = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{k \mathbb{P}(k) \sigma_k \Phi}{\left\{ \mu + \frac{r}{1+\gamma\Phi} + \sigma_k \Phi + \frac{\sigma_k \Phi}{\mu+\omega} \left[\frac{r}{1+\gamma\Phi} + \frac{\mathbf{u}_k}{\sigma_k \Phi} \left(\mu + d + \frac{r}{1+\gamma\Phi} \right) \right] \right\}},\tag{8}$$

which is equivalent to

$$\Phi\left(\frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{k\mathbb{P}(k)\sigma_{k}}{\left\{\mu + \frac{r}{1+\gamma\Phi} + \sigma_{k}\Phi + \frac{\sigma_{k}\Phi}{\mu+\omega} \left[\frac{r}{1+\gamma\Phi} + \frac{\mathbf{u}_{k}}{\sigma_{k}\Phi} \left(\mu + \frac{r}{1+\gamma\Phi}\right)\right]\right\}} - 1\right) = 0.$$

Here, we can see that the self-consistency equation (8) is a nonlinear equation w.r.t the variable Φ and if the equation (8) has a non-trivial solution $\Phi \in (0, 1)$, the fractional network-based SIRS epidemic model admits an endemic equilibrium \mathbf{P}_* . Now, our aim is to determine conditions for the existence of at least one non-trivial solution $\Phi \in (0, 1)$ of the equation (8). For this aim, we denote

$$\mathscr{F}(\Phi) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{k \mathbb{P}(k) \sigma_k \Phi}{\left\{ \mu + \frac{r}{1+\gamma\Phi} + \sigma_k \Phi + \frac{\sigma_k \Phi}{\mu+\omega} \left[\frac{r}{1+\gamma\Phi} + \frac{\mathbf{u}_k}{\sigma_k \Phi} \left(\mu + \frac{r}{1+\gamma\Phi} \right) \right] \right\}}.$$

Then, a solution $\Phi^* \in (0,1)$ of the equation $\mathscr{F}(\Phi) - 1 = 0$ is also a non-trivial solution of the equation (8). In addition, it should be noted that

- The function $\mathscr{F}(\Phi)$ is continuous in [0, 1] and differentiable on (0, 1).
- At the point $\Phi = 1$, we have

$$\mathscr{F}(1) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{k \mathbb{P}(k) \sigma_{k}}{\left\{ \mu + \frac{r}{1+\gamma} + \sigma_{k} + \frac{\sigma_{k}}{\mu+\omega} \left[\frac{r}{1+\gamma} + \frac{\mathbf{u}_{k}}{\sigma_{k}} \left(\mu + \frac{r}{1+\gamma} \right) \right] \right\}} < 1.$$

• At the point $\Phi = 0$, one gets

$$\mathscr{F}(0) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^2 \mathbb{P}(k) \sigma(\mu + \omega)}{(\mu + r) (\mu + \omega + \mathbf{u}_k)} = \mathfrak{R}_0$$

Therefore, if the basic reproductive ratio $\Re_0 > 1$ then it follows that $\mathscr{F}(0) > 1$. By virtue of intermediate value theorem, we can conclude that the equation $\mathscr{F}(\Phi) - 1 = 0$ has at least one solution $\Phi^* \in (0, 1)$, that solves an endemic equilibrium \mathbf{P}_* of the fractional network-based SIRS epidemic model.

Remark 4.3. Note that the condition $\Re_0 > 1$ only ensures the existence of at least one endemic equilibrium \mathbf{P}_* but does not imply the uniqueness of this equilibrium. Furthermore, even if the condition $\Re_0 < 1$ is satisfied, we still can't conclude that the malware programs are completely absence on complex heterogeneous network, that is, the requirement $\Re_0 < 1$ is only the necessary but not sufficient for the malware elimination. Now, we will find a threshold value that ensures the non-existence of any endemic equilibrium \mathbf{P}_* . For this aim, denote

$$\overline{\mathfrak{R}}_{0} = \frac{\sigma(\mu+\omega)}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^{2} \mathbb{P}(k)}{(\mu+\omega+v_{k}) \left(\mu+\frac{r}{1+\gamma}\right)} = \frac{\sigma(\mu+\omega) \langle k^{2} u \rangle}{\left(\mu+\frac{r}{1+\gamma}\right) \langle k \rangle}$$

We can see that the threshold value $\overline{\mathfrak{R}}_0$ satisfies $\overline{\mathfrak{R}}_0 > \mathfrak{R}_0$. In addition, for each $\Phi \in (0,1)$, we have

$$\begin{aligned} \mathscr{F}(\Phi) &= \frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{k \mathbb{P}(k) \sigma_{k}}{\left\{ \mu + \frac{r}{1+\gamma\Phi} + \sigma_{k}\Phi + \frac{\sigma_{k}\Phi}{\mu+\omega} \left[\frac{r}{1+\gamma\Phi} + \frac{\mathbf{u}_{k}}{\sigma_{k}\Phi} \left(\mu + \frac{r}{1+\gamma\Phi} \right) \right] \right\}} \\ &< \frac{\sigma}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^{2} \mathbb{P}(k)}{\mu + \frac{r}{1+\gamma\Phi} + \frac{\mathbf{u}_{k}}{\mu+\omega} \left(\mu + \frac{r}{1+\gamma\Phi} \right)} \\ &< \overline{\mathfrak{R}}_{0}. \end{aligned}$$

As a result, if $\overline{\mathfrak{R}}_0 < 1$ then we can conclude that $\mathscr{F}(\Phi) < 1$ for all $\Phi \in [0, 1]$ and hence, the equation $\mathscr{F}(\Phi) - 1 = 0$ has no solution $\Phi \in [0, 1]$, that is, there isn't any endemic equilibrium \mathbf{P}_* when $\overline{\mathfrak{R}}_0 < 1$. Therefore, when the number of infectious nodes is large and the capacity of treatment is reached, the condition $\mathfrak{R}_0 < 1$ is not enough for the elimination of malware programs.

4.3. The asymptotic stability of malware-free equilibrium \mathbf{P}_0

In the following, based on linearization method for fractional differential systems (see [6]), we discuss the relationship between basic reproductive ratio \Re_0 and the local asymptotic stability of malware-free equilibrium \mathbf{P}_0 .

Theorem 4.2. The following assertions are fulfilled

- (i) If $\mathfrak{R}_0 > 1$ then the malware-free equilibrium \mathbf{P}_0 is unstable.
- (ii) If $\mathfrak{R}_0 < 1$ then the malware-free equilibrium \mathbf{P}_0 is locally asymptotically stable.

Proof. According to the stability theory of fractional differential systems, the local asymptotic stability of malwarefree equilibrium \mathbf{P}_0 depends on the modulus of $\arg(\lambda_j)$, where the value λ_j is an eigenvalue of Jacobi matrix $J(\mathbf{P}_0)$ at the point \mathbf{P}_0 , defined by

$$\mathbf{J}(\mathbf{P}_0) = \begin{bmatrix} \mathbf{J}_{11} & \mathbf{J}_{12} & \cdots & \mathbf{J}_{1n} \\ \mathbf{J}_{21} & \mathbf{J}_{22} & \cdots & \mathbf{J}_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{J}_{n1} & \mathbf{J}_{n2} & \cdots & \mathbf{J}_{nn} \end{bmatrix}.$$

Here, for each $k, j = \overline{1, n}$, the matrices J_{kk} and J_{kj} are square matrices of order 3 given by

$$\mathbf{J}_{kk} = \begin{bmatrix} -(\mu + \mathbf{u}_k) & -\frac{\sigma_k S_k^0 k \mathbb{P}(k)}{\langle k \rangle} & \omega \\ 0 & \frac{\sigma_k S_k^0 k \mathbb{P}(k)}{\langle k \rangle} - (\mu + r) & 0 \\ \mathbf{u}_k & r & -(\mu + \omega) \end{bmatrix}, \qquad \mathbf{J}_{kj} = \begin{bmatrix} 0 & -\frac{\sigma_k S_k^0 j \mathbb{P}(j)}{\langle k \rangle} & 0 \\ 0 & \frac{\sigma_k S_k^0 j \mathbb{P}(j)}{\langle k \rangle} & 0 \\ 0 & 0 & 0 \end{bmatrix}.$$

Therefore, by applying the mathematical induction principle, we immediately get that the characteristic polynomial with respect to Jacobian matrix $J(\mathbf{P}_0)$ is given by

$$\det\left(\overline{\lambda}\mathbb{I}_{3n} - \mathcal{J}(\mathbf{P}_{0})\right) = \left(\overline{\lambda} + \mu\right)^{n} \left[\prod_{k=1}^{n} \left(\overline{\lambda} + (\mu + \omega + \mathbf{u}_{k})\right)\right] \left(\overline{\lambda} + (\mu + r)\right)^{n-1} \times \left[\overline{\lambda} + (\mu + r) - \frac{\sigma(\mu + \omega)}{\langle k \rangle} \sum_{k=1}^{n} \frac{k^{2}\mathbb{P}(k)}{\mu + \omega + \mathbf{u}_{k}}\right]$$
$$= \left(\overline{\lambda} + \mu\right)^{n} \left[\prod_{k=1}^{n} \left(\overline{\lambda} + (\mu + \omega + \mathbf{u}_{k})\right)\right] \left(\overline{\lambda} + (\mu + r)\right)^{n-1} \left[\overline{\lambda} + (\mu + r)(1 - \mathfrak{R}_{0})\right].$$

Therefore, we directly get that the characteristic equation det $(\overline{\lambda}\mathbb{I}_{3n} - J(\mathbf{P}_0)) = 0$ admits *n* roots consisting of a negative solution $\overline{\lambda}_{11} = -\mu$ with multiplicity *n*, a negative solution $\overline{\lambda}_{21} = -(\mu + r)$ with multiplicity *n* - 1 and *n* negative solutions $\overline{\lambda}_{3k} = -(\mu + \omega + \mathbf{u}_k)$ for each $k = \overline{1, n}$. According to Theorem 7.20 in [8], the malware-free equilibrium \mathbf{P}_0 is locally asymptotically stable if and only if

$$\left|\arg\left(\overline{\lambda}_{j}\right)\right| > \frac{\beta\pi}{2}$$
 for all $j = \overline{1, 3n}$.

Indeed, since Jacobian matrix $J(\mathbf{P}_0)$ has 3n-1 negative real eigenvalues $\overline{\lambda}_{11} = -\mu$, $\overline{\lambda}_{21} = -(\mu+r)$ and $\overline{\lambda}_{3k} = -(\mu + \omega + \mathbf{u}_k)$, we immediately get that their arguments are $|\arg(\overline{\lambda}_j)| = \pi > \frac{\beta\pi}{2}$ for all $j = \overline{1, 3n-1}$. As a consequence, the local asymptotic stability of \mathbf{P}_0 only depends on the last eigenvalue

$$\overline{\lambda}_{41} = -(\mu + r)(1 - \Re_0)$$

Finally, we can conclude that

- If $\mathfrak{R}_0 > 1$ then it implies that the eigenvalue $\overline{\lambda}_{41}$ is a positive real value and hence, its argument is 0. Hence, the malware-free equilibrium \mathbf{P}_0 is unstable if $\mathfrak{R}_0 > 1$.
- If $\mathfrak{R}_0 < 1$ then it implies that the eigenvalue $\overline{\lambda}_{41}$ is negative and hence, its argument is $\pi > \frac{\beta \pi}{2}$. Hence, the malware-free equilibrium \mathbf{P}_0 is locally asymptotically stable if $\mathfrak{R}_0 < 1$.

According to Remark 4.3, note that the condition $\Re_0 < 1$ is only necessary but not sufficient for the malware elimination on complex heterogeneous network. In the following, we discuss the conditions for which the malware-free equilibrium \mathbf{P}_0 is globally attractive, i.e., the malware spread fades out.

Theorem 4.3. Denote

$$\tilde{\mathfrak{R}}_0 = \frac{\sigma(\mu + \omega) \langle k^2 \mathbf{u} \rangle}{\mu \langle k \rangle}$$

Then, if the threshold value $\overline{\mathfrak{R}}_0 < 1$, the malware-free equilibrium \mathbf{P}_0 is globally asymptotically stable.

Proof. Let $(S_1(t), I_1(t), R_1(t), \ldots, S_n(t), I_n(t), R_n(t))$ be a non-negative solution of the fractional network-based SIRS epidemic model satisfying the initial condition (5). Since the set Σ^+ is a positively invariant set of the proposed network-based epidemic model, we directly get that

$$\begin{cases} 0 < S_k(t) \le 1 \\ R_k(t) \le 1 - S_k(t) - I_k(t) \end{cases}$$

for each $k = \overline{1, n}$ and $t \ge 0$. Hence, from the first fractional differential equation of (6), it implies that

$$C_{0}^{C} \mathscr{D}_{t}^{\beta} S_{k}(t) = \Lambda - \sigma_{k} \Phi(t) S_{k}(t) - (\mu + \mathbf{u}_{k}) S_{k}(t) + \omega R_{k}(t)$$

$$\leq \Lambda - (\mu + \mathbf{u}_{k}) S_{k}(t) + \omega (1 - S_{k}(t) - I_{k}(t))$$

$$\leq (\mu + \omega) - (\mu + \omega + \mathbf{u}_{k}) S_{k}(t).$$

Consider the following auxiliary system

$${}_{0}^{C} \mathscr{D}_{t}^{\beta} \overline{S}_{k}(t) = (\mu + \omega) - (\mu + \omega + \mathbf{u}_{k}) \overline{S}_{k}(t).$$

This fractional differential system admits a unique positive equilibrium $S_k^0 = \frac{\mu + \omega}{\mu + \omega + \mathbf{u}_k}$, which is globally asymptotically stable. Then, by using fractional comparison principle, it follows that for any $\varepsilon > 0$, there exists a sufficiently large time T_0 such that the inequality $S_k(t) \leq S_k^0 + \varepsilon$ holds for all $t > T_0$. Then, for each $k = \overline{1, n}$ and for all $t > T_0$, the non-negativeness of the function $I_k(t)$ implies that

$$C_{0}^{C} \mathscr{D}_{t}^{\beta} I_{k}(t) = \sigma_{k} \Phi(t) S_{k}(t) - \mu I_{k}(t) - \frac{r I_{k}(t)}{1 + \gamma \Phi(t)}$$
$$\leq \sigma_{k} \left(S_{k}^{0} + \varepsilon \right) \Phi(t) - \mu I_{k}(t).$$

Next, by definition of the probability $\Phi(t)$, we directly get that

$$\begin{split} {}^{C}_{0}\mathscr{D}^{\beta}_{t}\Phi(t) &\leq \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k \mathbb{P}(k) \left[\sigma_{k} \left(S_{k}^{0} + \varepsilon \right) \Phi(t) - \mu I_{k}(t) \right] \\ &= \left[\frac{1}{\langle k \rangle} \sum_{k=1}^{n} k \mathbb{P}(k) \sigma_{k} \left(\frac{\mu + \omega}{\mu + \omega + \mathbf{u}_{k}} + \varepsilon \right) \right] \Phi(t) - \mu \Phi(t) \\ &= \mu \Phi(t) \left[\tilde{\mathfrak{R}}_{0} - 1 + \frac{\varepsilon \sigma \langle k^{2} \rangle}{\mu \langle k \rangle} \right], \end{split}$$

in which $\langle k^2 \rangle = \frac{1}{\langle k \rangle} \sum_{k=1}^n k^2 \mathbb{P}(k)$ represents for the second origin moment of node's degree. Then, the assumption $\tilde{\mathfrak{R}}_0 < 1$ indicates that we can choose $\varepsilon > 0$ sufficiently small such that

$$\tilde{\mathfrak{R}}_0 + \frac{\varepsilon \sigma \langle k^2 \rangle}{\mu \langle k \rangle} < 1.$$

In addition, due to the assumption that malware programs are present on the network, it is true that $\Phi(0) > 0$ and hence, by virtue of fractional comparison theorem, we receive

$$0 \le \Phi(t) \le \Phi(0) \mathbb{E}_{\beta} \left(\mu \left(\tilde{\mathfrak{R}}_{0} - 1 + \frac{\varepsilon \sigma \langle k^{2} \rangle}{\mu \langle k \rangle} \right) t^{\beta} \right), \qquad t \ge 0,$$

which follows that $\lim_{t\to\infty} \Phi(t) = 0$. Based on the definition of $\Phi(t)$, we also have $\lim_{t\to\infty} I_k(t) = 0$ for all $k = \overline{1, n}$. Thus, for any $\varepsilon > 0$, there exists a sufficiently large $T_1 > 0$ such that the function $I_k(t) < \varepsilon$ for all $t > T_1$. As a result, we receive

$$\Phi(t) = \frac{1}{\langle k \rangle} \sum_{k=1}^{n} k \mathbb{P}(k) I_k(t) < \frac{\varepsilon}{\langle k \rangle} \sum_{k=1}^{n} k \mathbb{P}(k) = \varepsilon \quad \text{for all } t > T_1.$$

Hence, for all $t > T_1$, we have

$$\begin{split} & \stackrel{C}{_{0}}\mathscr{D}_{t}^{\beta}S_{k}(t) = \Lambda - \sigma_{k}\Phi(t)S_{k}(t) - (\mu + \mathbf{u}_{k})S_{k}(t) + \omega\left(1 - S_{k}(t) - I_{k}(t)\right) \\ & > \Lambda - S_{k}(t)\left(\sigma_{k}\varepsilon + \mu + \omega + \mathbf{u}_{k}\right) + \omega - \omega\varepsilon \\ & = (\mu + \omega - \omega\varepsilon) - (\sigma_{k}\varepsilon + \mu + \omega + \mathbf{u}_{k})S_{k}(t). \end{split}$$

Note that the following auxiliary system ${}_{0}^{C}\mathscr{D}_{t}^{\beta}\overline{S}_{k}(t) = (\mu + \omega - \omega\varepsilon) - (\sigma_{k}\varepsilon + \mu + \omega + \mathbf{u}_{k})\overline{S}_{k}(t)$ admits a unique equilibrium $S_{k}^{0,\varepsilon} = \frac{\mu + \omega - \omega\varepsilon}{\sigma_{k}\varepsilon + \mu + \omega + \mathbf{u}_{k}}$. Thus, for a sufficiently large time t, it yields

$$S_k(t) > \frac{\mu + \omega - \omega\varepsilon}{\sigma_k \varepsilon + \mu + \omega + \mathbf{u}_k}.$$

Furthermore, for all $t > T_1$, we also have ${}_0^C \mathscr{D}_t^\beta R_k(t) > -(\mu + \omega)R_k(t) + \frac{\mathbf{u}_k(\mu + \omega - \omega\varepsilon)}{\sigma_k\varepsilon + \mu + \omega + \mathbf{u}_k}$, which follows that

$$R_k(t) > \frac{\mathbf{u}_k(\mu + \omega - \omega\varepsilon)}{(\sigma_k\varepsilon + \mu + \omega + \mathbf{u}_k)(\mu + \omega)}$$

Finally, by letting $\varepsilon \to 0$, we directly obtain

$$\lim_{t \to \infty} S_k(t) = \frac{\mu + \omega}{\mu + \omega + \mathbf{u}_k} = S_k^0$$
$$\lim_{t \to \infty} R_k(t) = \frac{\mathbf{u}_k}{\mu + \omega + \mathbf{u}_k} = R_k^0$$

Therefore, the proof is completed.

Remark 4.4. It is easy to check that

 $\mathfrak{R}_0 < \tilde{\mathfrak{R}}_0,$

which means that the condition $\Re_0 < 1$ is not sufficient enough to ensure the global attractivity of the malware-free equilibrium \mathbf{P}_0 and in this case, one cannot eradicate the malware attacks unless the value of \Re_0 decreases such that $\Re_0 < \tilde{\Re}_0 < 1$ for some critical value $\tilde{\Re}_0$. In addition, it should be noted that even though the constant treatment rate r may be not enough for the malware elimination, we still can make the disease extinct while improving the immunization rate such that the threshold value $\tilde{\Re}_0 < 1$. In another hand, immunization strategy sometimes is more effective than treatment strategy in controlling the malware spread.

4.4. Bifurcation analysis

According to the results of Theorem 4.3, we can see that the condition $\mathfrak{R}_0 < 1$ is not enough for the absence of malware programs on complex heterogeneous network and it requires to stifle the cure parameter such that the basic reproductive ratio \mathfrak{R}_0 reaches to the threshold value $\tilde{\mathfrak{R}}_0$. In addition, as a consequence of Remark 4.3, there may have an endemic equilibrium \mathbf{P}_* co-existing with a stable malware-free equilibrium \mathbf{P}_0 even $\mathfrak{R}_0 < 1$. This leads to a bifurcating phenomena on the behavior of network-based epidemic models, so-called backward bifurcation. Some epidemiological mechanisms that can induce the phenomenon of backward bifurcation in epidemic models

are identified as follows: the use of an imperfect vaccine, the effects of limited resources for treatment, exogenous re-infection in the transmission process or sometimes, since the initial infectious population size is too large, etc. For more clearly, a schematic diagram of backward bifurcation is depicted in Figure 3.



Figure 3: The schematic diagram of backward bifurcation phenomena

In the following, we will establish the necessary and sufficient condition on the model's parameter for which the backward bifurcation at $\Re_0 = 1$ occurs.

Theorem 4.4. The fractional network-based SIRS epidemic model exhibits a backward bifurcation at $\mathfrak{R}_0 = 1$ if

$$\gamma > \frac{(\mu + r)\langle k^3 a \rangle \langle k \rangle}{r \langle k^2 u \rangle} \left(1 + \frac{r}{\mu + \omega} \right),$$

where the term $\langle k^3 a \rangle = \frac{1}{\langle k \rangle} \sum_{k=1}^n \frac{k^3 \mathbb{P}(k)}{\mu + \omega + \mathbf{u}_k}.$

Proof. In this proof, we consider the scenario that endemic equilibrium exists, i.e., the infectious state $I_k(t)$ is positive for all $t \ge 0$ and i = 1, 2, ..., n and hence, it implies that $\Phi(t) > 0$ for all $t \ge 0$. In addition, note that an endemic equilibrium of the fractional network-based SIRS epidemic model is a non-trivial solution of the self-consistency equation (8) or equivalently, it must satisfy the following nonlinear equation

$$\frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{\sigma k^2 \mathbb{P}(k)}{\left\{ \left(1 + \frac{\mathbf{u}_k}{\mu + \omega} \right) \left(\mu + \frac{r}{1 + \gamma \Phi} \right) + \sigma_k \Phi \left(1 + \frac{r}{(\mu + \omega)(1 + \gamma \Phi)} \right) \right\}} = 1$$

Next, by multiplying both sides of the above equation by $\frac{(\mu + \omega)\langle k^2 u \rangle}{(\mu + r)\langle k \rangle}$, it can be represented as an expression in terms of \Re_0 and Θ . As a result, one gets

$$\frac{1}{\langle k \rangle} \sum_{k=1}^{n} \frac{\Re_0 k^2 \mathbb{P}(k)}{\left\{ \frac{(\mu+\omega)\langle k^2 u \rangle}{(\mu+r)\langle k \rangle} \left(1 + \frac{\mathbf{u}_k}{\mu+\omega} \right) \left(\mu + \frac{r}{1+\gamma\Phi} \right) + k \Re_0 \Phi \left(1 + \frac{r}{(\mu+\omega)(1+\gamma\Phi)} \right) \right\}} = 1.$$
(9)

Then, the equality (9) can be known as the implicit equation of endemic equilibrium curve in the (\mathfrak{R}_0, Θ) -positive quadrant and the function Θ can be regarded as a function of \mathfrak{R}_0 . Now, in order to derive a necessary and sufficient condition of the model's parameters for which the backward bifurcation phenomena occurs, we evaluate the derivative $\frac{\partial \Theta}{\partial \mathfrak{R}_0}$ at point $(\mathfrak{R}_0, \Theta) = (1, 0)$ by implicitly differentiating the equation (9) w.r.t. the variable \mathfrak{R}_0 . Indeed, we directly get that

$$\sum_{k=1}^{n} \frac{\mathcal{A}_{1} - \mathcal{A}_{2}}{\left\{\frac{(\mu+\omega)\langle k^{2}u\rangle}{(\mu+r)\langle k\rangle} \left(1 + \frac{\mathbf{u}_{k}}{\mu+\omega}\right) \left(\mu + \frac{r}{1+\gamma\Phi}\right) + k\Re_{0}\Phi\left(1 + \frac{r}{(\mu+\omega)(1+\gamma\Phi)}\right)\right\}^{2}} = 0,$$
(10)

where the terms \mathcal{A}_1 and \mathcal{A}_2 are given as follows:

$$\begin{aligned} \mathcal{A}_{1} &= \frac{k^{2}\mathbb{P}(k)}{\langle k \rangle} \left\{ \frac{(\mu+\omega)\langle k^{2}u \rangle}{(\mu+r)\langle k \rangle} \left(1 + \frac{\mathbf{u}_{k}}{\mu+\omega} \right) \left(\mu + \frac{r}{1+\gamma\Phi} \right) + k\mathfrak{R}_{0}\Phi \left(1 + \frac{r}{(\mu+\omega)(1+\gamma\Phi)} \right) \right\}, \\ \mathcal{A}_{2} &= \frac{\mathfrak{R}_{0}k^{2}\mathbb{P}(k)}{\langle k \rangle} \left\{ -\frac{(\mu+\omega)\langle k^{2}u \rangle}{(\mu+r)\langle k \rangle} \left(1 + \frac{\mathbf{u}_{k}}{\mu+\omega} \right) \frac{r\gamma}{(1+\gamma\Phi)^{2}} \frac{\partial\Phi}{\partial\mathfrak{R}_{0}} + k\Phi \left(1 + \frac{r}{(\mu+\omega)(1+\gamma\Phi)} \right) \right. \\ &+ k\mathfrak{R}_{0} \frac{\partial\Phi}{\partial\mathfrak{R}_{0}} \left(1 + \frac{r}{(\mu+\omega)(1+\gamma\Phi)} \right) - \frac{r\gamma k\mathfrak{R}_{0}\Phi}{(\mu+\omega)(1+\gamma\Phi)^{2}} \frac{\partial\Phi}{\partial\mathfrak{R}_{0}} \right\}. \end{aligned}$$

At the point $(\mathfrak{R}_0, \Theta) = (1, 0)$, these above expressions are

$$\mathcal{A}_{1} = \frac{k^{2}\mathbb{P}(k)}{\langle k \rangle} \left\{ \frac{(\mu+\omega)\langle k^{2}u \rangle}{\langle k \rangle} \left(1 + \frac{\mathbf{u}_{k}}{\mu+\omega}\right) \right\}$$
$$\mathcal{A}_{2} = \frac{k^{2}\mathbb{P}(k)}{\langle k \rangle} \left\{ -\frac{r\gamma(\mu+\omega)\langle k^{2}u \rangle}{(\mu+r)\langle k \rangle} \left(1 + \frac{\mathbf{u}_{k}}{\mu+\omega}\right) + k\left(1 + \frac{r}{\mu+\omega}\right) \right\} \frac{\partial\Phi}{\partial\mathfrak{R}_{0}} \Big|_{(\mathfrak{R}_{0},\Theta)=(1,0)}.$$

As a result, the equation (10) becomes

$$\frac{1}{\langle k \rangle} \sum_{k=1}^{n} k^{2} \mathbb{P}(k) \frac{\left\{ -\frac{r\gamma(\mu+\omega)\langle k^{2}u \rangle}{(\mu+r)\langle k \rangle} \left(1+\frac{\mathbf{u}_{k}}{\mu+\omega}\right) + k\left(1+\frac{r}{\mu+\omega}\right) \right\} \frac{\partial \Phi}{\partial \mathfrak{R}_{0}} \Big|_{(\mathfrak{R}_{0},\Theta)=(1,0)}}{\left\{ \frac{(\mu+\omega)\langle k^{2}u \rangle}{\langle k \rangle} \left(1+\frac{\mathbf{u}_{k}}{\mu+\omega}\right) \right\}^{2}} = 1,$$

or equivalently,

$$\left\{-\frac{r\gamma}{\mu+r}+\frac{\langle k^3a\rangle\langle k\rangle}{\langle k^2u\rangle}\left(1+\frac{r}{\mu+\omega}\right)\right\}\frac{\partial\Phi}{\partial\Re_0}\Big|_{(\Re_0,\Theta)=(1,0)}=1$$

Since we consider locally Φ as a function of \Re_0 , the backward bifurcation is characterized by a negative value of the derivative $\frac{\partial \Phi}{\partial \Re_0}$ at the point $(\Re_0, \Theta) = (1, 0)$, which implies that

$$-\frac{r\gamma}{\mu+r}+\frac{\langle k^3a\rangle\langle k\rangle}{\langle k^2u\rangle}\left(1+\frac{r}{\mu+\omega}\right)<0.$$

Hence, the proof is completed.

5. Stabilization problem for a controlled fractional network-based SIRS epidemic model on wireless sensor network

Let the parameters of fractional network-based SIRS epidemic model be as follows:

$$\Lambda = \mu = 0.14, \qquad \omega = 0.1, \qquad \sigma = 0.8, \qquad r = 0.6, \qquad \gamma = 2.$$
(11)

Our aim in this section is to consider a stabilization problem for a controlled fractional network-based SIRS epidemic model on wireless sensor network which are regarded as a Barabási-Albert scale-free network (see Figure 4).

5



Figure 4: A simple wireless sensor network

Here, we assume that the maximum contact of a node on the network is n = 2 and the probability, that a randomly node has degree k, is given by $\mathbb{P}(k) = ck^{-3}$, where c is a parameter such that $\sum_{k=1}^{n} \mathbb{P}(k) = 1$. Thus, by using some simple computations, we obtain $c = \frac{8}{9}$. In addition, we can also compute the parameters $\langle k \rangle$ and $\langle k^2 \rangle$, that characterize the average degree and second moment of the network, as follows:

$$\langle k \rangle = \sum_{k=1}^{n} k \mathbb{P}(k) = \frac{10}{9}, \qquad \qquad \langle k^2 \rangle = \sum_{k=1}^{n} k^2 \mathbb{P}(k) = \frac{4}{3}$$

This section considers a scenario when epidemic disease due to malware programs breaks out and indeed, based on the above parameters, we can see that the basic reproductive ratio \Re_0 without control input $\mathbf{u}(t)$ satisfies $\Re_0 = 1.8018 > 1$. According to the result of Theorem 4.2, it is true that the malware-free equilibrium is unstable. In addition, to the best of our knowledge, it is not reasonable to administer the intervention at a constant rate when carrying out the immunization for complex heterogeneous network to deal with malware propagation. Motivated by aforesaid, our aim is to find a time-dependent immunization policy $\mathbf{u}(t)$ to stabilize the unstable malware-free equilibrium of the fractional network-based SIRS epidemic model, that is, the input control will steer the model's state to the no-malware state $\tilde{\mathbf{P}}_0 = (1, 0, 0, 1, 0, 0, \dots, 1, 0, 0)$ as time increases. Then, by denoting

$$\tilde{\mathbf{e}}(t) = \tilde{\mathbf{x}}(t) - \tilde{\mathbf{P}}_0 = \underbrace{(S_1 - 1, I_1, R_1, S_2 - 1, I_2, R_2, \dots, S_n - 1, I_n, R_n)}_{3n}$$

Hence, the requirement that the fractional network-based SIRS epidemic model is stabilized to malware-free equilibrium state is equivalent to the state vector $\mathbf{e}(t)$ approaches to zero state as time increases. Then, we directly receive a system of n following fractional differential systems

$$\begin{cases} {}^{C}_{0}\mathscr{D}^{\beta}_{t}S_{i}(t) = \Lambda - \sigma_{i}\Phi(t)\left(S_{i}(t) - 1\right) - \mu\left(S_{i}(t) - 1\right) - \mathbf{u}_{i}(t)\left(S_{i}(t) - 1\right) + \omega R_{i}(t) \\ {}^{C}_{0}\mathscr{D}^{\beta}_{t}I_{i}(t) = \sigma_{i}\Phi(t)\left(S_{i}(t) - 1\right) - \mu I_{i}(t) - \frac{rI_{i}(t)}{1 + \gamma\Phi(t)} \\ {}^{C}_{0}\mathscr{D}^{\beta}_{t}R_{i}(t) = \mathbf{u}_{i}(t)\left(S_{i}(t) - 1\right) + \frac{rI_{i}(t)}{1 + \gamma\Phi(t)} - (\mu + \omega)R_{i}(t), \end{cases}$$
(12)

for each i = 1, 2, ..., n, where c_i are the positive constants to keep a balance in the size of infection and $\mathbf{u}_i^2(t)$ reflects the severity of area effects of the immunization. It is well-known that there have been various works on the stabilization problems for network-based nonlinear systems [1, 19, 29, 30] and fractional Takagi-Sugeno fuzzy systems [2, 28, 29, 31, 46]. However, to the best of our knowledge, there doesn't have any literature studying the stabilization problem for fractional network-based differential systems. This may come from the complexity

in computation and numerical simulation of dynamical systems that take into account both large-scale systems and fractional differential systems. Hence, in this section, we will apply the interconnected Takagi-Sugeno model approach to design an appropriate fuzzy state-feedback controller $\mathbf{u}(t)$ for stabilizing the proposed network-based epidemic model. As mentioned, we firstly consider the i^{th} -differential subsystem (4) of the fractional networkbased SIRS epidemic model and apply the non-linearity sector method (see [24] for more detail) to construct the interconnected Takagi-Sugeno fuzzy system for this subsystem. Due to the fact that $\Lambda = \mu$, i.e., the total population $N_i(t)$ is unchanged and in order to reduce the complexity of computations, the behavior in time of the state3 function $S_i = 1 - I_i - R_i$ can be identified by considering only the (I_i, R_i) -dynamics. Based on the results of Lemma 4.1, it should be noted that the density function $S_i(t)$ of susceptible individuals is always positive and bounded above by 1. Additionally, the i^{th} -group is assumed to be received a constant rate Λ of new node's attendance. Hence, we reasonably assume that $S_i(t) \in [0.1, 0.9]$ for all t > 0, yielding $I_i + R_i \in [0.1, 0.9]$. Then, we can rewrite the fractional network-based differential system (12) as the following nonlinear dynamic system with the state vector $\mathbf{e}_i(t) = [I_i(t) \quad R_i(t)]^{\top}$ and the control input $\mathbf{u}_i(t)$:

$${}_{0}^{C}\mathscr{D}_{t}^{\beta} \begin{bmatrix} I_{i}(t) \\ R_{i}(t) \end{bmatrix} = \begin{bmatrix} -\mu - \frac{r}{1+\gamma\Phi(t)} + \frac{\sigma_{i}i\mathbb{P}(i)(S_{i}(t)-1)}{\langle k \rangle} & 0 \\ \frac{r}{1+\gamma\Phi(t)} & -(\mu+\omega) \end{bmatrix} \begin{bmatrix} I_{i}(t) \\ R_{i}(t) \end{bmatrix} + \begin{bmatrix} \frac{\sigma_{i}(S_{i}(t)-1)}{\langle k \rangle} \sum_{\substack{j=1\\ j\neq i}}^{n} j\mathbb{P}(j)I_{j}(t) \\ 0 \end{bmatrix} + \begin{bmatrix} 0 \\ S_{i}(t) - 1 \end{bmatrix} \mathbf{u}_{i}(t)$$

or equivalently,

Now, in order to establish a corresponding interconnected Takagi-Sugeno fuzzy system for the fractional networkbased SIRS epidemic model, we regard two non-constant terms $S_i(t)$ and $\frac{r}{1+\gamma\Phi(t)}$ in the expression (13) as the premise variables. Then, for each of these two terms, the weighting functions of the i^{th} -differential subsystem are computed as follows:

(i) The premise variable is $z_{i1} = S_i \in [0.1, 0.9]$. Then, the corresponding weighting functions are

$$\eta_{i0}^{1}(z_{i1}) = \frac{1 - z_{i1}}{0.8} \qquad \qquad \eta_{i1}^{1}(z_{i1}) = 1 - \eta_{i0}^{1}(z_{i1}) = \frac{z_{i1} - 0.2}{0.8}.$$

Then, by applying Lemma 1.1 in [2], the premise variable z_{i1} can be represented as a weighted sum

$$z_{i1} = 0.1\eta_{i0}^{1}(z_{i1}) + 0.9\eta_{i1}^{1}(z_{i1}).$$

(ii) The premise variable is $z_{i2} = \frac{r}{1 + \gamma \Phi(t)} \in \left[\frac{r}{1 + \gamma}, r\right] = [0.2, 0.6]$. Then, the corresponding weighting functions are given by

$$\eta_{i0}^2(z_{i2}) = \frac{5(1-z_{i2})}{2} \qquad \qquad \eta_{i1}^2(z_{i2}) = 1 - \eta_{i0}^2(z_{i2}) = \frac{5z_{i2}-3}{2}.$$

Then, the premise variable z_{i2} can be represented as a weighted sum

$$z_{i2} = 0.2\eta_{i0}^2 (z_{i2}) + 0.6\eta_{i1}^2 (z_{i2}) + 0.6\eta_{i1}^2$$

For each weighting function, we denote the corresponding fuzzy set by F_{ik}^{χ} for each $i = \overline{1, n}$, k = 0, 1 and $\chi = 1, 2$, i.e., the fuzzy set F_{i0}^1 corresponds to the weighting function $\eta_{i0}^1(z_{i1})$ and for convenience in computation, we use triangular membership functions to describe fuzzy sets in all fuzzy rules. The graphical representations of antecedent fuzzy sets F_{ik}^{χ} are given in Figure 5.



Figure 5: The membership functions of antecedent fuzzy sets F_{ik}^1 and F_{ik}^2

As a consequence, this technique leads to 4n local models by combining 2n membership functions of antecedent fuzzy sets F_{ik}^1 and F_{ik}^2 . Denote by $\mathbf{z}_i(t) = \begin{bmatrix} z_{i1}(t) & z_{i2}(t) \end{bmatrix}^{\top}$ the measurable premise variable vector. Then, we can establish an interconnected fractional Takagi-Sugeno fuzzy system for the i^{th} -fractional network-based differential subsystem (13) whose fuzzy rules are given as follows:

Rule \mathbf{E}_{i}^{p} : If z_{i1} is F_{i0}^{χ} and z_{i2} is F_{i1}^{χ} then

$${}_{0}^{C} \mathscr{D}_{t}^{\beta} \mathbf{e}_{i}(t) = A_{i}^{p} \mathbf{e}_{i}(t) + B_{i}^{p} \mathbf{u}_{i}(t) + \sum_{j \neq i}^{n} \alpha_{ij}^{p} \mathbf{e}_{j}(t),$$

where A_i^p , B_i^p and α_{ij}^p are real matrices with appropriate matrix for all $i = \overline{1, n}$, $p = \overline{1, r_i}$ and $r_i = 4$. Then, according to the formulation (3), we directly receive the membership function $w_i^p(\mathbf{z}_i(t))$, that indicates the activation degree of the p^{th} -local model of the subsystem \mathbf{E}_i , as follows:

$$\varphi_{i}^{p}\left(\mathbf{z}_{i}(t)\right) = \eta_{ik}^{1}\eta_{ij}^{2},$$
$$w_{i}^{p}\left(\mathbf{z}_{i}(t)\right) = \frac{\varphi_{i}^{p}\left(\mathbf{z}_{i}(t)\right)}{\sum_{p=1}^{r_{i}}\varphi_{i}^{p}\left(\mathbf{z}_{i}(t)\right)}.$$

Next, according to the formulation (2), the i^{th} -fractional network-based differential subsystem (13) can be characterized by the following interconnected fractional Takagi-Sugeno fuzzy system

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{e}_{i}(t) = \sum_{p=1}^{r_{i}} w_{i}^{p}\left(\mathbf{z}_{i}(t)\right) \left\{ A_{i}^{p}\mathbf{e}_{i}(t) + B_{i}^{p}\mathbf{u}_{i}(t) + \sum_{j=1 \atop j \neq i}^{n} \alpha_{ij}^{p}\mathbf{e}_{j}(t) \right\} \qquad \left(i = \overline{1, n}\right),$$

and the interconnected fractional Takagi-Sugeno fuzzy system for the fractional network-based SIRS epidemic

model is given in following vector form

$${}^{C}_{0}\mathscr{D}^{\beta}_{t}\mathbf{e}(t) = \begin{bmatrix} \sum_{p=1}^{r_{1}} w_{1}^{p}\left(\mathbf{z}_{1}(t)\right) \left\{ A_{1}^{p}\mathbf{e}_{1}(t) + B_{1}^{p}\mathbf{u}_{1}(t) + \sum_{\substack{j=1\\j\neq i}}^{n} \alpha_{1j}^{p}\mathbf{e}_{j}(t) \right\} \\ \vdots \\ \sum_{p=1}^{r_{n}} w_{n}^{p}\left(\mathbf{z}_{n}(t)\right) \left\{ A_{n}^{p}\mathbf{e}_{n}(t) + B_{n}^{p}\mathbf{u}_{n}(t) + \sum_{\substack{j=1\\j\neq i}}^{n} \alpha_{nj}^{p}\mathbf{e}_{j}(t) \right\} \end{bmatrix}.$$
(14)

In the following, based on the idea of parallel distributed compensation (PDC) introduced in [3, 49], we would like to design a fuzzy state-feedback controller $\mathbf{u}(t)$ for stabilizing the unstable malware-free equilibrium of the fractional network-based SIRS epidemic model. For this aim, according to the derivation in [30, 42], each subsystem in form (4) of the fractional network-based SIRS epidemic model can be represented by the interconnected fractional Takagi-Sugeno fuzzy system (14). Then, the p^{th} -rule (p = 1, 2, 3) of fuzzy controller in the subsystem \mathbf{E}_i can be considered in following PDC form

Rule \mathbf{E}_{i}^{p} : If z_{i1} is F_{i0}^{χ} and z_{i2} is F_{i1}^{χ} then

$$\mathbf{u}_i(t) = K_i^p \mathbf{x}_i(t).$$

Hence, the final output of the fuzzy state-feedback controller $\mathbf{u}_i(t)$ of the subsystem \mathbf{E}_i is

$$\mathbf{u}_{i}(t) = \sum_{p=1}^{r_{i}} w_{i}^{p} \left(\mathbf{z}_{i}(t) \right) K_{i}^{p} \mathbf{x}_{i}(t) \qquad \left(i = \overline{1, n} \right).$$
(15)

Denote by $\mathbf{u}(t) = (\mathbf{u}_1(t), \dots, \mathbf{u}_n(t))^{\top}$ the fuzzy state-feedback controller for the fractional network-based SIRS epidemic model. In the following, we will establish some necessary conditions for which the fuzzy state-feedback controller $\mathbf{u}(t)$ stabilizes the unstable disease-free equilibrium of the proposed network-based epidemic model:

Theorem 5.1. Assume that there exist some symmetric positive definite matrices P_i , positive definite matrices $Q_i \succ 0$, symmetric matrices U_i^{pm} and U_{ij}^{pm} , matrices K_i^p satisfying the following linear matrix inequalities

$$\mathbf{Q}_i^{pm} \preceq \mathbf{U}_i^{pm} \tag{LMI.1}$$

$$\left(\alpha_{ij}^{p}\right)^{\top} \mathbf{P}_{i} + \mathbf{P}_{i} \alpha_{ij}^{p} + \left(\alpha_{ji}^{m}\right)^{\top} \mathbf{P}_{j} + \mathbf{P}_{j} \alpha_{ji}^{m} \preceq 2\mathbf{U}_{ij}^{pm}$$
(LMI.2)

$$\mathbb{U} = \begin{bmatrix} U_1 & U_{12} & \cdots & U_{1n} \\ U_{12}^{\top} & U_2 & \cdots & U_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ U_{1n}^{\top} & U_{2n}^{\top} & \cdots & U_n \end{bmatrix} \prec 0,$$
(LMI.3)

where for each $i, j = \overline{1, n}$, the matrices U_i and U_{ij} $(i \neq j)$ are given by

$$\mathbf{U}_{i} = \begin{bmatrix} \mathbf{U}_{i}^{11} & \mathbf{U}_{i}^{12} & \cdots & \mathbf{U}_{i}^{1r_{i}} \\ (\mathbf{U}_{i}^{12})^{\top} & \mathbf{U}_{i}^{22} & \cdots & \mathbf{U}_{i}^{2r_{i}} \\ \vdots & \vdots & \ddots & \vdots \\ (\mathbf{U}_{i}^{1r_{i}})^{\top} & (\mathbf{U}_{i}^{2r_{i}})^{\top} & \cdots & \mathbf{U}_{i}^{r_{i}r_{i}} \end{bmatrix}, \qquad \mathbf{U}_{ij} = \begin{bmatrix} \mathbf{U}_{ij}^{11} & \mathbf{U}_{ij}^{12} & \cdots & \mathbf{U}_{ij}^{1r_{j}} \\ \mathbf{U}_{ij}^{21} & \mathbf{U}_{ij}^{22} & \cdots & \mathbf{U}_{ij}^{2r_{j}} \\ \vdots & \vdots & \ddots & \vdots \\ \mathbf{U}_{ij}^{r_{i}1} & \mathbf{U}_{ij}^{r_{i}2} & \cdots & \mathbf{U}_{ij}^{r_{i}r_{j}} \end{bmatrix}$$

Then, the interconnected fractional Takagi-Sugeno fuzzy system (14) is asymptotically stable under the fuzzy state-feedback control $\mathbf{u}(t)$.

Proof. Under the fuzzy state-feedback control $\mathbf{u}_i(t)$ defined by the formula (15), the i^{th} -interconnected fractional Takagi-Sugeno fuzzy system (14) becomes

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{e}_{i}(t) = \sum_{p=1}^{r_{i}} \sum_{m=1}^{r_{i}} w_{i}^{p}\left(\mathbf{z}_{i}(t)\right) w_{i}^{m}\left(\mathbf{z}_{i}(t)\right) \left(A_{i}^{p} + B_{i}^{p}K_{i}^{m}\right) \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p}\left(\mathbf{z}_{i}(t)\right) \alpha_{ij}^{p}\mathbf{e}_{j}(t).$$
(16)

Note that the stabilization of the i^{th} -interconnected fractional Takagi-Sugeno fuzzy system (14) under the fuzzy state-feedback controller $\mathbf{u}_i(t)$ is equivalent to the asymptotic stability of the closed-loop interconnected fractional Takagi-Sugeno fuzzy system (16). Here, we rewrite the fractional differential system (16) as follows:

$$\begin{split} {}_{0}^{C} \mathscr{D}_{t}^{\beta} \mathbf{e}_{i}(t) &= \sum_{p=1}^{r_{i}} \sum_{m=1}^{r_{i}} w_{i}^{p} \left(\mathbf{z}_{i}(t) \right) w_{i}^{m} \left(\mathbf{z}_{i}(t) \right) \left(A_{i}^{p} + B_{i}^{p} K_{i}^{m} \right) \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p} \left(\mathbf{z}_{i}(t) \right) \alpha_{ij}^{p} \mathbf{e}_{j}(t) \\ &= \sum_{p=1}^{r_{i}} \left[w_{i}^{p} \left(\mathbf{z}_{i}(t) \right) \right]^{2} \left(A_{i}^{p} + B_{i}^{p} K_{i}^{p} \right) \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p} \left(\mathbf{z}_{i}(t) \right) \alpha_{ij}^{p} \mathbf{e}_{j}(t) \\ &+ \sum_{p=1}^{r_{i}} \sum_{m=p+1}^{r_{i}} w_{i}^{p} \left(\mathbf{z}_{i}(t) \right) w_{i}^{m} \left(\mathbf{z}_{i}(t) \right) \left\{ \left(A_{i}^{p} + B_{i}^{p} K_{i}^{m} \right) \mathbf{e}_{i}(t) + \left(A_{i}^{m} + B_{i}^{m} K_{i}^{p} \right) \mathbf{e}_{i}(t) \right\} \end{split}$$

For simplicity in representation, we use the notations w_i^p , w_i^m instead of the terms $w_i^p(\mathbf{z}_i(t))$ and $w_i^m(\mathbf{z}_i(t))$. In addition, for each $p, m = \overline{1, r_i}$ and $i = \overline{1, n}$, we denote

$$\mathbf{G}_i^{pm} = A_i^p + B_i^p K_i^m, \qquad \qquad \mathbf{Q}_i^{pm} = \left(\mathbf{G}_i^{pm}\right)^\top \mathbf{P}_i + \mathbf{P}_i \mathbf{G}_i^{pm}.$$

Then, we receive the closed-loop interconnected fractional Takagi-Sugeno fuzzy system in following compact form

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{e}_{i}(t) = \sum_{p=1}^{r_{i}} (w_{i}^{p})^{2} \operatorname{G}_{i}^{pp}\mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p} \alpha_{ij}^{p} \mathbf{e}_{j}(t) + \sum_{p=1}^{r_{i}} \sum_{m=p+1}^{r_{i}} w_{i}^{p} w_{i}^{m} \left(\operatorname{G}_{i}^{pm} + \operatorname{G}_{i}^{mp}\right) \mathbf{e}_{i}(t).$$
(17)

Next, we consider the Lyapunov function for the system (17) as follows:

$$\mathbf{V}(\mathbf{e}(t)) = \sum_{i=1}^{n} \mathbf{V}_{i}(\mathbf{e}(t)),$$

where the function $\mathbf{V}_i(\mathbf{e}(t))$ is given by $\mathbf{V}_i(\mathbf{e}(t)) = \mathbf{e}_i^{\top}(t)\mathbf{P}_i\mathbf{e}_i(t)$. Next, by taking the fractional derivative of order β in Caputo sense of the function $\mathbf{V}_i(\mathbf{e}(t))$ along the solution of (17) and using Lemma 2.2, we receive

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{V}_{i}\left(\mathbf{e}(t)\right) \leq \left[{}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{e}_{i}^{\top}(t)\right]\mathbf{P}_{i}\mathbf{e}_{i}(t) + \mathbf{e}_{i}^{\top}(t)\mathbf{P}_{i}\left[{}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{e}_{i}(t)\right]$$

Next, by using the right-hand side of the fractional differential equation (17), we directly obtain

$$\begin{split} {}_{0}^{C} \mathscr{D}_{t}^{\beta} \mathbf{V}_{i} \left(\mathbf{e}(t) \right) &\leq \sum_{p=1}^{r_{i}} \left(w_{i}^{p} \right)^{2} \mathbf{e}_{i}^{\top}(t) \left\{ \left(\mathbf{G}_{i}^{pp} \right)^{\top} \mathbf{P}_{i} + \mathbf{P}_{i} \mathbf{G}_{i}^{pp} \right\} \mathbf{e}_{i}(t) \\ &+ \sum_{p=1}^{r_{i}} \sum_{m=p+1}^{r_{i}} w_{i}^{p} w_{i}^{m} \mathbf{e}_{i}^{\top}(t) \left\{ \left(\mathbf{G}_{i}^{pm} \right)^{\top} \mathbf{P}_{i} + \mathbf{P}_{i} \mathbf{G}_{i}^{pm} + \left(\mathbf{G}_{i}^{mp} \right)^{\top} \mathbf{P}_{i} + \mathbf{P}_{i} \mathbf{G}_{i}^{mp} \right\} \mathbf{e}_{i}(t) \\ &+ \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p} \mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p} \mathbf{e}_{i}^{\top}(t) \mathbf{P}_{i} \alpha_{ij}^{p} \mathbf{e}_{j}(t) \\ &= \sum_{p=1}^{r_{i}} \left(w_{i}^{p} \right)^{2} \mathbf{e}_{i}^{\top}(t) \mathbf{Q}_{i}^{pp} \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{m=p+1}^{r_{i}} w_{i}^{p} w_{i}^{m} \mathbf{e}_{i}^{\top}(t) \left\{ \mathbf{Q}_{i}^{pm} + \mathbf{Q}_{i}^{mp} \right\} \mathbf{e}_{i}(t) \\ &+ \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p} \mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{j=1}^{n} w_{i}^{p} \mathbf{e}_{i}^{\top}(t) \mathbf{P}_{i} \alpha_{ij}^{p} \mathbf{e}_{j}(t). \end{split}$$
After that, by using the expression $\sum_{m=1}^{r_j} w_j^m(\mathbf{z}_j(t)) = 1$ and the inequality (LMI.1), we deduce that

$$\begin{split} {}_{0}^{C} \mathscr{D}_{t}^{\beta} \mathbf{V}_{i} \left(\mathbf{e}(t) \right) &\leq \sum_{p=1}^{r_{i}} \left(w_{i}^{p} \right)^{2} \mathbf{e}_{i}^{\top}(t) \mathbf{U}_{i}^{pp} \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} \sum_{m=p+1}^{r_{i}} w_{i}^{p} w_{i}^{m} \mathbf{e}_{i}^{\top}(t) \left\{ \mathbf{U}_{i}^{pm} + \mathbf{U}_{i}^{mp} \right\} \mathbf{e}_{i}(t) \\ &+ \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) + \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \mathbf{e}_{i}^{\top}(t) \mathbf{P}_{i} \alpha_{ij}^{p} \mathbf{e}_{j}(t) \\ &\leq \sum_{i=1}^{n} \left\{ \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \left[\begin{array}{c} \mathbf{U}_{i}^{11} \cdots \mathbf{U}_{i}^{1r_{i}} \\ \vdots & \ddots & \vdots \\ \left(\mathbf{U}_{i}^{1r_{i}} \right)^{\top} \cdots \mathbf{U}_{i}^{pr_{i}} \mathbf{e}_{i} \right] \right] \left\{ \begin{array}{c} w_{i}^{1} \mathbf{e}_{i}(t) \\ \vdots \\ w_{i}^{r_{i}} \mathbf{e}_{i}(t) \end{array} \right\} \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) + \sum_{i=1}^{n} \sum_{p=1}^{n} w_{j}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \mathbf{e}_{j}^{\top}(t) \mathbf{P}_{i} \alpha_{ij}^{p} \mathbf{e}_{j}(t) \\ &= \sum_{i=1}^{n} \left\{ \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \mathbf{U}_{i} \left[\begin{array}{c} w_{i}^{1} \mathbf{e}_{i}(t) \\ \vdots \\ w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \right\} \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \frac{\mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) \\ \vdots \\ w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \right\} \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \frac{\mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{n} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{n} w_{j}^{m} \frac{\mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{n} w_{i}^{m} \frac{\mathbf{e}_{i}^{\top}(t) \mathbf{P}_{i} \alpha_{ij}^{p} \mathbf{e}_{j}(t) \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{n} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{n} w_{j}^{m} \frac{\mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{n} w_{i}^{m} \frac{\mathbf{e}_{i}^{\top}(t) \mathbf{P}_{i} \alpha_{ij}^{p} \mathbf{e}_{i}(t) \\ &+ \sum_{i=1}^{n} \sum_{p=1}^{n} w_{i}^{m} \sum_{j=1}^{n} \sum_{m=1}^{n} w_{j}^{m} \frac{\mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf$$

Then, by changing indices and the inequality (LMI.2), we directly receive

$$\begin{split} {}_{0}^{C} \mathscr{D}_{i}^{\beta} \mathbf{V}_{i} \left(\mathbf{e}(t) \right) \leq & \sum_{i=1}^{n} \left\{ \begin{bmatrix} w_{i}^{1} \mathbf{e}_{i}(t) & \cdots & w_{i}^{r_{i}} \mathbf{e}_{i}(t) \end{bmatrix} \mathbf{U}_{i} \begin{bmatrix} w_{i}^{1} \mathbf{e}_{i}(t) \\ \vdots \\ w_{i}^{T_{i}} \mathbf{e}_{i}(t) \end{bmatrix} \right\} \\ & + \sum_{i=1}^{n} \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \frac{\mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) }{2} + \sum_{i=1}^{n} \sum_{p=1}^{r_{i}} w_{j}^{p} \sum_{j=1}^{r_{j}} \sum_{m=1}^{n} w_{j}^{m} \frac{\mathbf{e}_{j}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{i} \mathbf{e}_{i}(t) }{2} \\ & + \sum_{i=1}^{n} \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \frac{\mathbf{e}_{i}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{j} \mathbf{e}_{j}(t) \\ & + \sum_{i=1}^{n} \sum_{p=1}^{r_{i}} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \frac{\mathbf{e}_{i}^{\top}(t) \left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{j} \mathbf{e}_{j}(t) \\ & = \sum_{i=1}^{n} \left\{ \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \mathbf{U}_{i} \begin{bmatrix} w_{i}^{1} \mathbf{e}_{i}(t) \\ \vdots \\ w_{i}^{n} \mathbf{e}_{i}(t) \end{bmatrix} \right\} + \sum_{i=1}^{n} \sum_{p=1}^{r_{j}} w_{j}^{p} \mathbf{e}_{j}^{\top}(t) \frac{\left(\alpha_{ij}^{p} \right)^{\top} \mathbf{P}_{j} \mathbf{P}_{j} \mathbf{P}_{j} \\ & + \sum_{i=1}^{n} \sum_{p=1}^{n} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{r_{j}} w_{j}^{m} \mathbf{e}_{i}^{\top}(t) \frac{\mathbf{P}_{i} \alpha_{ij}^{p} + \left(\alpha_{ji}^{pi} \right)^{\top} \mathbf{P}_{j} \\ & = \sum_{i=1}^{n} \left\{ \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \mathbf{U}_{i} \begin{bmatrix} w_{i}^{1} \mathbf{e}_{i}(t) \\ \vdots \\ w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \right\} + \sum_{i=1}^{n} \sum_{p=1}^{n} w_{i}^{p} \sum_{j=1}^{n} \sum_{m=1}^{n} w_{j}^{p} \mathbf{e}_{j}^{\top}(t) \frac{\mathbf{U}_{ij}^{pm}}{2} \\ & = \sum_{i=1}^{n} \left\{ \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{i}^{r_{i}} \mathbf{e}_{i}(t) \right] \mathbf{U}_{i} \begin{bmatrix} w_{i}^{1} \mathbf{e}_{i}(t) \\ \vdots \\ w_{i}^{r_{i}} \mathbf{e}_{i}(t) \end{bmatrix} \right\} \\ & = \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{1}^{r_{i}} \mathbf{e}_{i}^{T}(t) \frac{\mathbf{U}_{ij}^{pm}}{2} \\ & = \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{1}^{r_{i}} \mathbf{e}_{i}(t) \frac{\mathbf{U}_{ij}}{2} \\ & = \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{1}^{r_{i}} \mathbf{e}_{i}(t) \cdots w_{n}^{n} \mathbf{e}_{n}^{n}(t) \frac{\mathbf{U}_{ij}^{pm}}{2} \\ & = \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{1}^{r_{i}} \mathbf{e}_{i}(t) \frac{\mathbf{U}_{ij}}{2} \\ & = \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{1}^{r_{i}} \mathbf{e}_{i}(t) \frac{\mathbf{U}_{ij}}{2} \\ & = \left[w_{i}^{1} \mathbf{e}_{i}(t) \cdots w_{1}^{r_{i}} \mathbf{e}_{i}(t$$

For simplicity, we denote $w\mathbf{e}(t) = \begin{bmatrix} w_1^1\mathbf{e}_1(t) & \cdots & w_1^{r_1}\mathbf{e}_1(t) & \cdots & w_n^1\mathbf{e}_n(t) \end{bmatrix} \in \mathbb{R}^{n \times r_i}$. Then, by using the linear matrix inequality (LMI.3) and since the fact that \mathbb{U} is a symmetry matrix, Rayleigh inequality can be applied to show that there exists a negative constant $\lambda_{\max}(\mathbb{U})$ such that

$${}_{0}^{C}\mathscr{D}_{t}^{\beta}\mathbf{V}_{i}\left(\mathbf{e}(t)\right) \leq \lambda_{\max}\left(\mathbb{U}\right) \|w\mathbf{e}(t)\|^{2} < 0.$$

Additionally, since the matrix P_i is symmetric positive definite then by applying Rayleigh inequality, there exist two positive constants $\lambda_{\min}(P_i)$ and $\lambda_{\max}(P_i)$ such that

$$\lambda_{\min} \left(\mathbf{P}_i \right) \| \mathbf{e}(t) \|^2 \le \mathbf{V}_i \left(\mathbf{e}(t) \right) \le \lambda_{\max} \left(\mathbf{P}_i \right) \| \mathbf{e}(t) \|^2.$$

Finally, we can apply the result of Theorem 2.2 to guarantee that the interconnected fractional Takagi-Sugeno fuzzy system (14) is asymptotically stable. \Box

Remark 5.1. It should be noted that the conditions of Theorem 5.1 are not easy to verify by MatLab toolbox. Therefore, we will apply Schur complement theorem to transform the linear matrix inequalities (LMI.1), (LMI.2) and (LMI.3) into some linear matrix inequalities that can be solvable more easily. To do this, for each $i, j = \overline{1, n}$ and $p, m = \overline{1, r_i}$, we carry out the following transformations

$$\mathbf{C}_i = \mathbf{P}_i^{-1}, \qquad K_i^p = \mathbf{W}_i^p \mathbf{C}_i^{-1}, \qquad \tilde{\mathbf{U}}_i^{pm} = \mathbf{C}_i \mathbf{U}_i^{pm} \mathbf{C}_i, \qquad \tilde{\mathbf{U}}_{ij}^{pm} = \mathbf{C}_i \mathbf{U}_{ij}^{pm} \mathbf{C}_j + \mathbf{C}_j \mathbf{U}_{ij}^{pm} \mathbf{C}_i$$

and

$$\begin{split} \tilde{\mathbf{Q}}_{i}^{pm} &= \mathbf{C}_{i} \left\{ \left(\mathbf{G}_{i}^{pm} \right)^{\top} \mathbf{P}_{i} + \mathbf{P}_{i} \mathbf{G}_{i}^{pm} \right\} \mathbf{C}_{i} \\ &= \mathbf{C}_{i} \left\{ \left(A_{i}^{p} + B_{i}^{p} K_{i}^{m} \right)^{\top} \mathbf{P}_{i} + \mathbf{P}_{i} \left(A_{i}^{p} + B_{i}^{p} K_{i}^{m} \right) \right\} \mathbf{C}_{i} \\ &= \mathbf{C}_{i} \left(A_{i}^{p} \right)^{\top} + A_{i}^{p} \mathbf{C}_{i} + B_{i}^{p} \mathbf{W}_{i}^{m} + \left(\mathbf{W}_{i}^{m} \right)^{\top} \left(B_{i}^{p} \right)^{\top}. \end{split}$$

Next, by multiplying on the left and right of the matrices U_i and U_{ij} by diag $\underbrace{[C_i, \ldots, C_i]}_n$, we obtain

$$\begin{split} \tilde{\mathbf{U}}_{i} &= \begin{bmatrix} \tilde{\mathbf{U}}_{i}^{11} & \tilde{\mathbf{U}}_{i}^{12} & \cdots & \tilde{\mathbf{U}}_{i}^{1r_{i}} \\ (\tilde{\mathbf{U}}_{i}^{12})^{\top} & \tilde{\mathbf{U}}_{i}^{22} & \cdots & \tilde{\mathbf{U}}_{i}^{2r_{i}} \\ \vdots & \vdots & \ddots & \vdots \\ (\tilde{\mathbf{U}}_{i}^{1r_{i}})^{\top} & (\tilde{\mathbf{U}}_{i}^{2r_{i}})^{\top} & \cdots & \tilde{\mathbf{U}}_{i}^{r_{i}r_{i}} \end{bmatrix} \\ \tilde{\mathbf{U}}_{ij} & \tilde{\mathbf{U}}_{ij}^{12} & \cdots & \tilde{\mathbf{U}}_{ij}^{1r_{j}} \\ \tilde{\mathbf{U}}_{ij}^{21} & \tilde{\mathbf{U}}_{ij}^{22} & \cdots & \tilde{\mathbf{U}}_{ij}^{2r_{j}} \\ \vdots & \vdots & \ddots & \vdots \\ \tilde{\mathbf{U}}_{ij}^{r_{i}1} & \tilde{\mathbf{U}}_{ij}^{r_{i}2} & \cdots & \tilde{\mathbf{U}}_{ij}^{r_{i}r_{j}} \end{bmatrix}. \end{split}$$

Hence, we can rewritten the system of linear matrix inequalities in Theorem 5.1 as follows:

$$\tilde{Q}_i^{pm} \preceq \tilde{U}_i^{pm} \tag{LMI.4}$$

$$\left(\alpha_{ij}^{p}\right)^{\top} \mathbf{C}_{i} + \mathbf{C}_{i} \alpha_{ij}^{p} + \left(\alpha_{ji}^{m}\right)^{\top} \mathbf{C}_{j} + \mathbf{C}_{j} \alpha_{ji}^{m} \preceq 2\tilde{\mathbf{U}}_{ij}^{pm}$$

$$\left[\tilde{\mathbf{U}}_{1} \quad \tilde{\mathbf{U}}_{12} \quad \cdots \quad \tilde{\mathbf{U}}_{1n}\right]$$

$$(LMI.5)$$

$$\tilde{\mathbb{U}} = \begin{bmatrix} \tilde{U}_1 & \tilde{U}_1^T & \tilde{U}_1^T & \tilde{U}_1^T & \tilde{U}_2^T & \cdots & \tilde{U}_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ \tilde{U}_{1n}^T & \tilde{U}_{2n}^T & \cdots & \tilde{U}_n \end{bmatrix} \prec 0.$$
(LMI.6)

Under the above model's parameters (11), the interconnected fractional Takagi-Sugeno fuzzy system for the fractional network-based SIRS epidemic model can be presented by the following fuzzy rules:

Rule E¹_i: If z_{i1} is F_{i0}^1 and z_{i2} is F_{i0}^2 then ${}_0^C \mathscr{D}_t^\beta \mathbf{e}_i(t) = A_i^1 \mathbf{e}_i(t) + B_i^1 \mathbf{u}_i(t) + \sum_{\substack{j=1\\j\neq i}}^n \alpha_{ij}^1 \mathbf{e}_j(t)$,

Rule \mathbf{E}_i^2 : If z_{i1} is F_{i0}^1 and z_{i2} is F_{i1}^2 then ${}_0^C \mathscr{D}_t^\beta \mathbf{e}_i(t) = A_i^2 \mathbf{e}_i(t) + B_i^2 \mathbf{u}_i(t) + \sum_{\substack{j=1\\j\neq i}}^n \alpha_{ij}^2 \mathbf{e}_j(t)$,

Rule E³_i: If
$$z_{i1}$$
 is F_{i1}^1 and z_{i2} is F_{i0}^2 then ${}_0^C \mathscr{D}_t^\beta \mathbf{e}_i(t) = A_i^3 \mathbf{e}_i(t) + B_i^3 \mathbf{u}_i(t) + \sum_{\substack{j=1\\j\neq i}}^n \alpha_{ij}^3 \mathbf{e}_j(t)$

Rule E⁴_i: If
$$z_{i1}$$
 is F_{i1}^1 and z_{i2} is F_{i1}^2 then ${}_0^C \mathscr{D}_t^\beta \mathbf{e}_i(t) = A_i^4 \mathbf{e}_i(t) + B_i^4 \mathbf{u}_i(t) + \sum_{j=1 \ j \neq i}^n \alpha_{ij}^4 \mathbf{e}_j(t)$,

where for i = 1, 2, we have

$A_1^1 = \begin{bmatrix} -0.916\\0.2\end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_1^1 = \begin{bmatrix} 0\\ -0.9 \end{bmatrix},$	$\alpha_{12}^1 = \begin{bmatrix} -0.144\\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$
$A_1^2 = \begin{bmatrix} -1.316\\ 0.6 \end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_1^2 = \begin{bmatrix} 0\\ -0.9 \end{bmatrix},$	$\alpha_{12}^2 = \begin{bmatrix} -0.144\\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$
$A_1^3 = \begin{bmatrix} -0.404\\0.2\end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_1^3 = \begin{bmatrix} 0\\ -0.1 \end{bmatrix},$	$\alpha_{12}^3 = \begin{bmatrix} -0.016\\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$
$A_1^4 = \begin{bmatrix} -0.804\\ 0.6 \end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_1^4 = \begin{bmatrix} 0\\ -0.1 \end{bmatrix},$	$\alpha_{12}^4 = \begin{bmatrix} -0.016\\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$
$A_2^1 = \begin{bmatrix} -0.628\\0.2\end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_2^1 = \begin{bmatrix} 0\\ -0.9 \end{bmatrix},$	$\alpha_{21}^1 = \begin{bmatrix} -1.152\\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$
$A_2^2 = \begin{bmatrix} -1.028\\ 0.6 \end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_2^2 = \begin{bmatrix} 0\\ -0.9 \end{bmatrix},$	$\alpha_{21}^2 = \begin{bmatrix} -1.152\\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$
$A_2^3 = \begin{bmatrix} -0.372\\0.2\end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_2^3 = \begin{bmatrix} 0\\ -0.1 \end{bmatrix},$	$\alpha_{21}^3 = \begin{bmatrix} -0.128\\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$
$A_2^4 = \begin{bmatrix} -0.772\\ 0.6 \end{bmatrix}$	$\begin{bmatrix} 0\\ -0.24 \end{bmatrix},$	$B_2^4 = \begin{bmatrix} 0\\ -0.1 \end{bmatrix},$	$\alpha_{21}^4 = \begin{bmatrix} -0.128 \\ 0 \end{bmatrix}$	$\begin{bmatrix} 0\\ 0 \end{bmatrix}$

After that, by applying LMI toolbox in MATLAB for the proposed linear matrix inequalities (LMI.4), (LMI.5) and (LMI.6), we directly obtain the symmetric positive definite matrices C_i , P_i and the matrices K_i^p as follows:

(i) For the subsystem \mathbf{E}_1 , we have

$$\begin{aligned} \mathbf{C}_1 &= \begin{bmatrix} 21.8174 & 1.3860 \\ 1.3860 & 92.7029 \end{bmatrix} \\ \mathbf{P}_1 &= \begin{bmatrix} 0.0459 & -0.0007 \\ -0.0007 & 0.0108 \end{bmatrix} \\ K_1^1 &= \begin{bmatrix} 0.1067 & 0.2151 \end{bmatrix} \\ K_1^2 &= \begin{bmatrix} 0.2983 & 0.0581 \end{bmatrix} \\ K_1^3 &= \begin{bmatrix} 0.3112 & -0.0300 \end{bmatrix} \\ K_1^4 &= \begin{bmatrix} 0.3358 & -0.0330 \end{bmatrix}. \end{aligned}$$

(ii) For the subsystem \mathbf{E}_2 , we have

$$\begin{split} \mathbf{C}_2 &= \begin{bmatrix} 18.5841 & 1.0680 \\ 1.0680 & 91.7758 \end{bmatrix} \\ \mathbf{P}_2 &= \begin{bmatrix} 0.0538 & -0.0006 \\ -0.0006 & 0.0109 \end{bmatrix} \\ K_2^1 &= \begin{bmatrix} 0.0605 & 0.2203 \end{bmatrix} \\ K_2^2 &= \begin{bmatrix} 0.2944 & 0.0617 \end{bmatrix} \\ K_2^3 &= \begin{bmatrix} 0.3172 & -0.0280 \end{bmatrix} \\ K_2^4 &= \begin{bmatrix} 0.3598 & -0.0315 \end{bmatrix}. \end{split}$$

Therefore, we can see that the system of linear matrix inequalities (LMI.1), (LMI.2) and (LMI.3) of Theorem 5.1 has a feasible solution, which implies that the unstable malware-free equilibrium of the fractional network-based SIRS epidemic model can be stabilized under the fuzzy state-feedback controller $\mathbf{u}(t) = \begin{bmatrix} \mathbf{u}_1(t) & \mathbf{u}_2(t) \end{bmatrix}^{\mathsf{T}}$, where the control $\mathbf{u}_i(t)$ is defined by the formula (15).

6. Conclusions

This work proposes a controlled fractional network-based SIRS epidemic model with saturated treatment function and designs a fuzzy parallel distributed compensation (PDC) controller $\mathbf{u}(t)$ to study the malware infection controlling on complex heterogeneous network. In order to get a better description of real-world scenario that the number of infected individuals reach a saturation level and exceed the cure capacity, a saturated treatment function is used instead of a linear ones. After that, we carry out a detailed study on epidemiological characteristic of the proposed network-based epidemic model. The first epidemiological factor we concentrate on is the basic reproduction number \mathfrak{R}_0 , which can be analytically evaluate by using next-generation matrix method. Next, we use this number as a threshold value to investigate the asymptotic stability of malware-free equilibrium and the presence of endemic equilibrium on complex heterogeneous network. After that, we determine the condition for which the backward bifurcation phenomena at $\mathfrak{R}_0 = 1$ occurs. Finally, in order to deal with the stabilization problem of the unstable malware-free equilibrium, we propose to use the approach of interconnected Takagi-Sugeno fuzzy systems and give some necessary conditions in LMIs form to solve the desired stabilization problem. By using LMI Toolbox to solve the obtained linear matrix inequalities, the proposed stabilization criteria can be conveniently used to implement state-feedback controller $\mathbf{u}(t)$ and guarantees the asymptotic stability of malware-free equilibrium. A further problem in regard to combining asymptotic stability with observability of the network-based SIRS epidemic model has not been considered in this work. In addition, due to the vagueness and incomplete of model's parameters in reality, there is an open question that how to establish the criteria for the stabilizability, reachability or observability of the proposed epidemic model with uncertainties. We think that these problems should be of considerably interest and can be done in our future studies.

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