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Dynamical Analysis of a Fractional SIRS Model on Complex Heterogeneous Networks

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Abstract: In this paper, a fractional SIRS model on heterogeneous complex networks is introduced. The asymptotic local and global stability of equilibrium points are studied, numerical simulation is used to support our theoretical results and show the effect of fractional order q and the influence of connectivity between individuals which represented as $\psi(t)$.

Keywords: Fractional order SIRS model; heterogeneous network; local stability; global stability; numerical simulation.

1 Introduction

The reasons of infectious disease are pathogens or parasites. The infection is the most important factor to spread disease in populations, infection caused by the connection between individuals in a population (individuals mean humans and animals). The average connection between infected individuals and healthy individuals specify the acceleration or slow down the disease, meanly if the average connection is very high, then the disease will be epidemic [1]. Classical model which describe diseases ignore a very important factor that effect directly in spreading diseases, this factor is a connection between individuals, meanly if we have a lot of links between individuals in the small region, the diseases spread more rapidly than fewer links. Each individual has links that differs from other individuals ([1], [2]). We mean that the infection transfer from an infected individual to susceptible individuals by these links and number of links affect directly in our modeling of spreading diseases so that we improve the classical model to networked model. Network means that individuals as nodes or vertices and links between individuals as the connection between them. We have two types of networks (homogeneous network and heterogeneous network) the main difference between them that homogeneous network considers the connection between individuals is equal to the average connectivity between them, other hands heterogeneous network more reality than homogeneous network because of each individual has owner links different from other ones. The reason for using homogeneous networks that study general behavior of these diseases and put some conditions to control this spreading, meanly take general vision for the dynamics of these models (equilibrium points, local and global stability,... etc). In our previous study [3], we study the dynamics of the fractional model in the homogeneous network of (SIRS) model and the effect of fractional order appears on stability and numerical simulations. In this paper, we improve our study to the dynamics of the fractional model in a heterogeneous network of (SIRS) model. The heterogeneous network has several types to represent a random network, our study focuses on a random scale-free network, which follows Barabsi-Albert (BA) model ([3]-[5]).

We consider a fractional (SIRS) model:

$$\begin{aligned}
\frac{d^q x_k}{dt} &= \mu - \beta k x_k \psi(t) + \delta z_k - (\alpha+\mu)x_k, \\
\frac{d^q y_k}{dt} &= \beta k x_k \psi(t) - (\gamma+\eta)y_k, \\
\frac{d^q z_k}{dt} &= \gamma y_k - (\delta+\mu)z_k + \alpha x_k,
\end{aligned} \tag{1}$$

where x_k, y_k, z_k are density of susceptible, infected and recovered individuals of degree k, respectively.

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Susceptible individuals infected from connection with infected individuals at rate β, an infected individual becomes healthy at rate γ and recovered individuals return to susceptible individuals at rate δ. Susceptible individual is vaccinated at rate α. μ represents to birth rate and death rate without a disease. If the disease will spread, infected individuals die at rate η, that mean if $\eta > \mu$, the disease become very dangerous and epidemic. According to uncorrlation of the connection between nodes in network. All rates are positive constants and $0 < q \leq 1$ is fractional order.

We denote

$$\psi(t) = \frac{\sum_{i=1}^{n} ip(i)y_i}{\langle k \rangle},$$

is the probability of a contact pointing to an infected individual, where $p(i)$ is distribution function that describe the connection between individuals, $\langle k \rangle = \sum_{i=1}^{n} ip(i)$ is the average degree of network, n is the maximum positive integer number of contact in each individual.

We reduce system (1) to

$$\begin{aligned} \frac{d^q x_k}{dt} &= \lambda - \beta k x_k \psi(t) - \delta\omega y_k - (\delta+\alpha+\mu)x_k, \\ \frac{d^q y_k}{dt} &= \beta k x_k \psi(t) - (\gamma+\eta)y_k, \end{aligned} \tag{2}$$

where $z_k = 1 - x_k - \omega y_k$ and $\lambda = \mu + \delta$, $\omega = \frac{\eta}{\mu}$.

The dynamics of solutions of system (2) will be in the bounded region:

$$\Omega = \{(x_1, y_1, ..., x_k, y_k) \in R^{2k}, 0 \leq x_k, y_k \leq 1, x_k + y_k \leq 1, 1 \leq k \leq n\}, \tag{3}$$

we can easily show that region Ω is positively invariant.

All definitions, theorems of fractional order are considered in ([4], [6]-[12]) and theorems of local and global asymptotic stability are considered in ([13]-[18]).

In the rest of the paper, we study how a fractional-order q effects on local and global stability of equilibrium points, (which was calculated in section 2) in section 3, section 4 and section 5 are numerical results which support our theories in previous sections.

2 Equilibrium points

We study equilibrium points of system (2)

$$\begin{aligned} 0 &= \lambda - \beta k x_k^*(t)\psi^*(t) - \delta\omega y_k^* - (\delta+\alpha+\mu)x_k^*, \\ 0 &= \beta k x_k^*(t)\psi^*(t) - (\gamma+\eta)y_k^*. \end{aligned}$$

We have two equilibrium points, disease-free point E_0 and endemic equilibrium point E_1.

Firstly, we obtain E_0 at $y_k^* = 0, (k = 1, 2, ..., n)$. Substituting them into (2), we get

$$E_0 = \left\{ \left(\frac{\lambda}{\delta+\mu+\alpha}, 0 \right) \right\}_k.$$

Secondly, we obtain $E_1 = (x_k^*, y_k^*)$ at the presence of disease that mean $y_k^* \neq 0, (k = 1, 2, ..., n)$, we have

$$E_1 = \left\{ \left(\frac{\lambda}{\beta k \psi^*(t) + \frac{\delta\omega\beta k\psi^*(t)}{\gamma+\eta} + \delta + \mu + \alpha}, \frac{\beta k \psi^*(t)\lambda}{\beta k\psi^*(t)(\gamma+\eta+\delta\omega) + (\delta+\mu+\alpha)(\gamma+\eta)} \right) \right\}_k.$$

Theorem 2.1. Define

$$R_0 = \frac{\langle k^2 \rangle \beta\lambda}{\langle k \rangle (\eta+\gamma)(\delta+\mu+\alpha)},$$

where $\langle k^2 \rangle = \sum_{i=1}^{n} i^2 p(i)$. Then endemic point E_1 is non-trivial unique solution under condition $R_0 > 1$.

Proof. Firstly, we obtain the self-consistency equality

$$\psi^* = \frac{\sum_i ip(i)y_i^*}{\langle k \rangle} = \frac{\sum_i ip(i)\left(\frac{\beta i\psi^*(t)\lambda}{\beta i\psi^*(t)(\gamma+\eta+\delta\omega)+(\delta+\mu+\alpha)(\gamma+\eta)}\right)}{\langle k \rangle}.$$

Now, we define function

$$F(\psi) = \frac{\sum_i ip(i)\left(\frac{\beta i\psi(t)\lambda}{\beta i\psi(t)(\gamma+\eta+\delta\omega)+(\delta+\mu+\alpha)(\gamma+\eta)}\right)}{\langle k \rangle} - \psi \tag{4}$$

to support the proof of the existence and uniqueness of epidemic equilibrium point E_1. We can easily see that $\psi = 0$ is the solution of (4) and

$$F(1) = \frac{\sum_i ip(i)\left(\frac{\beta i\lambda}{\beta i(\gamma+\eta+\delta\omega)+(\delta+\mu+\alpha)(\gamma+\eta)}\right)}{\langle k \rangle} - 1 < 0$$

where $\eta > \mu$, that mean there exist nontrivial solution of system (1) where $0 < \psi < 1$, we correspond the following inequality

$$\frac{dF(\psi)}{d\psi}|_{\psi=0} = \frac{d}{d\psi}\left(\frac{\sum_i ip(i)\left(\frac{\beta i\psi(t)\lambda}{\beta i\psi(t)(\gamma+\eta+\delta\omega)+(\delta+\mu+\alpha)(\gamma+\eta)}\right)}{\langle k \rangle} - \psi \right)|_{\psi=0} =$$

$$(R_0 - 1) > 0, \text{ if } R_0 > 1,$$

therefore, the system (1) has a unique endemic equilibrium point E_1.

3 Local stability of equilibrium points

3.1 Local stability of free disease point E_0

Theorem 3.1.1 If $R_0 < 1$, then the disease-free equilibrium E_0 is locally asymptotically stable, but unstable if $R_0 > 1$.

proof. We linearized system (2) at E_0

$D^q(x_1,...,x_n,y_1,...,y_n)^T = J(E_0)(x_1,...,x_n,y_1,...,y_n)^T$, where

$$J(E_0) = \begin{pmatrix} M_{11} & ... & M_{1n} \\ \vdots & \ddots & \vdots \\ M_{n1} & ... & M_{nn} \end{pmatrix}_{2n\times 2n}, \quad (5)$$

$$M_{11} = \begin{pmatrix} -(\delta+\mu+\alpha) & -\zeta_1-\delta\omega \\ 0 & \zeta_1-(\gamma+\eta) \end{pmatrix}, M_{n1} = \begin{pmatrix} 0 & -\zeta_{n1} \\ 0 & \zeta_{n1} \end{pmatrix},$$

$$M_{1n} = \begin{pmatrix} 0 & -\zeta_{1n} \\ 0 & \zeta_{1n} \end{pmatrix}, M_{nn} = \begin{pmatrix} -(\delta+\mu+\alpha) & -\zeta_n-\delta\omega \\ 0 & \zeta_n-(\gamma+\eta) \end{pmatrix},$$

and $\zeta_i = \frac{\beta\lambda i^2 p(i)}{(\delta+\mu+\alpha)\langle k\rangle}, \zeta_{ij} = \frac{\beta\lambda ijp(i)}{(\delta+\mu+\alpha)\langle k\rangle}, i\neq j = 1,2,...,n.$

The characteristic polynomial of free disease point is

$$(\rho+(\delta+\mu+\alpha))^n(\rho+\eta+\gamma)^{n-1}(\rho-\sum_{i=1}^{n}\zeta_i-\eta-\gamma)$$
$$= (\rho+(\delta+\mu+\alpha))^n(\rho+\eta+\gamma)^{n-1}(\rho-(\eta+\gamma)(R_0-1)) = 0$$

we have n eigenvalues equal to $-(\delta+\mu+\alpha)<0$, $n-1$ eigenvalues equal to $-(\eta+\gamma)<0$, and the last eigenvalue is $(\eta+\gamma)(R_0-1)<0$ if $R_0<1$.

Then the disease-free equilibrium E_0 is locally asymptotically stable if $R_0<1$ ([6], [7]).

3.2 Local stability of endemic disease point E_1

Theorem 3.2.1 If $R_0>1$, then the endemic equilibrium E_1 is locally asymptotically stable.

proof. We construct Jacobian matrix of E_1 :

$$J(E_1) = \begin{pmatrix} -(a+b_1) & ... & 0 & -(\xi_1 g_1+\delta\omega) & ... & -\xi_1 g_n \\ \vdots & \ddots & \vdots & \vdots & \ddots & \vdots \\ 0 & ... & -(a+b_n) & -\xi_n g_1 & ... & -(\xi_n g_n+\delta\omega) \\ b1 & ... & 0 & (\xi_1 g_1-b) & ... & \xi_1 g_n \\ \vdots & \ddots & \vdots & \vdots & \ddots & \vdots \\ 0 & ... & b_n & \xi_n g_1 & ... & (\xi_n g_n-b) \end{pmatrix}_{2n\times 2n}, \quad (6)$$

where $a=\delta+\alpha+\mu, b=\gamma+\eta, b_i=\beta i\psi_i^*(t), \xi_i = \beta i x_i^*(t), g_i = \frac{ip(i)}{\langle k\rangle}, i=1,2,...,n.$

The characteristic polynomial of endemic point is

$$(\rho+a)^n\prod_{i=1}^{n}(\rho+b+\Psi_i(\rho))(1-\sum_{i=1}^{n}\frac{\xi_i g_i}{\rho+b+\Psi_i(\rho)}) = 0,$$

where $\Psi_i(\rho) = \frac{\rho+b+\delta\omega}{\rho+a}b_i$.

We obtain n eigenvalues $-a<0$. Let

$$F(\rho) = \prod_{i=1}^{n}(\rho+b+\Psi_i(\rho))(1-\sum_{i=1}^{n}\frac{\xi_i g_i}{\rho+b+\Psi_i(\rho)})$$
$$= (\rho+b+\Psi_1(\rho))(\rho+b+\Psi_2(\rho))...(\rho+b+\Psi_n(\rho)) -$$
$$\xi_1 g_1(\rho+b+\Psi_2(\rho))(\rho+b+\Psi_3(\rho))...(\rho+b+\Psi_n(\rho)) -$$
$$\xi_2 g_2(\rho+b+\Psi_1(\rho))(\rho+b+\Psi_3(\rho))...(\rho+b+\Psi_n(\rho))... -$$
$$\xi_n g_n(\rho+b+\Psi_1(\rho))(\rho+b+\Psi_2(\rho))...(\rho+b+\Psi_{n-1}(\rho))$$
$$= 0.$$

Since $\Psi_i(\rho)$ is increasing function $(\Psi_n(\rho)>\Psi_1(\rho))$ and $F(-b-\Psi_1(\rho))<0, F(-b-\Psi_2(\rho))>0$

That mean we have at least one root in the interval $[-b-\Psi_2(\rho),-b-\Psi_1(\rho)]$, for general we have at least one root in $[-b-\Psi_{i+1}(\rho),-b-\Psi_i(\rho)]$.

Mainly, we have $n-1$ roots in $[-b-\Psi_n(\rho),-b-\Psi_1(\rho)]$.

Also $F(-b-\Psi_1(\rho))<0$ and

$$F(0) = \prod_{i=1}^{n}(b+\Psi_i(0)(1-\sum_{i=1}^{n}\frac{\xi_i g_i}{\rho+b+\Psi_i(\rho)})$$
$$= \prod_{i=1}^{n}(b+\frac{b+\delta\omega}{a}b_i)(1-\sum_{i=1}^{n}\frac{\beta i x_i^*(t)\frac{ip(i)}{\langle k\rangle}}{b+\frac{b+\delta\omega}{a}b_i})$$
$$> \prod_{i=1}^{n}(b+\frac{b+\delta\omega}{a}b_i)(1-\sum_{i=1}^{n}\frac{\beta i\lambda\frac{ip(i)}{\langle k\rangle}}{a(b+\frac{b+\delta\omega}{a}b_i)})$$
$$= 0.$$

Then we have root in the interval $[-b-\Psi_1(\rho),0]$. Hence we get n negative roots in $[-b-\Psi_n(\rho),0]$. Then the endemic point E_1 is locally asymptotically stable ([6], [7]).

4 Global Stability

In this section, the global stability of E_0 and E_1 are studied by using Lyapunov function.

4.1 Global stability of free disease point E_0

Theorem 4.1.1 If $R_0<1$, then the disease-free equilibrium E_0 is globally asymptotically stable.

Proof. Firstly, we recall first equation of system (2):

$$\frac{dx_k}{dt} = \lambda-\beta k x_k\psi(t)-\delta\omega y_k-(\delta+\alpha+\mu)x_k. \quad (7)$$

$$\frac{d^q x_k}{dt} < \lambda-(\delta+\alpha+\mu)x_k. \quad (8)$$

By using Laplace transform:

$$x_k(t) < E_q(-(\delta+\alpha+\mu)t^q)(x_k(0)-\frac{\lambda}{(\delta+\alpha+\mu)})+\frac{\lambda}{(\delta+\alpha+\mu)}, \quad (9)$$

$x_k(t) < \frac{\lambda}{(\delta+\alpha+\mu)}$ if $x_k(0)\leq\frac{\lambda}{(\delta+\alpha+\mu)}$, but if $x_k(0)>\frac{\lambda}{(\delta+\alpha+\mu)}$ and $\lim_{t\to\infty^+}E_q(-(\delta+\alpha+\mu)t^q)=0$, that mean $x_k(t)$ tends to $\frac{\lambda}{(\delta+\alpha+\mu)}$ with time tends to infinity.

For system (2), consider the following Lyapunov function:

$$L_0(t) = \sum_{k=1}^{n}d_k(y_k(t)), \quad (10)$$

where $d_k = \frac{kp(k)}{\langle k \rangle}$, the fractional time derivation of L_0 along the solution of system (2) is calculated, we get

$$D^q L_0 = \sum_k \frac{kp(k)}{\langle k \rangle}(\beta k x_k \psi(t) - (\gamma+\eta) y_k)$$

$$D^q L_0 = \psi(t)(\gamma+\eta)\sum_k (\frac{k^2 p(k)\beta x_k(t)}{\langle k \rangle (\gamma+\eta)} - 1)$$

$$D^q L_0 < \psi(t)(\gamma+\eta)\sum_k (\frac{k^2 p(k)\beta\lambda}{\langle k \rangle (\gamma+\eta)(\delta+\mu+\alpha)} - 1),$$

where $x_k(t) < \frac{\lambda}{\delta+\mu+\alpha}$,

$$D^q L_0 < \psi(t)(\gamma+\eta)(R_0 - 1), \tag{11}$$

then

$$D^q L_0 < 0 \text{ if } R_0 < 1.$$

Hence $L_0(t) > 0, L_0(t) = 0$ at E_0, if $R_0 < 1$ then $D^q L_0 < 0$. By Lemma 2.2 in [17] and Theorem 4.1.1, E_0 is globally asymptotically stable when $R_0 < 1$, which implies the disease will be die regardless the initial infected individuals.

4.2 Global stability of endemic point E_1

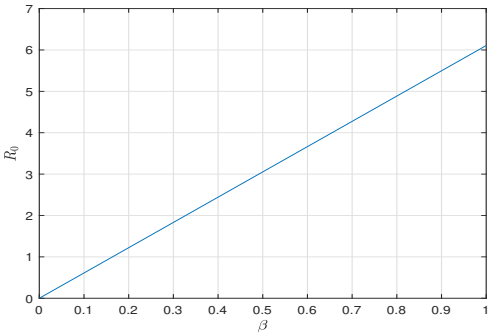
Theorem 4.2.1 If $R_0 > 1$, let (ζ, M) be weighted digraph is strongly connected, then E_1 is globally asymptotically stable in $\Omega^* = \Omega / \{E_0\}$.

Proof. Define Lyapunov function $L_1(t)$ as follows:

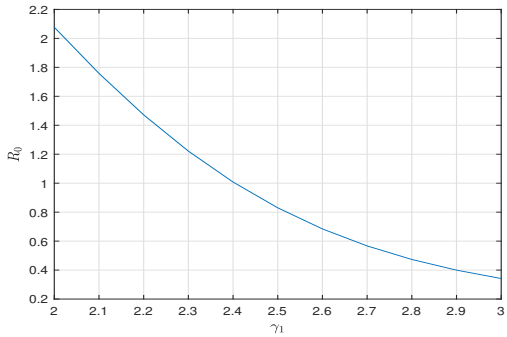
$$L_1(t) = \sum_{k=1}^{n} q c_k (y_k - y_k^* - y_k^* \ln(\frac{y_k}{y_k^*})) \tag{12}$$
$$+ \frac{1}{2}\frac{\beta k c_k \langle k \rangle^{-1} y_i^*}{y_k^*} \sum_i i p(i)(x_k - x_k^* + y_k - y_k^*)^2,$$

where $g(x) = 1 - x + \ln(x) < 0$, $\lambda = \beta k x_k^* \psi^*(t) + (\delta+\mu+\alpha)x_k^* + \delta\omega y_k^*$ and $(\gamma+\eta) = \frac{\beta k x_k^* \psi^*(t)}{y_k^*}$, $q = \gamma+\eta+\delta+\mu+\alpha+\delta\omega$.

Calculate the fractional time derivate of L_1 along the solution of system (2). By using the Lemma 2.3 [17] we



(a)



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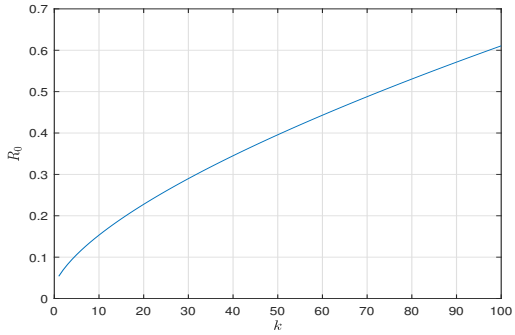

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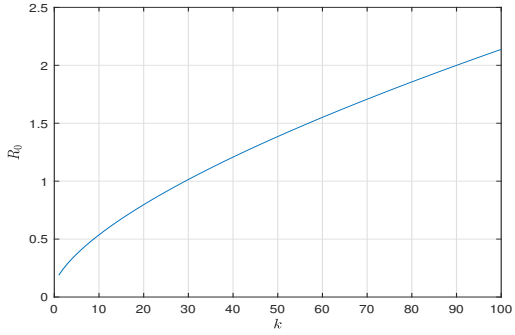
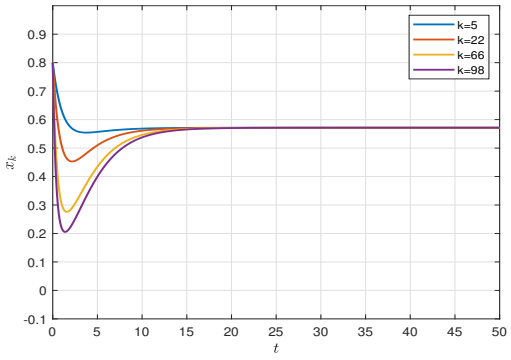
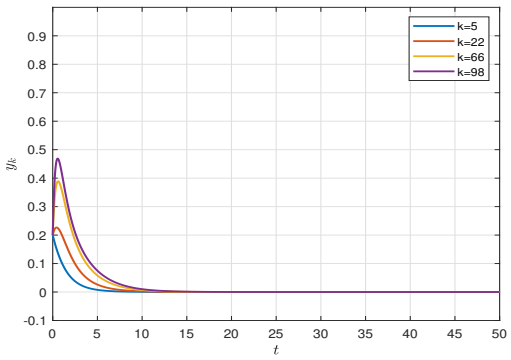


(d)

Fig. 1: The relation between R_0, β (a), γ_1 (b) and k (the number of nodes) (c), (d).

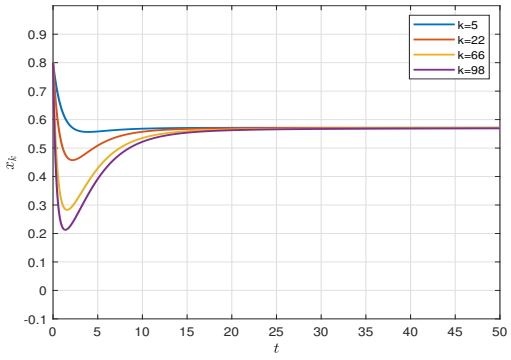


(a)



(b)

Fig. 2: $q = 1,\ R_0 = 0.6107 < 1.$



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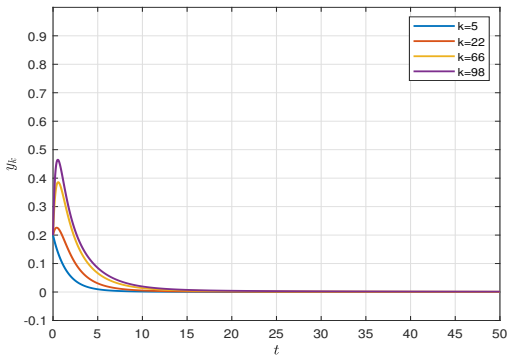


(b)

Fig. 3: $q = 0.98,\ R_0 = 0.6107 < 1.$

get

$$
\begin{aligned}
D^q L_1(t) \leq & \sum_k c_k((\gamma+\eta+\delta+\mu+\alpha+\delta\omega) \\
& \cdot (1-\frac{y_k^*}{y_k})(\beta k x_k \psi(t) - \beta k x_k^* \psi^*(t)\frac{y_k}{y_k^*}) \\
& + \langle k\rangle^{-1}\frac{y_i^*}{y_k^*}\sum_i ip(i)(x_k - x_k^* + y_k - y_k^*) \\
& \cdot (-(\delta+\mu+\alpha)(x_k - x_k^*) - (\gamma+\eta+\delta\omega)(y_k - y_k^*))) \\
D^q L_1(t) < & \sum_k c_k \beta k \langle k\rangle^{-1}\sum_i ip(i)(\gamma+\eta+\delta+\mu+\alpha+\delta\omega) \\
& \cdot ((y_k - y_k^*)(\frac{x_k y_i}{y_k} - \frac{x_k^* y_i^*}{y_k^*}) - \frac{y_i^*}{y_k^*}(y_k - y_k^*)(x_k - x_k^*)) \\
D^q L_1(t) < & \sum_{i=1}^n\sum_{k=1}^n \beta k \langle k\rangle^{-1} c_k(m_{ik} - n_{ik})(\frac{y_i}{y_i^*} - \frac{y_k}{y_k^*}),
\end{aligned}
$$

where $m_{ik} = x_k ip(i) y_i^*, n_{ik} = x_k ip(i) y_i$.

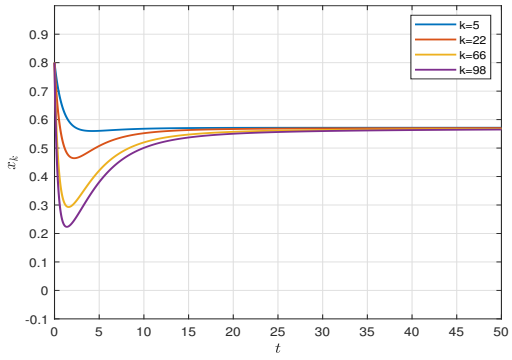
Let (ζ, M) be weighted digraph with matrix M . If (ξ, M) is strongly connected, then matrix M is irreducible [19] and choose c_k as cofactor of k^{th}main diagonal of Laplacian matrix of M. From the tree cycle identity (Theorem 2.3, [19]), we obtain the following identity:

$$
\sum_{i=1}^n\sum_{k=1}^n \beta k \langle k\rangle^{-1} c_k(m_{ik} - n_{ik})(\frac{y_i}{y_i^*} - \frac{y_k}{y_k^*}) = 0. \qquad (13)
$$

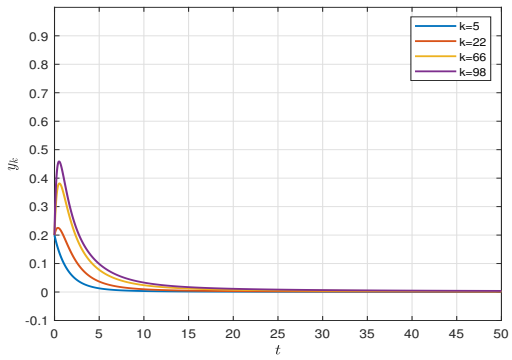
Hence $L_1(t) = 0$, if $(x_k, y_k) = (x_k^*, y_k^*)$ and $D^q L_1(t) < 0$. Furthermore, the largest invariant set the singleton $\{E_1\}$ in $\Omega^* = \Omega/\{E_0\}$. By Lemma 2.2 in [17] and Theorem 4.2.1, E_1 is globally asymptotically stable, which implies that the disease still remaining in endemic level and never die out. This result leads biological scientist to find methods to, reduce the basic reproduction number R_0 to be less than one.

5 Numerical simulations

In this section, we solve system (1) by using Adams-type predictor-corrector method ([12], [13]) to show main results in previous sections on BA scale-free network with $p(k) = mk^{-\gamma_1}$, $2 < \gamma_1 < 3$ is variable of power law distribution. We have $n = 100$, m is such that $\sum_k p(k) = 1$

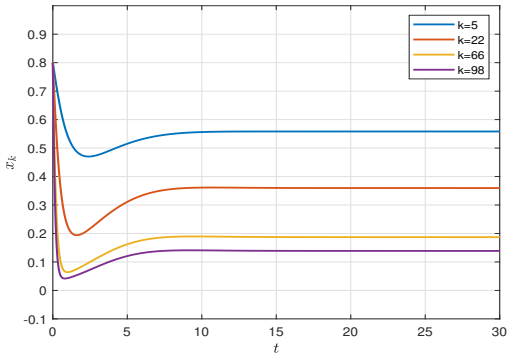


(a)

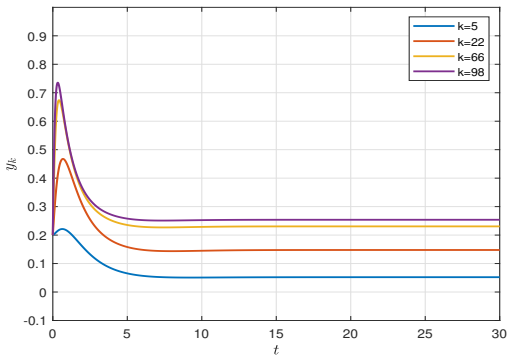


(b)

Fig. 4: $q = 0.95,\ R_0 = 0.6107 < 1.$



(a)



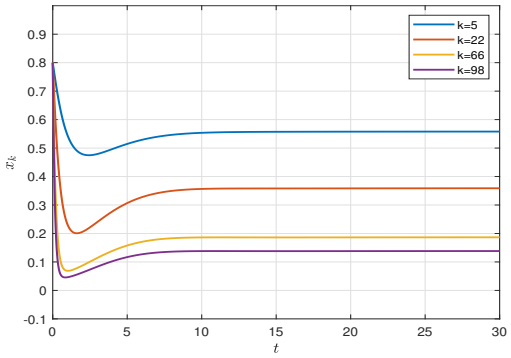
(b)

Fig. 5: $q = 1,\ R_0 = 2.1375 > 1.$

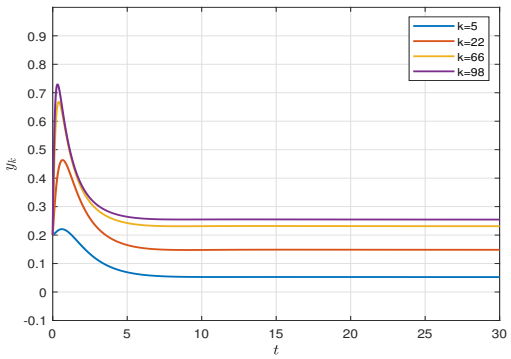
and $\gamma_1 = 2.3$. We use the initial values of system (2) as: $x_k(0) = 0.8, y_k(0) = 0.2, z_k(0) = 0$.

In Figure (1-a), $\mu = 0.3, \delta = 0.1, \alpha = 0.3, \eta = 0.561, \gamma = 0.5$ and $\beta \in [0,1]$. In Figure (1-b) $\mu = 0.3, \delta = 0.1, \beta = 0.1, \alpha = 0.3, \eta = 0.561, \gamma = 0.5$ and $\gamma_1 \in]2,3[$. In Figure (1-c) $\mu = 0.3, \delta = 0.1, \beta = 0.1, \alpha = 0.3, \eta = 0.561, \gamma = 0.5, 1 < k < 100$. In Figure (1-d) $\mu = 0.3, \delta = 0.1, \beta = 0.3, \alpha = 0.2, \eta = 0.561, \gamma = 0.5, 1 < k < 100$. In Figures (2-4) $\mu = 0.3, \delta = 0.1, \beta = 0.1, \alpha = 0.3, \eta = 0.561, \gamma = 0.5$ and hence $R_0 = 0.6107$. In Figures (5-7) $\mu = 0.3, \delta = 0.1, \beta = 0.3, \alpha = 0.2, \eta = 0.561, \gamma = 0.5$ and hence $R_0 = 2.1375$.

Figure 1(a) shows the relation between infected individuals and infection rate β with positive relation, figure 1(b) shows the important rule of power γ_1 in power-law distribution we can easily see that inverse relation between them that mean higher values of γ_1 mean the connection between nodes very weak and reproductive ratio has lower values and vice versa. Figure 1(c) shows that the importance of the number of nodes if the number of nodes is high, the reproductive ratio grows up still $R_0 < 1$ but we have several values from 0 to nearly 1 depending on the number of nodes. If the number of nodes is high that means the chance for connecting is high and spreading infection become faster. Figure 1(d) with $R_0 > 1$, we can see the effect of the number of nodes that reproductive ratio reaches maximum for $k = 100$ that mean the spreading infection becomes deadly with higher values of k. Figure 3, where $R_0 < 1$ shows that the number of infected individuals at a peak in a lower degree of nodes is smaller than at a higher degree.meanly higher values of degree reflect the connection is very strong and the peak of infected individuals is higher than lower degrees which mean weak connection. Figure 3 show the effect of fractional order q that has lower peak and the number of infected need a longer time to go to the stable region (where $y_k(t) = 0$) than integer-order. This feature helps biological scientists to study infection and comparing our data to more clinical data. By calculating coefficient of variability in fractional and integer order, fractional-order has a lower coefficient of the variability than integer-order that means fractional-order has better fit data than integer-order. Figure 5 show the global stability of the system (1) and go the positive level stationary because of $R_0 > 1$. Figure 5 shows the fractional-order has lower peak than integer-order and

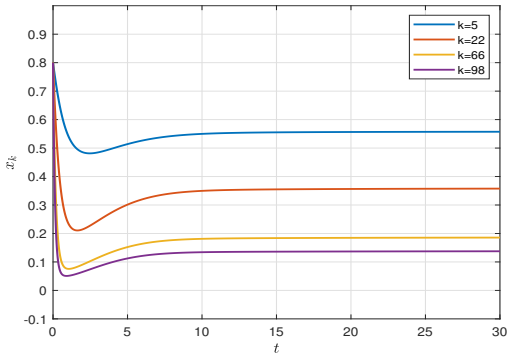


(a)

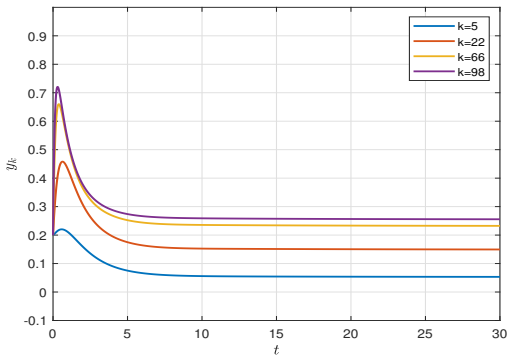


(b)

Fig. 6: $q = 0.98,\ R_0 = 2.1375 > 1.$



(a)



(b)

Fig. 7: $q = 0.95,\ R_0 = 2.1375 > 1.$

needs more time to go to the stable region that means the number of infected is wider than integer-order. Finally, because of non locally of fractional order we can obtain more appropriate fractional-order q by comparing with integer order. The obtained results can be used in different applications see i.e. [20]-[30].

6 Conclusion

In this paper, we have examined the epidemic dynamics of the SIRS model on complex heterogeneous networks in fractional order. We have proved that the degree distribution of nodes plays an important rule not only in the existence of reproductive ratio R_0 but determine the value of it as in figs. 1b and c. By the degree distribution, we can control the spreading of disease. In figs. 3 and 5, we can see that the lower values of k, we have a lower peak of infected individuals that means the connection between individuals is the main factor in the spreading of diseases. In $R_0 < 1$, we show that free disease equilibrium point E_0 is locally and globally asymptotically stable but in $R_0 > 1$, we have proved that the existence of an epidemic equilibrium point E_1 which locally and globally asymptotically stable. the effect of fractional order q appears in numerical results especially in figs. 3 and 5. We can see that the solution with fractional order q has a lower and wider peak than integer order which permits getting more accurate fitting data.in fig. 3, we can see that the solution of fractional order takes more time than integer-order to go the stable region which helps us to more studying the behavior of disease before tends to zero. Finally, the fractional-order model has a big advantage is non local order. Mainly, we can choose a more appropriate fractional-order that suitable of clinical data as in [20].

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Competing interests

The authors declare that they have no competing interests.

Availability of data and materials

Not applicable.

References

[1] S. N. Dorogovtsev, A. V. Goltsev, and J. F. F. Mendes, Reviews of Modern Physics, **80**, 1275–1335 (2008).
[2] M. E. J. Newman, SIAM Review, **45**, 167-256 (2003).
[3] De-gang Xu, Xi-yang Xu, Chun-hua Yang, and Wei-hua Gui, Mathematical Problems in Engineering (2015), https://doi.org/10.1155/2015/365049.
[4] H. A. A. El-Saka, A. A. M. Arafa and M. I. Gouda, Advances in Difference Equations, **144** (2019), https://doi.org/10.1186/s13662-019-2079-3.
[5] Yao Hua, Lequan Minab and Yang Kuang, Applicable Analysis: An International Journal, **94,** 2308-2330 (2014).
[6] E. Ahmed, A. M. A. El-Sayed, H. A. A. El-Saka, Physics Letters A, **358,** 1-4 (2006).
[7] E. Ahmed, A. M. A. El-Sayed, H. A. A. El-Saka, J. Math. Anal. Appl., **325**, 542-553, 2007.
[8] L. Hong, Z. Long, H. Cheng, Y. Jiang, and T. Zhidong, Nonlinear Analysis Modelling and Control, **22**, 303-316 (2017).
[9] L. Hong, M. Ahmadjan, Z. Long, and T. Zhidong, Advances in Difference Equations, **325** (2018), https://doi.org/10.1186/s13662-018-1776-7.
[10] H. A. A. El-Saka and E. Ahmed, bioRxiv preprint first posted online Feb. 18 (2016), http://dx.doi.org/10.1101/039917.
[11] I. Podlubny, Fractional differential equations, Academic Press, 1999.
[12] Kai Diethelm, The analysis of fractional differential equations: an application-oriented exposition using differential operators of caputo type (Lecture Notes in Mathematics), Springer-Verlag Berlin Heidelberg 2010.
[13] H. A. El-Saka and A. El-Sayed, Fractional order equations and dynamical systems, Lambrt Academic Publishing, Germany, ISBN 978-3-659-40197-8, 2013.
[14] D. Matignon, Computational Eng. in Sys. Appl., **2** (1996).
[15] J. Huo, H. Zhao, and L. Zhu, Nonlinear Analysis: Real World Applications, **26**, 289-305 (2015).
[16] C. Vargas-De-Lénn, Commun. Nonlinear Sci. Numer. Simul., **24**, 75-85 (2015).
[17] J. Huo, H. Zhao, Physica A , **448**, 41-56 (2016).
[18] H. A. A. El-Saka, Seyeon Lee and Bongsoo Jang, Nonlinear Dynamics, **96**, 407-416 (2019).
[19] M.Y. Li, Z. Shuai, J. Differential Equations, **248**, 1-20 (2010).
[20] H. A. A. El-Saka, I. Obaya and H.N. Agiza, Advances in Difference Equations, **5** (2021), https://doi.org/10.1186/s13662-020-03182-y.
[21] A. A. M. Teamah, W. A. Afifi, Javid Gani Dar, Abd Al-Aziz Hosni El-Bagoury and Sndus Naji Al-Aziz, J. Stat. Appl. Prob., **9**, 473-481 (2020).
[22] Abdullah Ali H. Ahmadini, Nitesh K. Adichwal, Mutum Zico Meetei, Yashpal Singh Raghav, Mohammed Ali H. Ahmadini, Ahmed Msmali, Neha Seth, Knowledge, J. Stat. Appl. Prob., **10**, 487-497 (2021).
[23] Hamid El Maroufy, Adil Lahrouz and PGL Leach, Appl. Math. Inf. Sci., **5**, 220-238 (2011).
[24] H. A. A. El-Saka, Math. sci. Lett., **2**, 195-200 (2013).
[25] Ahmed M. Yousef, Saad Z. Rida, Yassein Gh. Gouda and Asmaa S. Zaki, Prog. Frac. Diff. Appl., **5**, 297-306 (2019).
[26] Walaa M. Abd-Elhafiez and Hanan H. Amin, Inf. Sci. Lett., **10**, 141- 152 (2021).
[27] P. Veeresha, Wei Gao, D. G. Prakasha, N. S. Malagi, E. Ilhan and Haci Mehmet Baskonus, Inf. Sci. Lett., **10**, 205-212 (2021).
[28] Wael Mustafa, Shrink, Inf. Sci. Lett., **10**, 255-261 (2021).
[29] Osama Yaseen M. Al-Rawi, Wisam Subhi Al-Dayyeni and Ibrahim Reda, Inf. Sci. Lett., **10**, 427-433 (2021).
[30] Ismail Atia, Mohamed L Salem, Aya Elkholy, Wael Elmashad and Gomaa A. M. Ali, Inf. Sci. Lett., **10**, 561-570 (2021).