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# 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 well-mixed, 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 taken 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 [29] 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 [13], Huo et al. proposed a three-compartmental epidemic model with susceptible, infected and recovered states to describe the virus infection on scale-free network. Firstly, the basic reproduction number $\mathcal{R}_0$ was evaluated to study

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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 [17] introduced the saturated treatment function in their work. After formulating a network-based SIRS epidemic model with saturation, the paper [17] 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 [19], 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 [21] 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 novelty 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. [32], 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 [10] 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 [11]), 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 [4] 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. [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 $R_0^\alpha$ 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 [12, 15, 28], 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 [5, 6, 7, 9]. 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. [5, 6], Mahato et. al. [22], Mondal et. al. [24], Nandi et. al. [26].

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 real-world 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, 33], 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 $\sigma_k$, 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 [1] 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 $\mathbb{P}(k_i) = \dfrac{k_i}{\sum_j k_j}$, 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 $\sigma$ as a function of the available malware program. In particular, this parameter is proposed to describe through the following fuzzy set:

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

Here, we can see that there always exists a lower threshold $\tau_m$ for the malware propagation, that is, the disease infection occurs only if the amount of malware program on the network must exceed $\tau_m$, otherwise, the chance of transmission is negligible. In addition, the value of $\tau_m$ 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 $\tau_0$, beyond which the transmission rate reaches the maximum value $\sigma(\tau) = 1$. From $\tau_m$ to $\tau_0$, the transmission rate is assumed to vary linearly. Furthermore, assume that the malware load has an upper bound, say $\tau_M$. 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 $\tau_m, \tau_0, \tau_M$, the malware load is expressed by using fuzzy numbers (see Figure 1). This approach can be found in [24, 26].

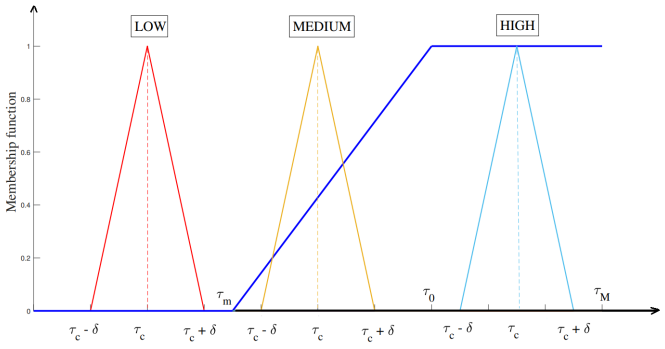


Figure 1: The membership function of fuzzy transmission rate $\sigma$ and linguistic variables of malware load

## 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.

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

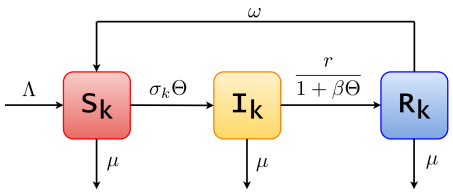


Figure 2: The flowchart of malware propagation among compartments: Susceptible (S), Infectious (I), Recovered (R)

equations:

$$\begin{cases} {}_0^C\mathfrak{D}_t^\beta S_k(t) = \Lambda - \sigma_k(\tau) S_k(t)\Theta(t) - \mu S_k(t) + \omega R_k(t) \\ {}_0^C\mathfrak{D}_t^\beta I_k(t) = \sigma_k(\tau) S_k(t)\Theta(t) - \mu I_k(t) - \dfrac{r I_k(t)}{1+\gamma\Theta(t)} \\ {}_0^C\mathfrak{D}_t^\beta R_k(t) = \dfrac{r I_k(t)}{1+\gamma\Theta(t)} - (\mu+\omega) R_k(t), \end{cases} \tag{1}$$

with the initial conditions

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

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 7.2 in Appendix). The meanings of the model's parameters are given in Table 1:

Table 1: The model's parameters

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

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 $\gamma$ 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 (1) with the initial condition (2) is defined for all $t > 0$ and $k = 1, 2, \ldots, n$. 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

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

Denote

$$\mathbf{x}(t) = (S_1(t), I_1(t), R_1(t), \ldots, S_n(t), I_n(t), R_n(t))^\top$$
$$\Sigma^+ = \left\{ \mathbf{x}(t) \in \mathbb{R}_+^{3n} : S_k + I_k + R_k \leq \frac{\Lambda}{\mu},\ k = \overline{1,n} \right\}.$$

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 $t \geq 0$.

**Lemma 2.1.** *Assume that* $\mathbf{x}(t)$ *is a solution of the fractional network-based SIRS epidemic model (1) with the initial condition (2) and* $\mathbf{x}(0)$ *belongs to* $\Sigma^+$*. Then, for all* $t > 0$*, the solution* $\mathbf{x}(t)$ *belongs to* $\Sigma^+$*.*

*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 $0 \leq t < t_0$ and $S_k(t) < 0$ for all $t_0 < t \leq t_0 + \varepsilon_0$ with sufficiently small $\varepsilon_0 > 0$. Then, we consider two following cases:
**Case 1:** If $I_k(t) \geq 0$ for all $t \geq 0$ then we have

$$ {}_0^C\mathfrak{D}_t^\beta R_k(t) = \frac{rI_k(t)}{1+\gamma\Theta(t)} - (\omega+\mu)R_k(t) \geq -(\omega+\mu)R_k(t). $$

Then, by applying fractional comparison principle (Lemma 10, [17]), it implies that the function

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

for all $t \geq 0$. 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 7.8 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 $t_0 \leq \xi \leq t_0 + \varepsilon_0$ and $0 < \varepsilon_0 \ll 1$ is small enough such that ${}_0^C\mathfrak{D}_t^\beta S_k(t)|_{t=\xi} \geq 0$. This means $S_k(t_0+\varepsilon_0) > 0$, which contradicts to 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 $t_1 < t \leq t_1 + \varepsilon_1$ with sufficiently small $\varepsilon_1 > 0$. Then our proof is proceeded in two following sub-cases:
*Sub-case 1:* If $t_1 \geq t_0$ 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 the 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 ${}_0^C\mathfrak{D}_t^\beta S_k(t)|_{t=\overline{\xi}} \geq 0$ with $\overline{\xi} \in [t_1, t_1+\varepsilon_1]$. Next, by using Lemma 7.8 for $a = 0$ and $t = t_1 + \varepsilon_1$, we obtain

$$ I_k(t_1+\varepsilon_1) = I_k(0) + \frac{\varepsilon_1^\beta}{\Gamma(\beta)} {}_0^C\mathfrak{D}_t^\beta I_k(t)|_{t=\overline{\xi}}. $$

It implies that $I_k(t_1+\varepsilon_1) > 0$, which contradicts to our assumption. Therefore, we can conclude that $S_k(t) > 0$ is always positive for all $t \geq 0$. 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 $t \geq 0$ and $k = \overline{1,n}$.

Next, by using the second assumption, we have $N_k(0) = S_k(0) + I_k(0) + R_k(0) \leq \frac{\Lambda}{\mu}$. By summing up all fractional differential equations of the system (1), we immediately obtain

$$ {}_0^C\mathfrak{D}_t^\beta N_k(t) = \Lambda - \mu N_k(t). \tag{3} $$

By applying Example 4.9 in [14], 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 7.5 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

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

which means that $\Sigma^+$ is a positively invariant set for the fractional network-based epidemic model (1). □

# 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 (1) admits a malware-free equilibrium (MFE) $\mathbf{E}_0 = (\underbrace{\frac{\Lambda}{\mu}, 0, 0, \ldots, \frac{\Lambda}{\mu}}_{3n}, 0, 0)$. 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 (1). 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. [2]. 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 (1), 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 $\mu I_k(t) + \dfrac{rI_k(t)}{1+\gamma\Theta(t)}$.

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 $\mathcal{V} = (\mu + r)\mathtt{I}_n$, where $\mathtt{I}_n$ is the $n \times n$ identity matrix. The basic reproduction number $\mathcal{R}_0$ is then the largest eigenvalue of the matrix $\mathcal{F}\mathcal{V}^{-1}$ 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 $\mathcal{R}_0 = \dfrac{\sigma(\tau)\Lambda\langle k^2\rangle}{\mu(r+\mu)\langle k\rangle}$, 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 $\dfrac{\langle k^2\rangle}{\langle k\rangle}$. 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 (1).

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

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

*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* $\gamma < \dfrac{\sigma(\tau)}{\mu+\omega}$ *then the endemic equilibrium* $\mathbf{E}_*$ *of the network-based epidemic model (1) is unique.*

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

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

where $\Theta = \dfrac{1}{\langle k\rangle}\sum_{i=1}^{n} i\mathbb{P}(i)I_i$. Next, by expressing the variables $S_k$, $R_k$ in the two first equations of the system (4) in the terms of $I_k$, we immediately get

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

After that, we substitute the expressions of $S_k^*$ and $R_k^*$ into the last equation of the system (4), we receive

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

or equivalently, $I_k^* = \dfrac{\Lambda\sigma_k(\tau)\Theta^*}{\mu\left[\mu + \sigma_k(\tau)\Theta^* + \frac{r\sigma_k(\tau)\Theta^*}{(\mu+\omega)(1+\gamma\Theta^*)} + \frac{r}{1+\gamma\Theta^*}\right]}$. Next, by substituting $I_k^*$ into the expression of the function $\Theta(t)$, the equation $\Theta^* = \dfrac{1}{\langle k\rangle}\sum_{i=1}^{n} i\mathbb{P}(i)I_i^*$ becomes the following self-consistency equation

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

It should be noted that the self-consistency equation (5) always admits the trivial solution $\Theta \equiv 0$. Now, we aim to determine a sufficient condition for which the equation (5) 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 $(0, 1)$.
- $f(0) = \dfrac{\Lambda\sigma(\tau)}{\mu(r+\mu)\langle k\rangle}\sum_{k=1}^{n} k^2\mathbb{P}(k) = \mathcal{R}_0.$
- For each $\Theta \in [0, 1]$, we have $f(\Theta) < \dfrac{1}{\langle k\rangle}\sum_{i=1}^{n} \dfrac{\Lambda\sigma(\tau)i^2\mathbb{P}(i)}{\mu^2} = \tilde{\mathcal{R}}_0.$

- 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 (5) is the solution of the following equation

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

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 (6) 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 (6) has at least one solution $\Theta \in (0,1)$, that is also the non-trivial solution of the equation (5). As a consequence, the solution $\Theta^* \in (0,1)$ of the self-consistency equation (5) 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

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

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 $\gamma \leq \dfrac{\sigma(\tau)}{\mu+\omega}$ then the derivative $\dfrac{d}{d\Theta}f(\Theta) < 0$ for all $\Theta \in [0,1]$ and hence, the equation (6) has a unique solution $\Theta \in (0,1)$. The proof is completed. □

# 4 The asymptotic behavior of malware-free equilibrium $\mathbf{E}_0$

## 4.1 The local asymptotic stability

**Theorem 4.1.** *The malware-free equilibrium $\mathbf{E}_0$ of the fractional network-based SIRS epidemic model (1) 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 $\mathrm{J}(\mathbf{E}_0)$. Let us consider the Jacobi matrix at $\mathbf{E}_0$ of the epidemic model (1) in the following form

$$\mathrm{J}(\mathbf{E}_0) = \begin{bmatrix} \mathrm{J}_{11} & \mathrm{J}_{12} & \cdots & \mathrm{J}_{1n} \\ \mathrm{J}_{21} & \mathrm{J}_{22} & \cdots & \mathrm{J}_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ \mathrm{J}_{n1} & \mathrm{J}_{n2} & \cdots & \mathrm{J}_{nn} \end{bmatrix}_{3n\times 3n},$$

where $\mathrm{J}_{kk}$, $\mathrm{J}_{kk}$ are $3\times 3$-square matrices given by

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

for each $k, j = \overline{1,n}$. Then, by applying the mathematical induction principle, the characteristic polynomial w.r.t. the Jacobi matrix $\mathrm{J}(\mathbf{E}_0)$ 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 [3], 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 $\mathrm{J}(\mathbf{E}_0)$ satisfy

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

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 $\mathcal{P}\left(\tilde{\lambda}\right)$ 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 $\mathrm{J}(\mathbf{E}_0)$ 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 [3], 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 4.1 is based on linearization method and stability criteria for fractional differential system in Theorem 7.20 in [3] related to modulus of eigenvalue's arguments. By applying linearization method, we get that the Jacobi matrix* $\mathrm{J}(\mathbf{E}_0)$ *is a square matrix of order* $3n$ *and then, the mathematical induction principle follows that the matrix* $\mathrm{J}(\mathbf{E}_0)$ *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 (1). 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 [18], we can conclude that the malware-free equilibrium* $\mathbf{E}_0$ *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 (1). For this aim, we denote a threshold value

$$\tilde{\mathcal{R}}_0 = \frac{\Lambda\sigma(\tau)\langle k^2\rangle}{\mu^2\langle k\rangle}.$$

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 network-based SIRS epidemic model (1) 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 (1). 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

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

According to Remark 7.7, 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 $S_k = \dfrac{\Lambda}{\mu}$. In addition, based on the non-negativeness of the solution $\mathbf{x}(t)$ stated in Lemma 2.1, 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 7.6, we receive

$$\begin{aligned}{}_0^C\mathfrak{D}_t^\beta \mathbf{V}(\mathbf{x}(t)) &= \frac{1}{\langle k\rangle}\sum_{k=1}^{n} k\mathbb{P}(k)\left({}_0^C\mathfrak{D}_t^\beta \Phi(S_k) + {}_0^C\mathfrak{D}_t^\beta I_k + {}_0^C\mathfrak{D}_t^\beta R_k\right)\\ &= \frac{1}{\langle k\rangle}\sum_{k=1}^{n} k\mathbb{P}(k)\left[\left(1-\frac{\Lambda}{\mu S_k}\right){}_0^C\mathfrak{D}_t^\beta S_k + {}_0^C\mathfrak{D}_t^\beta I_k + {}_0^C\mathfrak{D}_t^\beta R_k\right],\end{aligned}$$

where

$$\begin{aligned}\left(1-\frac{\Lambda}{\mu S_k}\right){}_0^C\mathfrak{D}_t^\beta S_k &= 2\Lambda - \sigma_k(\tau)S_k\Theta - \mu S_k + \omega R_k - \frac{\Lambda^2}{\mu S_k} + \frac{\sigma_k(\tau)\Lambda\Theta}{\mu} - \frac{\omega\Lambda R_k}{\mu S_k}\\ &= -\frac{\mu}{S_k}\left(\frac{\Lambda^2}{\mu^2} - 2S_k\frac{\Lambda}{\mu} + S_k^2\right) + \frac{\sigma_k(\tau)\Lambda\Theta}{\mu} - \sigma_k(\tau)S_k\Theta + \omega R_k\left(1-\frac{\Lambda}{\mu S_k}\right),\end{aligned} \tag{7}$$

and

$${}_0^C\mathfrak{D}_t^\beta I_k + {}_0^C\mathfrak{D}_t^\beta R_k = \sigma_k(\tau)S_k\Theta - \mu I_k - (\mu+\omega)R_k. \tag{8}$$

By combining two inequalities (7) and (8), 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 $-\dfrac{\mu}{S_k}\left(\dfrac{\Lambda}{\mu} - S_k\right)^2 \le 0$ and $\omega R_k\left(1 - \dfrac{\omega+\mu}{\omega} - \dfrac{\Lambda}{\mu S_k}\right) \le 0$. Hence, we have

$$\begin{aligned}{}_0^C\mathfrak{D}_t^\beta \mathbf{V}(\mathbf{x}(t)) &\le \frac{1}{\langle k\rangle}\sum_{k=1}^{n} k\mathbb{P}(k)\left[\frac{\sigma_k(\tau)\Lambda\Theta}{\mu} - \mu I_k\right]\\ &= \mu\Theta\left[\frac{\sigma(\tau)}{\mu^2\langle k\rangle}\sum_{k=1}^{n}\Lambda k^2\mathbb{P}(k) - 1\right]\\ &= \mu\Theta(\tilde{\mathcal{R}}_0 - 1).\end{aligned} \tag{9}$$

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

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

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 [11], 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 [8, 12, 16, 19, 20].*

**Remark 4.5.** *By the inequality (9), 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* $\mathcal{R}_0 \leq \dfrac{\mu}{\mu + r} < 1$ *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* $\mathbf{E}_0$*.*

# 5 Applications

Hand-Foot-Mouth Disease (HFMD) is a common infectious disease for children, especially children are under 5 years old. From December $19^{th}$, 2020 to January $18^{th}$, 2021, Vietnam had $2,901$ cases of HFMD, that is 2.3 times higher than the same period last year, see [34]. In this section, we will apply the proposed fractional network-based SIRS epidemic model (1) 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.

Table 2: The used parameters in the SIRS epidemic model

| **Parameter** | **Value** | **Parameter** | **Value** |
|---|---|---|---|
| $\Lambda$ | 0.12 | $\mu$ | 0.05 |
| $\sigma$ | 0.1 | $\omega$ | 0.06 |
| $r$ | 0.8 | $\gamma$ | 4 |

## 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 $\tau$, 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 $\tau$ 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 $\tau$. 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 $\tau$. 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 $\tau$. 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. [25], the sensitivity index of a quantity $\mathbf{x}$ depending on a parameter $\lambda$ can be determined by $\Upsilon_\lambda^{\mathbf{x}} = \dfrac{\partial \mathbf{x}}{\partial \lambda} \times \dfrac{\lambda}{\mathbf{x}}$. 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

$$\Upsilon_{\sigma(\tau)}^{\mathcal{R}_0} = 1, \qquad \Upsilon_{\Lambda}^{\mathcal{R}_0} = 1, \qquad \Upsilon_{r}^{\mathcal{R}_0} = -\frac{r}{\mu + r}, \qquad \Upsilon_{\mu}^{\mathcal{R}_0} = -\frac{(2\mu + r)}{\mu + r}, \qquad \Upsilon_{\frac{\langle k^2\rangle}{\langle k\rangle}}^{\mathcal{R}_0} = 1.$$

**Remark 5.1.** *We can see that the threshold value $\mathcal{R}_0$ is the most sensitive with the natural death rate $\mu$. 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 $\sigma$ 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.*

Table 3: The sensitivity indices of model's parameters

| No | Parameter | Description | Sensitivity index |
|---|---|---|---|
| 1 | $\sigma(\tau)$ | The fuzzy transmission rate | $+1$ |
| 2 | $r$ | The cure rate | $-\frac{16}{17}$ |
| 3 | $\mu$ | The natural death rate | $-\frac{18}{17}$ |
| 4 | $\Lambda$ | The natural birth rate | $+1$ |
| 5 | $\frac{\langle k^2\rangle}{\langle k\rangle}$ | The parameter of network structure | $+1$ |

*In addition, for convenience, we present the sensitivity of parameters in Figure 3.*

In the following, we discuss the change of the basic reproductive number $\mathcal{R}_0$ with respect to viral load $\tau$. Let us choose the normalized values of threshold quantities $\tau_m, \tau_0, \tau_M$ by $0.25, 0.65, 1$, respectively. Then, the transmission rate $\sigma$ 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

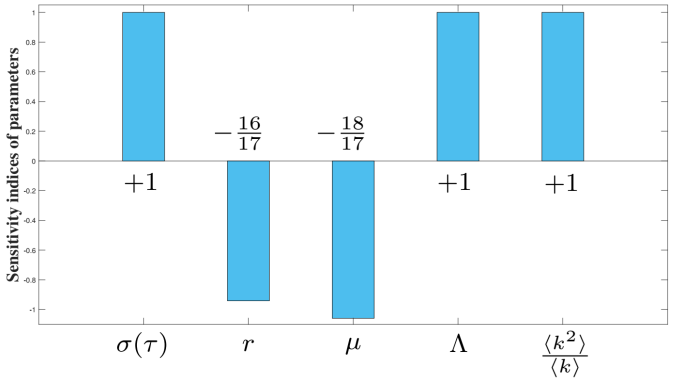


Figure 3: The sensitivity indices of the model's parameters

et al. [23] 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 $\alpha$ (level-sets index) and $\alpha_u$ (relative-distance-measure variable, see [23] 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 4.

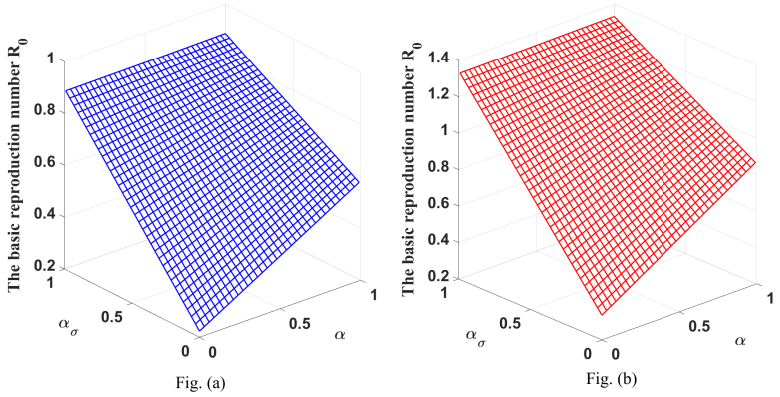


Figure 4: The relative change of the basic reproduction number $\mathcal{R}_0$ w.r.t. malware load: Fig. (a) $\sigma = 0.1$ and Fig. (b) $\sigma = 0.15$

Figure 4 (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 $\tau$. This phenomena will be studied in our next work. A similar result was discussed in [26].

# 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 (1) 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, 14] 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 $\beta$ is defined by*

$$ {}_a\mathfrak{I}_t^{\beta} f(t) = \frac{1}{\Gamma(\beta)} \int_a^t (t-s)^{\beta-1} f(s) ds, \quad t \in [a, b]. $$

**Definition 7.2.** [3] *Let $m := \lceil \beta \rceil$ be the smallest integer greater than or equal to $\beta$. The Caputo fractional derivative of order $\beta$ 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 $f = (f_1, f_2, \ldots, f_n)^{\top}$ is defined component-wise by*

$$ {}_a^C\mathfrak{D}_t^{\beta} f(t) = \left( {}_a^C\mathfrak{D}_t^{\beta} f_1(t), {}_a^C\mathfrak{D}_t^{\beta} f_2(t), \ldots, {}_a^C\mathfrak{D}_t^{\beta} f_n(t) \right). $$

Consider the initial value problem for the following fractional differential equations (FDS)

$$ {}_0^C\mathfrak{D}_t^{\beta} \mathbf{x}(t) = A\mathbf{x}(t) + f(\mathbf{x}(t)), \qquad t > 0 \tag{10} $$

subject to the initial conditions

$$ \mathbf{x}(0) = \mathbf{x}_0, \tag{11} $$

where $A \in \mathrm{Mat}_{n\times n}(\mathbb{R})$ 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 (10)-(11). Next, let $\varphi : [0, \infty) \to \mathbb{R}^n$ be a solution of the initial value problem (10)(11). Now, we recall from Definition 7.2 in [3] the notions of stability and asymptotic stability of trivial solution of (10).

**Definition 7.3.** [3] *The trivial solution $\mathbf{x}^* \equiv \overline{0}$ of the FDS (10) 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 (10)(11) satisfies $\|\varphi(t, \mathbf{x}_0)\| < \varepsilon$ for all $t \geq 0$ 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 (10) 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 $\mathbb{E}_{\alpha_1,\alpha_2}(x) = x\mathbb{E}_{\alpha_1,\alpha_1+\alpha_2}(x) - \dfrac{1}{\Gamma(\alpha_2)}$, where $\mathbb{E}_{\alpha_1,\alpha_2}(z)$ is the Mittag-Leffer functions of two parameters $\alpha_1$ and $\alpha_2$ (see [14]).*

**Lemma 7.6.** [16] *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.** [27] *Assume that $\beta \in (0,1]$ and both the function $\Phi$ and its fractional derivative ${}_0^C\mathfrak{D}_t^\beta\Phi$ belong to the space $C[a,b]$. Then we have $\Phi(t) = \Phi(a) + \dfrac{1}{\Gamma(\beta)} {}_a^C\mathfrak{D}_t^\beta\Phi(\xi)\,(t-a)^\beta$, for $a \leq \xi \leq t$ and $t \in [a,b]$.*

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