Information Sciences Letters

Volume 11
Issue 1 *Jan. 2022*

Article 20

2022

Asymptomatic and Pre-Symptoms Transmission of COVID-19 in Heterogeneous Epidemic Network

H. A. A. El-Saka
Mathematics Department, Faculty of Science, Damietta University, New Damietta 34517, Egypt, mmlashin@pnu.edu.sa

Ayman Al-Dmour
Department of Computer Science, Applied Science University, Bahrain, mmlashin@pnu.edu.sa

Ibrahim Obaya
Basic Science Department, Nile Higher Institute for Engineering & Technology, Mansoura, Egypt\\
Department of Mathematics, Faculty of Science, Mansoura University, P.O. Box 64, Mansoura 35516, Egypt, mmlashin@pnu.edu.sa

Information Sciences Letters
An International Journal

http://dx.doi.org/10.18576/isl/110116

Asymptomatic and Pre-Symptoms Transmission of COVID-19 in Heterogeneous Epidemic Network

H. A. A. El-Saka[1,*], *Ayman Al-Dmour*[2] *and Ibrahim Obaya*[3,4]

[1]Mathematics Department, Faculty of Science, Damietta University, New Damietta 34517, Egypt
[2]Department of Computer Science, Applied Science University, Bahrain
[3]Basic Science Department, Nile Higher Institute for Engineering & Technology, Mansoura, Egypt
[4]Department of Mathematics, Faculty of Science, Mansoura University, P.O. Box 64, Mansoura 35516, Egypt

Received: 19 Jun. 2021, Revised: 2 Aug. 2021; Accepted: 29 Sep. 2021
Published online: 1 Jan. 2022.

Abstract: The diversity of the spread pattern of the Corona virus is one of the most important reasons for the seriousness of the virus. Therefore, in this paper, we present a fractional mathematical SEIAS model that studies many ways of spreading (asymptomatic and pre-symptoms transmission) with the hypothesis of the spread of the virus in a heterogeneous network of individuals. The system consists of nonlinear equations which formed in fractional order. And it turns out that the system has two equilibrium positions (free and endemic positions). We also calculated the disease prevalence threshold ($\mathcal{R}_0$) within the network. The condition for the existence of the epidemiological situation has been determined. The stability of the free equilibrium position has been studied. The numerical part has been added to explain the proved theorems of the system in addition to clarifying the role of the heterogeneous network on the value of the virus spread threshold within the network.
Keywords : Complex Networks, Novel Coronavirus (COVID-19), Asymptomatic and Pre-Symptoms Transmission, Basic Reproductive Number and Fractional Calculus.

1 Introduction

After the announcement in January 2020 of the beginning of the spread of coronavirus (COVID-19) in the Chinese city of Wuhan, which quickly spread in China and all countries of the world until it became a global epidemic threatening the lives of many [21-22]. Since then, many government agencies and international organizations concerned with the spread of epidemics have sought to study the outbreak of the COVID-19. From the medical side, we find the continuous search for a vaccine against COVID-19, as virologists seek to know the nature of the virus, which helps to know the pattern of its spread and also how to confront it [18-20]. And we cannot exclude the role of mathematical modeling as a tool and a means in order to predict the pattern of the spread of virus and determine the basic reproductive number of the spread of virus. Mathematical models differ from one another due to the assumptions used in creating the mathematical model. In this regard, we present a mathematical model simulating The current situation based on the attained and announced characteristics of COVID-19.

After the novel corona virus was considered a global epidemic that invaded most of the countries of the world, we find that the nature of the spread of the virus is different from one region to another, which appears in the numbers of infected people. And when focusing on the method of spreading, we find that it is recognized that the transmission of infection occurs from a person with COVID-19 to an uninfected person. What is meant by a person infected with COVID-19 here is a person who shows and develops the common symptoms that characterize the infection of COVID-19 [8-11]. The infection is transmitted by many methods, such as close contact with an infected person, through the droplets that comes out of the infected person when coughing, sneezing or speaking and by touching the surfaces on which the injured person's droplets has fallen [1-7].

On the other hand, the transmission of infection is not

* Corresponding author E-mail: mmlashin@pnu.edu.sa

limited to infected persons who show complete symptoms of the disease. It is possible to transmit infection in the period before the symptoms (incubation period), which is the period from the beginning of exposure to the virus until the onset of symptoms. The transmission of infections from people in the incubation period appeared in a number of reports and medical studies. And those studies were based on the fact that some tests that were done for some people to detect the virus show positive results, and that is 1-3 days before the symptoms appear [6-16].

And not only that, some laboratory cases have been confirmed as having COVID-19 but without any symptoms (Asymptomatic) [12-17]. Such cases can help to transmit infection between individuals, while it is difficult to determine its source. And such cases are not counted among the infected numbers in addition to that they do not enter between the cases of health isolation. Such phenomena have an effective role on the spread of infection, as we need to take more precautionary and preventive measures not similar to the one taken if the infection is caused by a person with confirmed symptoms. Also, the transmission of the disease through an asymptomatic person or pre-symptoms one can lead to a continuous spread of the infection that prevents it from being controlled.

During the formation of mathematical models, society is divided into several groups, according to the nature of the infection caused by the virus. According to the data issued by the Korea Centers for Disease Control and prevention, positive cases of COVID-19 were discovered again after a complete recovery. On April 9, 2020, this number was 74 cases before it doubled within one week and reached 163 positive cases, which caused a state of panic and intense fear of the possibility of reinfection again. Accordingly, the SEIAS type was chosen to be appropriate to the prevalence of the virus with the addition of a new class that expresses the asymptomatic individuals.

The heterogeneous nature of daily interactions between individuals requires us to use heterogeneous network models.

because of their preference and accuracy in expressing the status of an infected community [31]. Where the infected community is represented by an epidemiological network in which the individuals are the network nodes and the connections (links) between those nodes represent the communication between the members of the community. In addition, we will use a fractional order for the system that used to describe infection transmission instead of using the integer order [23-30]. Where fractional orders are distinguished when used in differential equations with several features, among them they have the effect of nonlocality, as well as the memory in the definition of fractional order. We will use the definition of Caputo which is defined as [23].

Table 1: The definitions of the parameters.

Parameter	Definition
A	Birth rate.
β_1	The infectious rate from infected individual.
β_2	The infectious rate from asymptomatic individual.
β_3	The infectious rate from pre-symptoms individual.
γ_1	Recovery rate of infected individual and be susceptible again.
γ_2	Recovery rate of asymptomatic individual and be susceptible again.
B	Natural death rate.
d	Death rate due to disease.
σ	Rate of becoming infectious after latency.
μ	Rate of becoming asymptomatically infected.

$$ {}_a^C D_t^\alpha f(t) = \frac{1}{\Gamma(1-\alpha)} \int_a^t (t-s)^{-\alpha} f'(s) ds, $$

where $0 < \alpha \leq 1$.

2 Dynamical Models

In this modeling study, the population is divided into four compartments susceptible, exposed, infected and asymptomatic. The susceptible individuals can be exposed due to interacting with infected, asymptomatic and pre-symptoms one. The exposed person becomes asymptomatic at rate μ and infected at rate $(1-\mu)$ after the incubation period σ. The infected and the asymptomatic persons become susceptible again after the infectious period at rates γ_1 and γ_2 respectively. The population size is variant due to births and the deaths.

The dynamical model is defined as follow:

$$ {}_0^C D_t^\alpha S_k(t) = A - \frac{k\beta_1 S_k(t)\Theta_1(\mathrm{t})}{N_k(t)} - \frac{k\beta_2 S_k(t)\Theta_2(\mathrm{t})}{N_k(t)} $$

$$ - \frac{k\beta_3 S_k(t)\Theta_3(\mathrm{t})}{N_k(t)} + \gamma_1 I_k(t) + \gamma_2 A_k(t) $$

$$ - BS_k(t), \qquad (1) $$

$$ {}_0^C D_t^\alpha E_k(t) = \frac{k\beta_1 S_k(t)\Theta_1(\mathrm{t})}{N_k(t)} + \frac{k\beta_2 S_k(t)\Theta_2(\mathrm{t})}{N_k(t)} $$

$$ -BE_k(t), \qquad (2) $$

$$ {}_0^C D_t^\alpha I_k(t) = (1-\mu)\sigma E_k(t) - \gamma_1 I_k(t) - dI_k(t) $$

$$ -BI_k(t), \qquad (3) $$

$$
\begin{aligned}
{}_0^C D_t^{\alpha} A_k(t) &= \mu\sigma E_k(t) - \gamma_2 A_k(t) \\
&\quad - B A_k(t), \qquad (4)
\end{aligned}
$$

where, $S_k(t), E_k(t), I_k(t)$ and $A_k(t)$ are the number of susceptible, exposed, infected and asymptomatic persons at time t with degree k $(1 \le k \le n)$, n is the maximum degree of a node. $\Theta_1(t), \Theta_2(t)$ and $\Theta_3(t)$ are the probability to be linked with an infected, asymptomatic and exposed node respectively, which are defined as

$$
\Theta_1(t) = \frac{\sum_k kP(k)I_k(t)}{\langle k \rangle},
$$

$$
\Theta_2(t) = \frac{\sum_k kP(k)A_k(t)}{\langle k \rangle},
$$

$$
\Theta_3(t) = \frac{\sum_k kP(k)\,E_k(t)}{\langle k \rangle},
$$

where $\langle k \rangle = \sum_k kP(k)$, $P(k)$ is the degree distribution of the population and $N_k(t)$ is the total population of degree k.

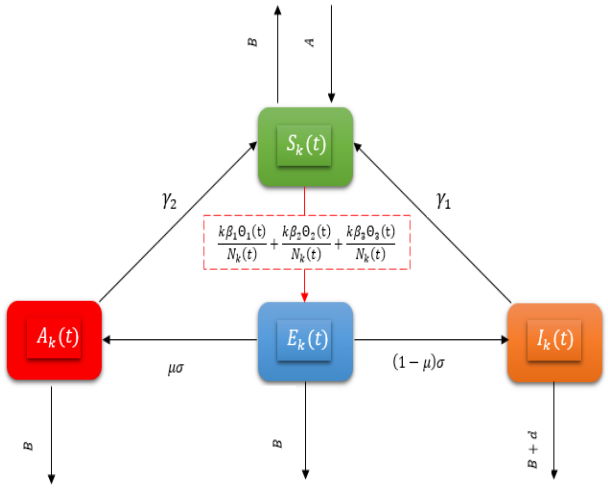

Fig. 1: The dynamical behavior of system (1)-(4).

2.1 Existence of Steady States

The next theorem explains the existence of the equilibrium points.

Theorem 1. Define

$$
\mathcal{R}_0 = \frac{\langle k^2 \rangle}{\langle k \rangle} \frac{[\beta_1(1-\mu)\sigma(\gamma_2+B) + \beta_2\mu\sigma(\gamma_1+d+B) + \beta_3(\gamma_1+d+B)(\gamma_2+B)]}{(\sigma+B)(\gamma_1+d+B)(\gamma_2+B)}. \qquad (5)
$$

If $\mathcal{R}_0 < 1$ then the system (1)-(4), has a unique free disease equilibrium point $P_0 = \left\{\frac{A}{B}, 0,0,0\right\}_{1\le k\le n}$ and if $\mathcal{R}_0 > 1$, the free disease equilibrium point P_0 is existing in addition to a unique endemic point $P_* = \{S_k^*, E_k^*, I_k^*, A_k^*\}_{1\le k\le n}$

where,

$$
S_k^* = \frac{(\sigma+B)(\gamma_1+d+B)I_k^*(A-dI_k^*)}{k\phi B(1-\mu)\sigma},
$$

$$
E_k^* = \frac{(\gamma_1+d+B)I_k^*}{(1-\mu)\sigma},
$$

$$
A_k^* = \frac{\mu(\gamma_1+d+B)I_k^*}{(\gamma_2+B)(1-\mu)},
$$

$$
\begin{aligned}
I_k^* = \frac{1}{2d(\gamma_2+B)(\gamma_1+d+B)(\sigma+B)} \big[& k\phi\big[B\gamma_1(B+\mu\sigma \\
& +\gamma_2) \\
& +(B+d)\big(B(B+d) \\
& +\gamma_2(B+(1-\mu)\sigma)\big)\big] \\
& +A(\gamma_2+B)(\gamma_1+d+B)(\sigma+B) \\
& -\sqrt{D}\big].
\end{aligned}
$$

Proof. After some algebraic calculations [33-37], we find the disease-free steady state is $P_0 = \left\{\frac{A}{B}, 0,0,0\right\}_{1\le k\le n}$.

For the existence of the endemic steady state, we have

$$
S_k = \frac{(\sigma+B)(\gamma_1+d+B)I_k(A-dI_k)}{k\phi B(1-\mu)\sigma},
$$

$$
E_k = \frac{(\gamma_1+d+B)I_k}{(1-\mu)\sigma},
$$

$$
A_k = \frac{\mu(\gamma_1+d+B)I_k}{(\gamma_2+B)(1-\mu)},
$$

$$
h(I_k) = r_2 I_k^2 - r_1 I_k + r_0 = 0, \qquad (6)
$$

where,

$$
r_2 = d(\gamma_2+B)(\gamma_1+d+B)(\sigma+B) > 0,
$$

$$
\begin{aligned}
r_1 = k\phi\big[& B\gamma_1(B+\mu\sigma+\gamma_2) + (B \\
& +d)\big(B(B+d) + \gamma_2(B+(1-\mu)\sigma)\big)\big] \\
& +A(\gamma_2+B)(\gamma_1+d+B)(\sigma+B) > 0,
\end{aligned}
$$

$$
r_0 = A(1-\mu)\sigma k\phi(\gamma_2+B) > 0,
$$

$$
\phi(t) = \beta_1\Theta_1(t) + \beta_2\Theta_2(t) + \beta_3\Theta_3(t). \qquad (7)
$$

Thus, the existence of the endemic equilibrium depending on solving equation (5) with respect to I_k where the solution $I_k^* \in \left[0, \frac{A}{B}\right]$. By solving the second-degree polynomial, we get two solutions as follow

$$I_k^1 = \frac{1}{2d(\gamma_2+B)(\gamma_1+d+B)(\sigma+B)}\big[k\phi\big[B\gamma_1(B+\mu\sigma+\gamma_2) + (B+d)\big(B(B+d)+\gamma_2(B+(1-\mu)\sigma)\big)\big] + A(\gamma_2+B)(\gamma_1+d+B)(\sigma+B)+\sqrt{D}\big],$$

$$I_k^2 = \frac{1}{2d(\gamma_2+B)(\gamma_1+d+B)(\sigma+B)}\big[k\phi\big[B\gamma_1(B+\mu\sigma+\gamma_2) + (B+d)\big(B(B+d)+\gamma_2(B+(1-\mu)\sigma)\big)\big] + A(\gamma_2+B)(\gamma_1+d+B)(\sigma+B)-\sqrt{D}\big],$$

where, $D = r_1^2 - 4r_2r_0$.

We have $I_k^1 * I_k^2 = \frac{r_0}{r_2} > 0$ and $I_k^1 > 0$. So, we get $0 < I_k^2 < I_k^1$.

Also, $h\left(\frac{A}{B}\right) < 0$. Therefore, $0 < I_k^2 < \frac{A}{B}$.

It leads to I_k^2 is the unique solution of $h(I_k)$ in $\left[0, \frac{A}{B}\right]$.

Then, the endemic equilibrium point is

$$S_k^* = \frac{(\sigma+B)(\gamma_1+d+B)I_k^*(A-dI_k^*)}{k\phi B(1-\mu)\sigma},$$

$$E_k^* = \frac{(\gamma_1+d+B)I_k^*}{(1-\mu)\sigma},$$

$$A_k^* = \frac{\mu(\gamma_1+d+B)I_k^*}{(\gamma_2+B)(1-\mu)},$$

$$I_k^* = \frac{1}{2d(\gamma_2+B)(\gamma_1+d+B)(\sigma+B)}\big[k\phi\big[B\gamma_1(B+\mu\sigma+\gamma_2)+(B+d)\big(B(B+d)+\gamma_2(B+(1-\mu)\sigma)\big)\big] + A(\gamma_2+B)(\gamma_1+d+B)(\sigma+B)-\sqrt{D}\big].$$

Substituting with I_k^*, A_k^* and E_k^* into the definition of $\phi(t)$ we get the following self-consistency equation:

$$\phi = \beta_1\frac{\sum_k kP(k)I_k^*}{\langle k\rangle} + \beta_2\frac{\sum_k kP(k)A_k^*}{\langle k\rangle} + \beta_3\frac{\sum_k kP(k)E_k^*}{\langle k\rangle},$$

$\phi = g(\phi)$, where,

$$g(\phi) = \frac{1}{\langle k\rangle}\sum_k kP(k)I_k^*\left(\beta_1 + \beta_2\frac{\mu(\gamma_1+d+B)}{(\gamma_2+B)(1-\mu)} + \beta_3\frac{(\gamma_1+d+B)}{(1-\mu)\sigma}\right) \quad (8)$$

and,

$$\phi(0) = 0, \qquad 0 < \phi(t) < \beta_1+\beta_2+\beta_3.$$

We have $g(0) = 0$, $\lim_{\phi\to\infty} g(\phi) > 0, \frac{dg}{d\phi} > 0$ and $\frac{d^2g}{d\phi^2} < 0$ then, a non-trivial solution exists if and only if $\left.\frac{dg}{d\phi}\right|_{\phi=0} > 1$.

Therefore,

$$\frac{\langle k^2\rangle}{\langle k\rangle}\frac{[\beta_1(1-\mu)\sigma(\gamma_2+B)+\beta_2\mu\sigma(\gamma_1+d+B)+\beta_3(\gamma_1+d+B)(\gamma_2+B)]}{(\sigma+B)(\gamma_1+d+B)(\gamma_2+B)} > 1.$$

We define

$$\mathcal{R}_0 = \frac{\langle k^2\rangle}{\langle k\rangle}\frac{[\beta_1(1-\mu)\sigma(\gamma_2+B)+\beta_2\mu\sigma(\gamma_1+d+B)+\beta_3(\gamma_1+d+B)(\gamma_2+B)]}{(\sigma+B)(\gamma_1+d+B)(\gamma_2+B)},$$

As the basic reproductive number, where

$$\langle k^2\rangle = \sum_k k^2P(k).$$

2.2 The Threshold Value with Next Generation Method and Local Stability

By using the next generation method [32], we present the following theorem.

Theorem 2. The disease-free equilibrium point P_0 of model (1)-(4) is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof. We take only the three equations of exposed, infected and asymptomatic persons $E_k(t), I_k(t)$ and $A_k(t)$ as follow:

$$\begin{aligned}
{}_0^C D_t^\alpha E_k(t) &= \frac{kS_k(t)\phi(t)}{N_k(t)} - \sigma E_k(t) - BE_k(t),\\
{}_0^C D_t^\alpha I_k(t) &= (1-\mu)\sigma E_k(t) - \gamma_1 I_k(t) - dI_k(t) - BI_k(t),\\
{}_0^C D_t^\alpha A_k(t) &= \mu\sigma E_k(t) - \gamma_2 A_k(t) - BA_k(t).
\end{aligned}$$

The matrix

$$F = \begin{pmatrix} \mathcal{F}_{11} & \mathcal{F}_{12} & \mathcal{F}_{13}\\ \mathcal{F}_{21} & \mathcal{F}_{22} & \mathcal{F}_{23}\\ \mathcal{F}_{31} & \mathcal{F}_{32} & \mathcal{F}_{33}\end{pmatrix}_{3n\times 3n},$$

referred to the new infected cases entering $E_k(t), I_k(t)$ and $A_k(t)$ compartments and evaluated at the disease-free equilibrium point P_0. We have

$$\mathcal{F}_{11} = \frac{\beta_3}{\langle k\rangle}\begin{pmatrix} P(1) & 2P(2) & \dots & nP(n)\\ 2P(1) & 2^2P(2) & \dots & 2nP(n)\\ \vdots & \vdots & \ddots & \vdots\\ nP(1) & 2nP(2) & \dots & n^2P(n)\end{pmatrix}_{n\times n},$$

$$\mathcal{F}_{12} = \frac{\beta_1}{\langle k\rangle}\begin{pmatrix} P(1) & 2P(2) & \dots & nP(n)\\ 2P(1) & 2^2P(2) & \dots & 2nP(n)\\ \vdots & \vdots & \ddots & \vdots\\ nP(1) & 2nP(2) & \dots & n^2P(n)\end{pmatrix}_{n\times n},$$

$$\mathcal{F}_{13} = \frac{\beta_2}{\langle k\rangle}\begin{pmatrix} P(1) & 2P(2) & \dots & nP(n)\\ 2P(1) & 2^2P(2) & \dots & 2nP(n)\\ \vdots & \vdots & \ddots & \vdots\\ nP(1) & 2nP(2) & \dots & n^2P(n)\end{pmatrix}_{n\times n},$$

$$\mathcal{F}_{21} = \mathcal{F}_{22} = \mathcal{F}_{23} = \mathcal{F}_{31} = \mathcal{F}_{32} = \mathcal{F}_{33} = \begin{pmatrix} 0 & 0 & \dots & 0\\ 0 & 0 & \dots & 0\\ \vdots & \vdots & \ddots & \vdots\\ 0 & 0 & \dots & 0\end{pmatrix}_{n\times n}.$$

And the matrix

$$V = \begin{pmatrix} \mathcal{V}_{11} & \mathcal{V}_{12} & \mathcal{V}_{13}\\ \mathcal{V}_{21} & \mathcal{V}_{22} & \mathcal{V}_{23}\\ \mathcal{V}_{31} & \mathcal{V}_{32} & \mathcal{V}_{33}\end{pmatrix}_{3n\times 3n},$$

referred to the transmission between and out of exposed, infected and asymptomatic compartments and evaluated at the disease-free equilibrium point P_0. We get

$$\mathcal{V}_{11} = (\sigma + B)\begin{pmatrix} 1 & 0 & \dots & 0 \\ 0 & 1 & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & 1 \end{pmatrix}_{n\times n},$$

$$\mathcal{V}_{21} = (-(1-\mu)\sigma)\begin{pmatrix} 1 & 0 & \dots & 0 \\ 0 & 1 & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & 1 \end{pmatrix}_{n\times n},$$

$$\mathcal{V}_{22} = (\gamma_1 + d + B)\begin{pmatrix} 1 & 0 & \dots & 0 \\ 0 & 1 & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & 1 \end{pmatrix}_{n\times n},$$

$$\mathcal{V}_{31} = (-\mu\sigma)\begin{pmatrix} 1 & 0 & \dots & 0 \\ 0 & 1 & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & 1 \end{pmatrix}_{n\times n},$$

$$\mathcal{V}_{33} = (\gamma_2 + B)\begin{pmatrix} 1 & 0 & \dots & 0 \\ 0 & 1 & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & 1 \end{pmatrix}_{n\times n},$$

$$\mathcal{V}_{12} = \mathcal{V}_{13} = \mathcal{V}_{23} = \mathcal{V}_{32} = \begin{pmatrix} 0 & 0 & \dots & 0 \\ 0 & 0 & \dots & 0 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \dots & 0 \end{pmatrix}_{n\times n}.$$

Then, the matrix FV^{-1} has the characteristic equation

$$x^{2n}\left(\frac{\langle k^2\rangle}{\langle k\rangle}\frac{[\beta_1(1-\mu)\sigma(\gamma_2+B)+\beta_2\mu\sigma(\gamma_1+d+B)+\beta_3(\gamma_1+d+B)(\gamma_2+B)]}{(\sigma+B)(\gamma_1+d+B)(\gamma_2+B)} - x\right)^n = 0.$$

It has $2n$ eigenvalues equal to zero and n eigenvalues equal to

$$\frac{\langle k^2\rangle}{\langle k\rangle}\frac{[\beta_1(1-\mu)\sigma(\gamma_2+B)+\beta_2\mu\sigma(\gamma_1+d+B)+\beta_3(\gamma_1+d+B)(\gamma_2+B)]}{(\sigma+B)(\gamma_1+d+B)(\gamma_2+B)},$$

which represents the basic reproductive number $\mathcal{R}_0$.

3 Numerical Simulations

Obviously, from equation (5) the value of $\mathcal{R}_0$ depends on an important parameter $\frac{\langle k^2\rangle}{\langle k\rangle}$ which represent the heterogeneity of the network. Figures 2 explain the change in $\mathcal{R}_0$ value with respect the value of maximum degree k.

In table (2) we have the parameters values for three countries (USA, South Korea and Italy). We used the Adams-type Predictor-Corrector method [27-29] for solving the system (1)-(4) and showing the prediction curve of infected individuals.

Considering our network is a scale free network with $p(k) = \rho k^{-\nu}$, where ρ is a constant satisfies $\sum_k p(k) = 1$ and $2 < \nu < 3$ is the exponent of the power law distribution. Choosing $\nu = 2.3$ and $n = 100$ [37, 38].

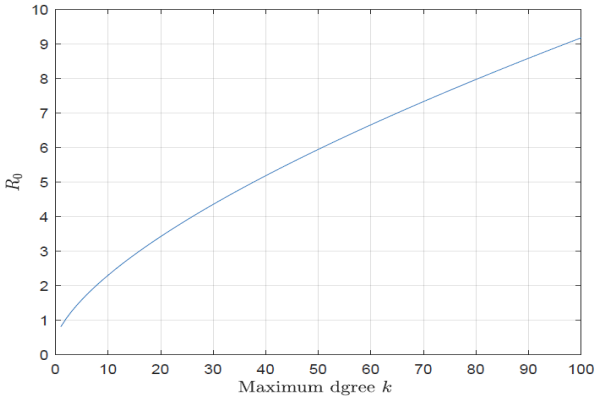

Fig. 2(a): The change in $\mathcal{R}_0$ value with respect the value of maximum degree k (South Korea).

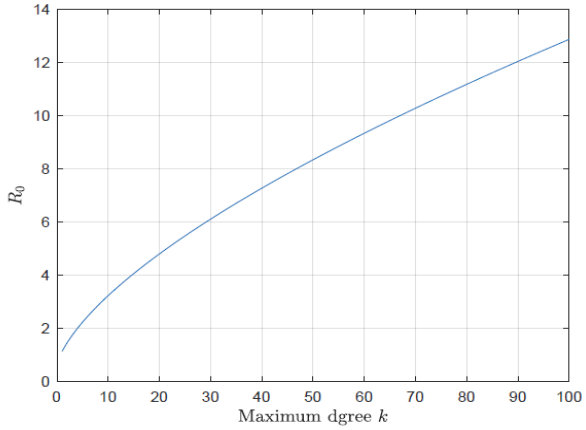

Fig. 2(b): The change in $\mathcal{R}_0$ value with respect the value of maximum degree k (Italy).

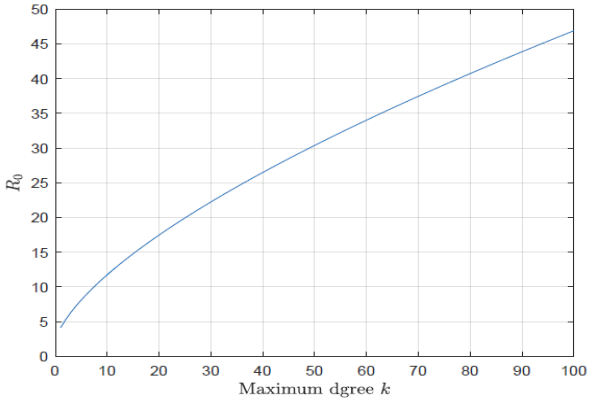

Fig. 2(c): The change in $\mathcal{R}_0$ value with respect the value of maximum degree k (USA).

Table (2-a): Parameters values.

Parameter	South Korea	Italy
A	980	1244
β_1	0.59	0.59
β_2	0.1	0.3
β_3	0.1	0.1
γ_1	0.4349	0.4349
γ_2	0.4349	0.4349
B	0.005573	0.0107
d	0.0000048	0.00046
σ	0.2657	0.2657
μ	0.8	0.8
N_0	51640000	60480000
S_0	51639556	60479556
E_0	84	9
I_0	6	1
A_0	22	2

Table (2-b): Parameters values.

Parameter	USA	NYC
A	3481608	300
β_1	0.3813	0.1813
β_2	0.7065	0.2065
β_3	0.7065	0.1065
γ_1	0.4349	0.0149
γ_2	0.4349	0.049
B	6.7063×10^-8	0.000037596
d	0.00019	0.0000011975
σ	0.2657	0.2657
μ	0.8	0.1
N_0	328.2 million	8202000
S_0	328199856	8200000
E_0	45	500
I_0	3	1000
A_0	12	500

Figure 3 for South Korea, figure 4 for Italy and figure 5 for USA for different α and k.

Also we used the transmission rates β_1, β_2 and β_3 as a function of time

$$\beta_1(t) = \frac{\beta_1 - \beta_{01}}{1 + ct} + \beta_{01}\,,$$
$$\beta_2(t) = \frac{\beta_2 - \beta_{02}}{1 + ct} + \beta_{02}, \qquad (9)$$
$$\beta_3(t) = \frac{\beta_3 - \beta_{03}}{1 + ct} + \beta_{03},$$

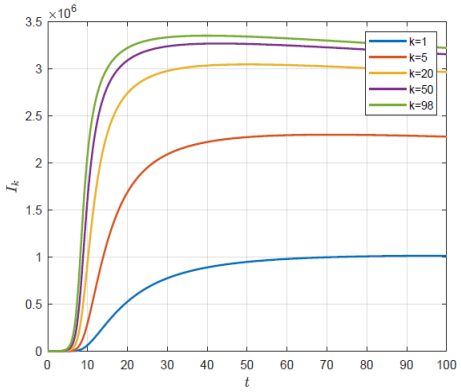

Fig. 3(a): $\alpha = 0.7$.

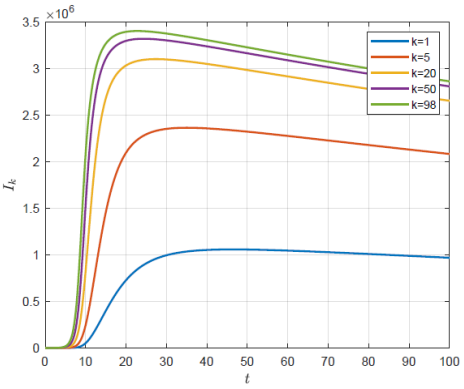

Fig. 3(b): $\alpha = 0.85$.

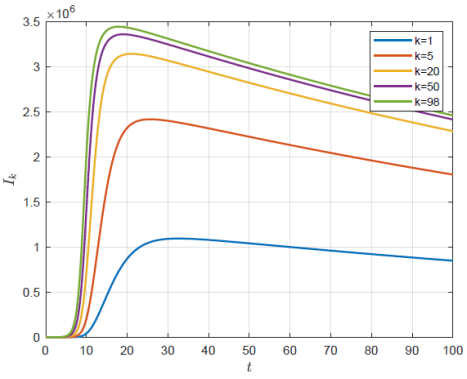

Fig. 3(c): $\alpha = 0.95$.

Fig. 3: $\mathcal{R}_0 = 9.1724$ **(South Korea).**

$0 \leq c \leq 1$, is a parameter that reflect the extent of adherence to the preventive instructions, β_{01}, β_{02} and β_{03} are the least value of transmission rates of infection due to infected, asymptomatic and pre-symptoms individual respectively.

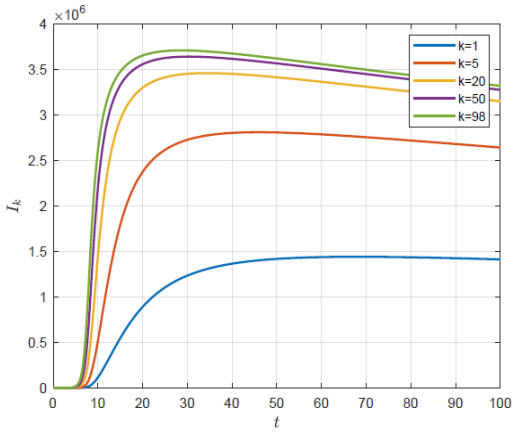

Fig. 4(a): $\alpha = 0.7$.

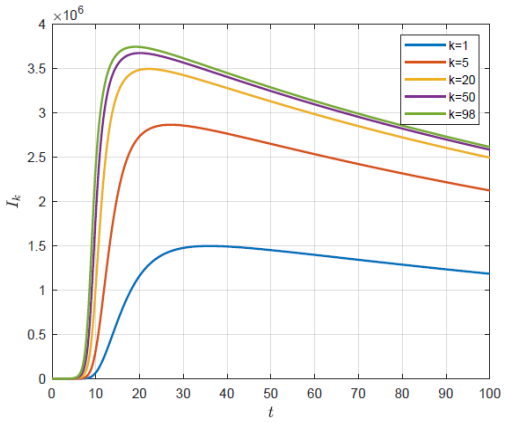

Fig. 4(b): $\alpha = 0.85$.

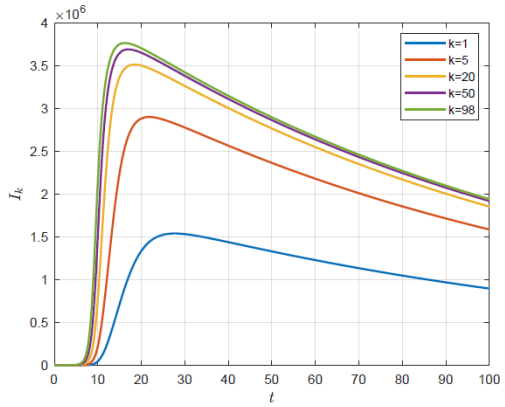

Fig. 4(c): $\alpha = 0.95$.

Fig. 4: $\mathcal{R}_0 = 12.8568$ (Italy).

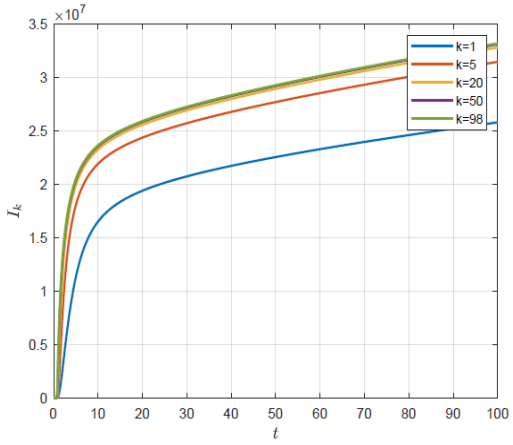

Fig. 5(a): $\alpha = 0.7$.

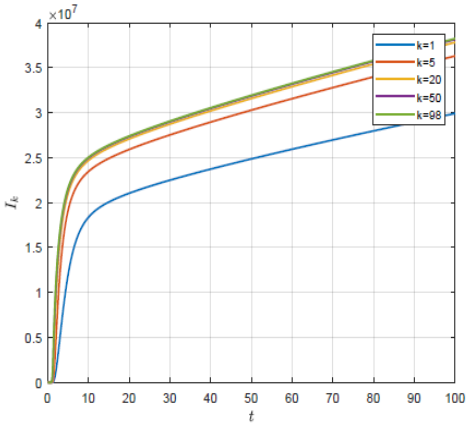

Fig. 5(b): $\alpha = 0.85$.

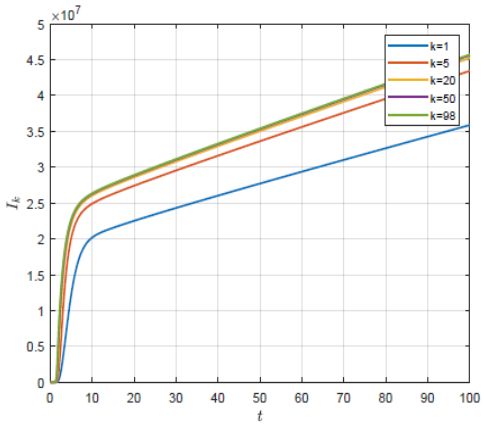

Fig. 5(c): $\alpha = 0.95$.

Fig. 5: $\mathcal{R}_0 = 46.8750$ (USA).

We assumed $\beta_{01} = 0.03, \beta_{02} = 0.009$ and $\beta_{03} = 0.009$.

In this case the value of $\mathcal{R}_0$ becomes a function in time and taking the form

$$\mathcal{R}_0(t) = \frac{\langle k^2\rangle}{\langle k\rangle}\frac{[\beta_1(t)(1-\mu)\sigma(\gamma_2+B)+\beta_2(t)\mu\sigma(\gamma_1+d+B)+\beta_3(t)(\gamma_1+d+B)(\gamma_2+B)]}{(\sigma+B)(\gamma_1+d+B)(\gamma_2+B)}.$$

The effect of parameter c appears in figures 6-11. A high value of c makes the peak of the infected individuals lower.

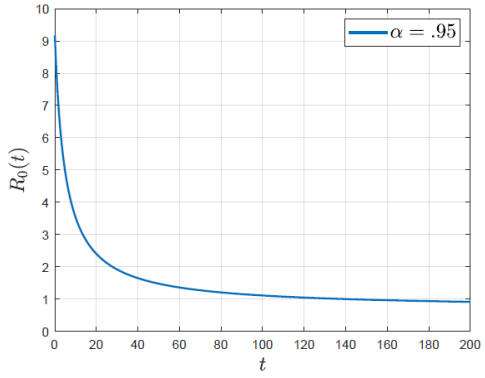

Fig. 6(a): $c = 0.2$ (South Korea).

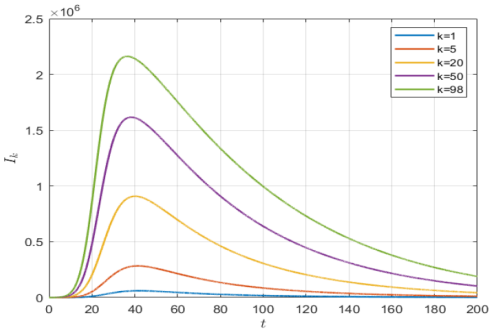

Fig. 6(b): $c = 0.2$ (South Korea).

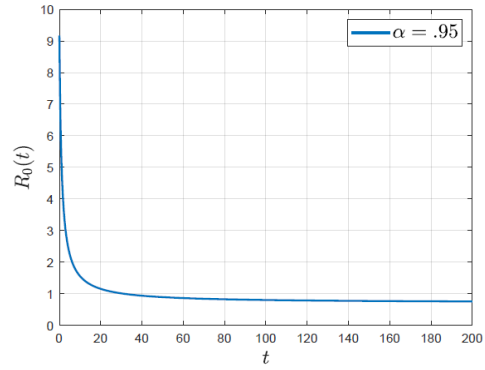

Fig. 7(a): $c = 0.9$ (South Korea).

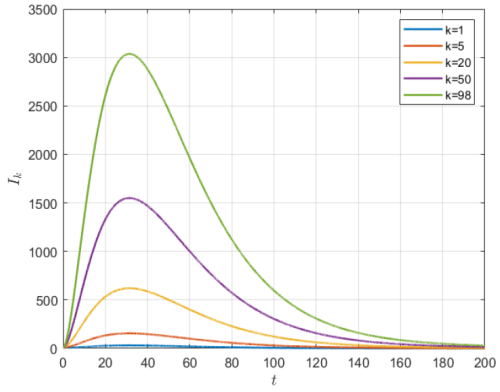

Fig. 7(b): $c = 0.9$ (South Korea).

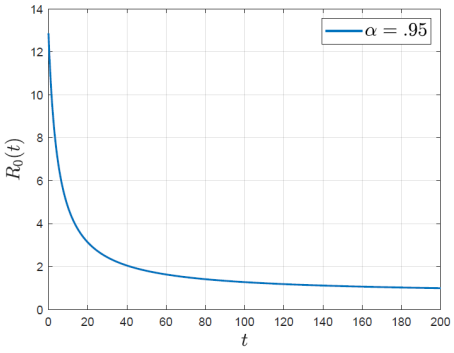

Fig. 8(a): $c = 0.2$ (Italy).

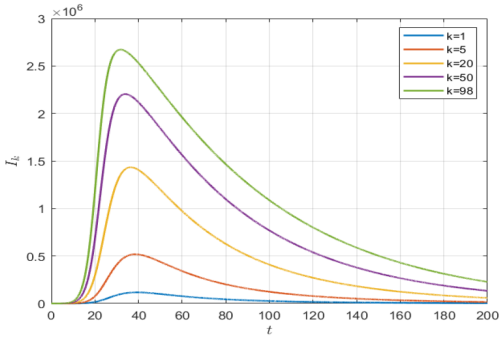

Fig. 8(b): $c = 0.2$ (Italy).

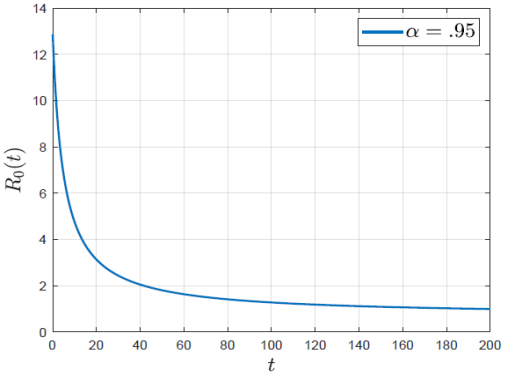

Fig. 9(a): $c = 0.9$ (Italy).

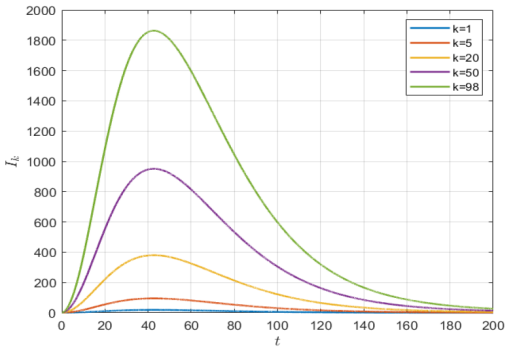

Fig. 9(b): $c = 0.9$ (Italy).

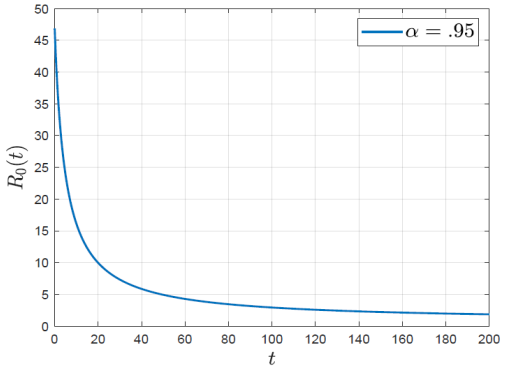

Fig. 10(a): $c = 0.2$ (USA).

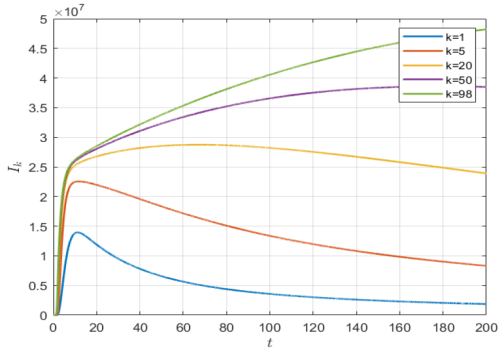

Fig. 10(b): $c = 0.2$ (USA).

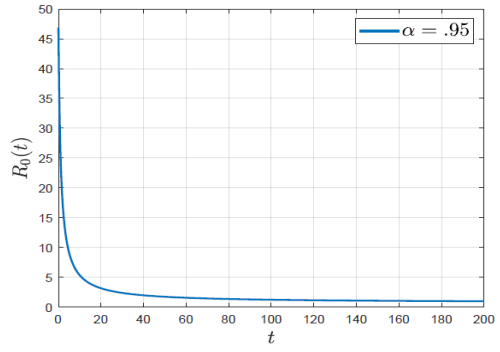

Fig. 11(a): $c = 0.9$ (USA).

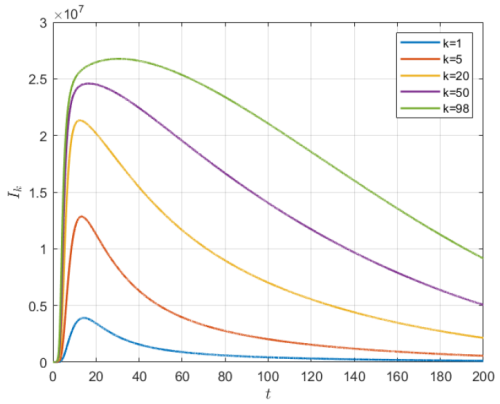

Fig. 11(b): $c = 0.9$ (USA).

In figures (12), we plot the actual data collected from the beginning of the outbreak in New York city with light blue curve. At plotting the prediction curves for different k, we choose β_1, β_2 and β_3 as a function of time that defined in Eqs. (9). We take $c = 0.92$, $\beta_{01} = 0.001, \beta_{02} = 0.001$, $\beta_{03} = 0.001$ and the parameters value defined in table (2) for different values α. We observed that, the actual data curve agreed with the prediction curve (yellow curve) when $k = 20$ and $\alpha = 0.75$ but after $t = 50$ see figure (12-a). In figure (12-b), with $\alpha = 0.85$ the prediction curve (red curve) at $k = 5$ matches more with the actual data. Changing the value of $\alpha = 0.9$, the actual curve does not match with any curve see figure (12-c). For another value for $\alpha = 0.98$, the actual data agree with the prediction curve (blue curve) at $k = 1$ see figure (12-d). From this comparison with the actual data, we can summarize that the fractional order α and the degree k of the node in the network give us more possible cases that could be agree with the real data.

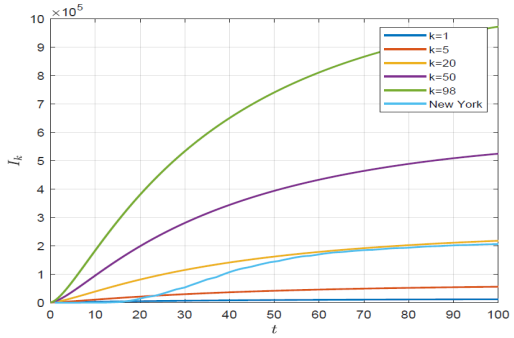

Fig. 12(a): $\alpha = 0.75$.

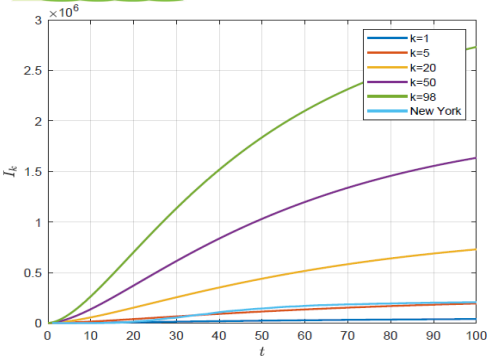

Fig. 12(b): $\alpha = 0.85$.

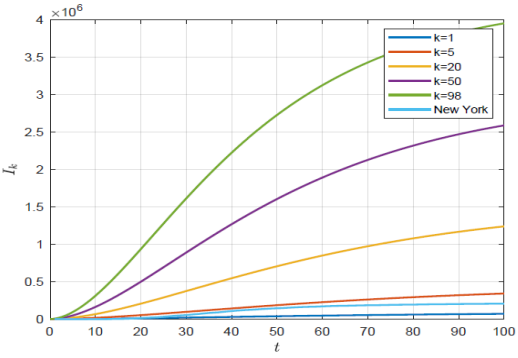

Fig. 12(c): $\alpha = 0.9$.

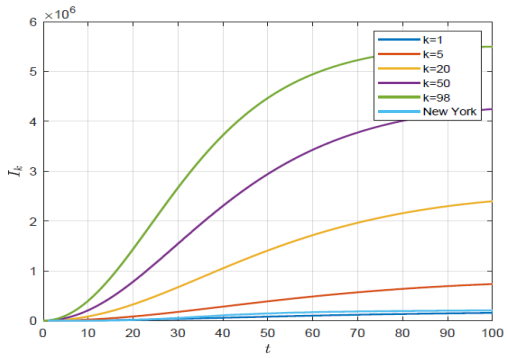

Fig. 12(d): $\alpha = 0.98$.

Fig. 12: $c = 0.92$ (New York).

4 Conclusions

In this paper, a fractional SEIAS model is presented in heterogeneous network. The nodes of the network are chosen to be varying. The transmission due to asymptomatic and pre-symptoms persons plays an important role in the outbreak of COVID-19 so, we take into account its effect. We proved the existence of the disease-free equilibrium point and the endemic point. Also, we calculated the threshold value of system (1)-(4) in addition we studied the local stability of the disease-free equilibrium point. In the numerical section, the role of the heterogeneity and the degree of the node in the network is illustrated. We observed that when the maximum degree k of a node is increasing the value of $\mathcal{R}_0$ increases see Fig. 2. On another hand, we supposed the transmission rates β_1, β_2 and β_3 as a function of time, it leads to the value of $\mathcal{R}_0$ becomes a function of t. This means that the $\mathcal{R}_0$ value may be greater or less than unity during the change of time see figures 6-11. It also depends directly on the definition of transmission functions and the parameter c. See Figs. 6-11 for Korea, Italy and the United States of America. When studying the impact of adherence to social distancing instructions, we found that the higher rate of commitment, the fewer number of infected individuals i.e., when parameter c takes a high value, it makes the peak of infectious lower.

Acknowledgements
We thank Prof. H. N. Agiza (Mathematics Department, Faculty of Science, Mansoura University, Mansoura, Egypt) for his support and comments.

Competing interests
The author declares that she has no competing interests.

Funding
Not applicable.

References

[1] Liu, J., Liao, X., Qian, S., Yuan, J., Wang, F., Liu, Y., Wang, Z., Wang, F.S., Liu, L. and Zhang, Z.. (2020). Community transmission of severe acute respiratory syndrome coronavirus 2, Shenzhen, China, 2020. Emerging infectious diseases., **26(6)**, 1320(2020).

[2] Chan, J.F.W., Yuan, S., Kok, K.H., To, K.K.W., Chu, H., Yang, J., Xing, F., Liu, J., Yip, C.C.Y., Poon, R.W.S. and Tsoi, H.W., (2020). A familial cluster of pneumonia associated with the 2019 novel coronavirus indicating person-to-person transmission: a study of a family cluster. The lancet, **395(10223)**, 514-523(2020).

[3] Li, Q., Guan, X., Wu, P., Wang, X., Zhou, L., Tong, Y., Ren, R., Leung, K.S., Lau, E.H., Wong, J.Y. and Xing, X., (2020). Early transmission dynamics in Wuhan, China, of novel coronavirus–infected pneumonia. New England journal of medicine.

[4] Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y., Zhang, L., Fan, G., Xu, J., Gu, X. and Cheng, Z., (2020). Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. The lancet., **395(10223)**, 497-506(2020).

[5] Burke, R.M., Midgley, C.M., Dratch, A., Fenstersheib, M., Haupt, T., Holshue, M., Ghinai, I., Jarashow, M.C., Lo, J., McPherson, T.D. and Rudman, S., (2020). Active monitoring of persons exposed to patients with confirmed COVID-19—United States, January–February 2020. Morbidity and Mortality Weekly Report., **69(9)**, 245(2020).

[6] World Health Organization. Report of the WHO-China Joint Mission on Coronavirus Disease 2019 (COVID-19) 16-24 February 2020 [Internet]. Geneva: World Health Organization; 2020 Available from: https://www.who.int/docs/default- source/coronaviruse/who-

china-joint-mission-on-covid-19-final-report.pdf

[7] Ong, S. W. X., Tan, Y. K., Chia, P. Y., Lee, T. H., Ng, O. T., Wong, M. S. Y., & Marimuthu, K. (2020). Air, surface environmental, and personal protective equipment contamination by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) from a symptomatic patient. Jama., **323(16)**, 1610-1612(2020).

[8] Wang, W., Xu, Y., Gao, R., Lu, R., Han, K., Wu, G., & Tan, W. (2020). Detection of SARS-CoV-2 in different types of clinical specimens. Jama., **323(18)**, 1843-1844(2020).

[9] Lauer, S.A., Grantz, K.H., Bi, Q., Jones, F.K., Zheng, Q., Meredith, H.R., Azman, A.S., Reich, N.G. and Lessler, J., (2020). The incubation period of coronavirus disease 2019 (COVID-19) from publicly reported confirmed cases: estimation and application. Annals of internal medicine., **172(9)**, 577-582(2020).

[10] Liu, Y., Yan, L.M., Wan, L., Xiang, T.X., Le, A., Liu, J.M., Peiris, M., Poon, L.L. and Zhang, W., (2020). Viral dynamics in mild and severe cases of COVID-19. The Lancet infectious diseases., **20(6)**, 656-657(2020).

[11] Wölfel, R., Corman, V.M., Guggemos, W., Seilmaier, M., Zange, S., Müller, M.A., Niemeyer, D., Jones, T.C., Vollmar, P., Rothe, C. and Hoelscher, M., (2020). Virological assessment of hospitalized patients with COVID-2019. Nature., **581(7809)**, 465-469(2020).

[12] Yu, P., Zhu, J., Zhang, Z., & Han, Y. (2020). A familial cluster of infection associated with the 2019 novel coronavirus indicating possible person-to-person transmission during the incubation period. The Journal of infectious diseases., **221(11)**, 1757-1761(2020).

[13] Huang, R., Xia, J., Chen, Y., Shan, C., & Wu, C. (2020). A family cluster of SARS-CoV-2 infection involving 11 patients in Nanjing, China. The Lancet Infectious Diseases., **20(5)**, 534-535(2020).

[14] Pan, X., Chen, D., Xia, Y., Wu, X., Li, T., Ou, X., Zhou, L. and Liu, J., (2020). Asymptomatic cases in a family cluster with SARS-CoV-2 infection. The Lancet Infectious Diseases., **20(4)**, 410-411(2020).

[15] Ling, A., & Leo, Y. (2020). Potential presymptomatic transmission of SARS-CoV-2, Zhejiang province, China, 2020. Emerging Infectious Diseases., **26**, 1052-1054(2020).

[16] Wei, W. E., Li, Z., Chiew, C. J., Yong, S. E., Toh, M. P., & Lee, V. J. (2020). Presymptomatic transmission of SARS-CoV-2—Singapore, January 23–march 16, 2020. Morbidity and Mortality Weekly Report., **69(14)**, 411(2020).

[17] Kimball, A., Hatfield, K. M., Arons, M., James, A., Taylor, J., Spicer, K., ... & Zane, S. (2020). Asymptomatic and presymptomatic SARS-CoV-2 infections in residents of a long-term care skilled nursing facility—King County, Washington, March 2020. Morbidity and Mortality Weekly Report., **69(13)**, 377(2020).

[18] Wu, J. T., Leung, K., & Leung, G. M. (2020). Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Wuhan, China: a modelling study. The Lancet., **395(10225)**, 689-697(2020).

[19] Huang, C., Wang, Y., Li, X., Ren, L., Zhao, J., Hu, Y., Zhang, L., Fan, G., Xu, J., Gu, X. and Cheng, Z., (2020). Clinical features of patients infected with 2019 novel coronavirus in Wuhan, China. The lancet., **395(10223)**, 497-506(2020).

[20] Zhou, F., Yu, T., Du, R., Fan, G., Liu, Y., Liu, Z., Xiang, J., Wang, Y., Song, B., Gu, X. and Guan, L., (2020). Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: a descriptive study. The lancet., **395(10223)**, 507-513(2020).

[21] https://www.who.int/westernpacific/emergencies/covid-19

[22]https://www.cdc.gov/media/dpk/diseases-and conditions/coronavirus/coronavirus-2020.html

[23] Podlubny, I. (1998). Fractional differential equations: an introduction to fractional derivatives, fractional differential equations, to methods of their solution and some of their applications. Elsevier.

[24] Matignon, D. (1996, July). Stability results for fractional differential equations with applications to control processing. In Computational engineering in systems applications., **2(1)**, 963-968).

[25] Ahmed, E., El-Sayed, A. M. A., & El-Saka, H. A. (2006). On some Routh–Hurwitz conditions for fractional order differential equations and their applications in Lorenz, Rössler, Chua and Chen systems. Physics Letters A, 358(1), 1-4.

[26] El-Sayed, A. M. A., El-Mesiry, A. E. M., & El-Saka, H. A. A. (2007). On the fractional-order logistic equation. Applied Mathematics Letters., **20(7)**, 817-823(2007).

[27] Ahmed, E., El-Sayed, A. M. A., & El-Saka, H. A. (2007). Equilibrium points, stability and numerical solutions of fractional-order predator–prey and rabies models. Journal of Mathematical Analysis and Applications., **325(1)**, 542-553(2007).

[28] El-Saka H. A. A., & Ahmed, E. S. (2013). Fractional Order Equations and Dynamical Systems. Lap Lambert Academic Publ.

[29] Diethelm, K. (2010). The analysis of fractional differential equations: An application-oriented exposition using differential operators of Caputo type. Springer Science & Business Media.

[30] Ahmed, E. M., & El-Saka, H. A. (2017). On a fractional order study of Middle East Respiratory Syndrome Corona virus (MERS-Co V). J. Fract. Calc. Appl., **8(1)**, 118-126(2017).

[31] Newman, M. E., Barabási, A. L. E., & Watts, D. J. (2006). The structure and dynamics of networks. Princeton university press.

[32] Diekmann, O., Heesterbeek, J. A. P., & Roberts, M. G. (2010). The construction of next-generation matrices for compartmental epidemic models. Journal of the Royal Society Interface., **7(47)**, 873-885(2010).

[33] Huo, J., & Zhao, H. (2016). Dynamical analysis of a fractional SIR model with birth and death on heterogeneous complex networks. Physica A: Statistical Mechanics and its

Applications., **448**, 41-56(2016).

[34] Latha, V. P., Rihan, F. A., Rakkiyappan, R., & Velmurugan, G. (2018). A fractional-order model for Ebola virus infection with delayed immune response on heterogeneous complex networks. Journal of Computational and Applied Mathematics., **339**, 134-146(2020).

[35] El-Saka, H. A. A., Arafa, A. A. M., & Gouda, M. I. (2019). Dynamical analysis of a fractional SIRS model on homogenous networks. Advances in Difference Equations., **2019(1)**, 1-15(2019).

[36] Jin, Z., Zhang, J., Song, L. P., Sun, G. Q., Kan, J., & Zhu, H. (2011). Modelling and analysis of influenza A (H1N1) on networks. BMC public health., **11(1)**, 1-9(2011).

[37] El-Saka, H. A. A., Obaya, I., & Agiza, H. N. (2021). A fractional complex network model for novel corona virus in China. Advances in Difference Equations., **(1)**, 1-19(2021).

[38] El-Saka, H. A. A., Arafa, A. A. M., Alshalabi, Riyad & Gouda, M. I.(2022). Dynamical Analysis of a Fractional SIRS Model on Complex Heterogeneous Networks, Information Sciences Letters., **11(1)**, 1-8(2022).