Since January 2020 Elsevier has created a COVID-19 resource centre with free information in English and Mandarin on the novel coronavirus COVID-19. The COVID-19 resource centre is hosted on Elsevier Connect, the company's public news and information website.

Elsevier hereby grants permission to make all its COVID-19-related research that is available on the COVID-19 resource centre - including this research content - immediately available in PubMed Central and other publicly funded repositories, such as the WHO COVID database with rights for unrestricted research re-use and analyses in any form or by any means with acknowledgement of the original source. These permissions are granted for free by Elsevier for as long as the COVID-19 resource centre remains active.
COVID-19 epidemic under the K-quarantine model: Network approach

K. Choia, Hoyun Choa, B. Kahngb,c,∗

a CCSS, CTP and Department of Physics and Astronomy, Seoul National University, Seoul 08826, Korea
b Center for Theoretical Physics, Seoul National University, Seoul 08826, Korea
c CCSS and KI for Grid Modernization, Korea Institute of Energy Technology, Naju, Jeonnam 58217, Korea

ABSTRACT

The COVID-19 pandemic is still ongoing worldwide, and the damage it has caused is unprecedented. For prevention, South Korea has adopted a local quarantine strategy rather than a global lockdown. This approach not only minimizes economic damage but also efficiently prevents the spread of the disease. In this work, the spread of COVID-19 under local quarantine measures is modeled using the Susceptible-Exposed-Infected-Recovered model on complex networks. In this network approach, the links connected to infected and so isolated people are disconnected and then reinstated when they are released. These link dynamics leads to time-dependent reproduction number. Numerical simulations are performed on networks with reaction rates estimated from empirical data. The temporal pattern of the accumulated number of confirmed cases is then reproduced. The results show that a large number of asymptomatic infected patients are detected as they are quarantined together with infected patients. Additionally, possible consequences of the breakdowns of local quarantine measures and social distancing are considered.

© 2022 Elsevier Ltd. All rights reserved.

1. Introduction

The COVID-19 pandemic has changed various aspects of our societies, ranging from public health and economic conditions to human rights. Two other recent coronavirus pandemics, Severe Acute Respiratory Syndrome (SARS) in 2002 and Middle East Respiratory Syndrome (MERS) in 2013, have produced 8437 and 2519 cases, respectively. On the other hand, within two years, there have been about 35 billion cases of COVID-19 and 4 million resulting deaths. This is mainly due to an abnormally high transmission rate and asymptomatic spreading [1,2]. Many countries are trying to vaccinate people, but it is difficult to produce a sufficient amount of vaccines in a short time to supply the whole world aside from their safety. Moreover, the highly contagious mutant viruses have been continuously detected. In case the vaccine has not been developed yet or is insufficient, non-pharmaceutical interventions such as social distancing among individuals, masking, and reinforcing personal hygiene are alternative approaches to prevention.

Beginning with Wuhan [3,4] in China, the majority of countries implemented a lockdown policy as an initial response to face COVID-19. The policy restricts travel from other countries and prevents people from participating in non-essential social activities. Much research has been done on the efficient implementation of the policy or its effectiveness with mobility data and metapopulation model [5,6]. However, such lockdown prevention is not sustainable, because it drastically reduces economic activities [7,8]. Indeed, the majority of countries that adopted the lockdown policy failed to sustain it for more than two to three months; they gradually returned to their former policies. Thus, related issues, such as the possibility of second waves and an exit strategy from the lockdown prevention, were addressed and studied [8–10].

The Korean Center for Disease Control and Prevention (KCDC) has achieved great success in the early stage of the epidemic using the so-called K-quarantine measure, which enforces local quarantine around confirmed patients rather than implementing a global lockdown. This approach, implemented by contact tracing and aggressive quarantine, efficiently prevents the spread of disease without critical economic damage.

Contact tracing is a primitive but efficient strategy to select people who require treatment or isolation. However, owing to high social costs and privacy invasions, this strategy has been applied to limited cases with low infection rates, such as sexually transmitted diseases [11] or early-stage SARS [12]. A simple theoretical model [13] was proposed and numerically investigated. For the simple model, the relationship between the infection rate of the target disease and the frequency of contact tracing was studied. This study was extended to more practical quantities, such as latent time and the rate of asymptomatic infection [14] for the spread of the SARS-CoV-2 virus [15].
In this paper, a K-quarantine model is proposed based on the basic principles of the K-quarantine measure (contact tracing and quarantine). The model was simulated on several types of complex networks, where the links of a node were disconnected when the node was quarantined and reinstated when it was released. The model control parameters were estimated based on the empirical data of the spread of the SARS-CoV-2 virus during its early stages in South Korea. The accumulated number of confirmed cases and the time-dependent reproduction number were successfully reproduced from simulations in the early stage. Further, the model was applied to an empirical network and a synthetic network with a modular structure, and the patterns of the number of daily confirmed cases were investigated.

The remainder of this paper is organized as follows. Sec. II describes the complex networks in which the model is simulated. Sec. III describes the K-quarantine model constructed using various reaction parameters. Sec. IV presents the estimation of the numerical values of the reaction parameters, which was achieved by comparing them with empirical data and simulating the model. Macroscopic measurable quantities, such as the accumulated number of confirmed cases and daily confirmed cases, were obtained from the model and compared with the empirical data. Sec. V discusses the consideration of temporally changing cases of reaction rates and network structure to reflect the empirical situations in which the virus species was changed and a social gathering in a street demonstration, respectively. A synthetic lockdown situation, which can be realized by deleting a fraction of links, was also considered. A summary of this study is presented in Sec. VI.

2. Networks

It is worth examining how the epidemic contagion spreads under the K-quarantine model as compared to its spread under global lockdown without local restrictions. To achieve this, a mathematical model is considered in this work. The conventional epidemiological model is a compartmental model in which each person is considered to be in one of the following possible states: susceptible (S), latent (L), infected (I), or recovered/deceased (R). The proportions of people in each state are regarded as continuous variables, and their rate equations (time derivatives) are set up as a function of these proportions with appropriate rate constants. By solving these differential equations, the fraction of each state as a function of time is obtained. In the past, this approach has successfully predicted the evolution of the fraction of infected populations. However, it may not be useful when considering the local quarantine effect under the K-quarantine measures stated above.

Here, the epidemic reactions are simulated on networks. A network is composed of nodes and links, which represent people and contact between a pair of connected people, respectively. The numbers of nodes and links that are simulated are taken as \( N = 2.1 \times 10^4 \) and \( L = 5 \times N \), respectively. This implies that a society composed of \( N \) people is being considered, and the average number of people in contact with each person (called the 'degree' in graph theory) is given as \( \langle d \rangle = 2L/N = 10 \). Note that these links are static and do not change temporally with people’s movement. One may suppose that these are essential links, representing close contact with family members and colleagues in the workplace everyday. In contrast, contact with people who met occasionally or only once (such as in street demonstrations or jazz bars) is called loose contact. The contagion through loose contact is implemented as follows. Once extra links are connected between susceptible and infectious nodes, random selection is performed, and it is then investigated whether the susceptible nodes are infected by the infectious nodes with a given infection rate. The infected nodes change their states to latent states. Subsequently, the extra links are disconnected. In simulations, this process is simply implemented as follows. A proportion of susceptible nodes is selected, and their state is changed to the latent state. The contagion through a large street demonstration around \( t = 209 \) is realized in this manner.

The K-quarantine process is realized by locally disconnecting the links to an infected node. As soon as the quarantine is completed and the patient is released, these links are reinstated. In the K-quarantine model, once a person is quarantined, they are required to take a diagnostic test. If the result is positive, then the people in contact with the patient are quarantined. Thus, links connected to the neighbors of the confirmed patient also need to be disconnected.

Networks are classified into two types based on their connection configurations: random networks and scale-free networks. For random networks, each link is added between two randomly selected nodes. Thus, the degree of each node has a Poisson distribution. Because this model was first proposed by Erdős and Rényi, it is often called the ER model [16]. For scale-free networks, following the power law, the degree of each node is heterogeneous. This implies that a few nodes have large degrees, but the remaining nodes have small ones. The nodes with large numbers of neighbors are called hubs. When a hub is infected, a large number of susceptible neighbors are exposed to the contagion. This may result in a spike in contagion. Scale-free networks were constructed using the models proposed by Goh et al. [17] and Chung and Lu [18].

3. Models

The epidemic reactions proceed as per Markovian dynamics, which are realized by the Gillespie algorithm [19,20]. Each node is in one of the following states [21,22]: susceptible (S), latent (L), asymptomatic infectious (\( I_a \)), symptomatic infectious (\( I_s \)), asymptomatic in quarantine (\( I^a \)), symptomatic in quarantine (\( I^s \)), or recovered (\( R, R' \), or \( R^* \)) [23–25]. The states of susceptible in quarantine (\( S' \)) and latent in quarantine (\( L' \)) also exist. The dynamic begins with one infected person, with all the others being in a susceptible state. When nodes in states \( L, I_a, \) and \( I_s \) are absent, the dynamic falls into an absorbing state, and the nodes in state \( S \) or \( R \) remain. The detailed dynamics are as follows:

A susceptible individual in contact with an infectious individual \( I_a \) and \( I_s \) enters the latent state (\( L \)) at the rate \( k_1 \). These reactions are expressed as

\[
S + I_a \xrightarrow{k_1} L + I_a \quad \text{and} \quad S + I_s \xrightarrow{k_1} L + I_s.
\]  
(1)

When the latency period ends, the individual becomes infectious, that is, they can transmit the infection with or without symptoms. These states are denoted as \( I_a \) or \( I_s \), respectively. These processes occur at rates \( k_2p_a \) and \( k_2(1 - p_a) \), respectively. Here, \( p_a \) represents the fraction of asymptomatic infectious patients. These reactions are expressed as

\[
L \xrightarrow{k_2p_a} I_a \quad \text{and} \quad L \xrightarrow{k_2(1 - p_a)} I_s.
\]  
(2)

When symptoms develop, the infected individual must go to the hospital and take a diagnostic test. If the result is positive, they are quarantined. This process occurs at the rate \( k_3 \) and is expressed as

\[
I_a \xrightarrow{k_3} I^a,
\]  
(3)

where the prime indicates that the individual is quarantined. On the other hand, an asymptomatic individual may recover naturally without any treatment. This process occurs at the rate \( k_4 \) and is expressed as

\[
I_a \xrightarrow{k_4} R.
\]  
(4)

The isolated individual in state \( I^a \) may be recovered through treatment or succumb to the disease. This recovered individual is
counted as a confirmed case of recovery, denoted by $R^t$. This process occurs at the rate $k_5$ and is expressed as

$$I'_t \xrightarrow{k_5} R^t.$$  

(5)

In the K-quarantine model, confirmed cases ($I'_t$) and their neighbors are self-quarantined as potential infectious people even if they are asymptomatic. Regardless of their state being $S, L, I_k, \text{ or } R$, they are quarantined at the rate $k_6$. This process is expressed as

$$I'_t + X \xrightarrow{k_6} I'_t + X', \quad X \in \{S, E, I_0, I_1, R\}.$$  

(6)

where $k_0$ is the quarantine rate. Because quarantined individuals must undergo a diagnostic test, isolated asymptomatic carriers $I'_t$ are identified as confirmed cases. Accordingly, the neighbors of the identified asymptotic carrier are also quarantined at the rate $k_6$:

$$I'_t + X \xrightarrow{k_6} I'_t + X', \quad X \in \{S, E, I_0, I_1, R\}.$$  

(7)

This trace process is repeated until no further confirmed cases are identified. [25,26]. During the quarantine period, identified asymptomatic infected individuals recover at the rate $k_4$, expressed as

$$I'_t \xrightarrow{k_4} R^t.$$  

(8)

Here, it is assumed that asymptomatic patients have the same recovery rate $k_5$ regardless of isolation. Individuals in the states $S, L'$, or $R'$ with negative diagnostic test results are released from quarantine. They then return to their original states.

$$X' \xrightarrow{\tau} X, \quad X \in \{S, L, R\},$$  

(9)

where $\tau$ is the quarantine period (not the rate).

The reproduction number (denoted as $R_0$), the number of individuals who are susceptible and become infectious by contacting an infected individual, is calculated as $R_0 = k_6 (d)/k_7$, where $(d)$ is the mean number of neighbors on a given network. Herd immunity is the level of immunity in a population that prevents the spread of a disease over the entire system. The herd immunity threshold is described as $R_e = 1 - (1/R_0)$ [27,28].

4. Reaction rates

To explore the effect of the self-quarantine measure on the transmission of COVID-19, the rates $k_2 - k_5$ and $p_0$ were estimated based on empirical data on COVID-19 provided by the Korea Center for Disease Control (KCDC) and others: First, to find the rate $k_2$ and $p_0$, the period between exposure and the onset of symptoms is used. This interval was estimated to be 6 (mean) days [5,31]. The infected individual can transmit the disease $1 - 3$ days before the onset of symptoms [1]. Thus, the interval between exposure and becoming infectious is estimated to be 4 (mean) days [6,32–35]. We take $k_2 (1 - p_u) = 0.25$. The resulting data show that the percentage of asymptomatic infections is estimated to be 15%–40% [36–38]. We take $p_u = 0.36$. Thus, $k_2 = 0.39$ and $k_5 p_u = 0.14$.

In South Korea, a potential symptomatic infectious individual develops symptoms and is then quarantined approximately in three days. Thus, $k_3 \approx 0.33$ is set. Further, it takes approximately 9 and 12 days for an asymptomatic carrier and a confirmed infected individual, respectively, to recover. Thus, $k_4 \approx 0.11$ and $k_5 \approx 0.08$ were set. $\tau$ was taken to be 14 days.

The infection rate $k_1$ is estimated using the relation $R_0 = k_1 (d)/k_7$ at the beginning of epidemic. Using the rate $k_3 = 1/3$ and the mean degree $(d) = 10$, $k_1 = 0.11$ is obtained when $R_0 = 3.3$ is taken [31]. Using these parameter values, it is observed that the simulation result fits the empirical data from the early stages of the COVID-19 outbreak in South Korea (March 2020) to the end of August. For the same outbreak, the value of $R_0$ directly measured from the empirical data is $R_0 \approx 3.2$ [39]. Note that when the spread of an epidemic is simulated on a scale-free network, the reproduction number is expressed as $R = [k_1 ((d^2) - (d))/k_7 (d)]$ [40]. This formula is reduced to $k_1 (d)/k_7$ when the degree distribution follows a Poisson distribution.

Here, the K-quarantine model was simulated with fixed rates $(k_1 - k_5)$ and $p_0$, and a controllable quarantine rate $(k_6)$ on several types of networks. These included random networks (Fig. 2(a)–(d)) scale-free networks (Fig. 2(e)), an empirical social network (Fig. 2(f)), and random networks with modules (Fig. 2(g)–(h)) [41,42]. It should be noted that all the rates are fixed throughout the epidemic spreading process unless otherwise specified. The proportions of nodes in each state are measured as a function of time in days. Note that the quarantine rate $k_6$ is taken as $k_6 = 0.09$. This value was obtained using a simulated annealing method. This method enables the calculation of an optimal parameter value that minimizes the mean-square error between the accumulated numbers of confirmed cases obtained by simulations and empirical values. The details of the algorithm are presented in Appendix B.

5. Temporal behaviors of several quantities

In Fig. 2(a), the SEIR model [44–46] is considered without any quarantine on random networks. Thus, $k_6 = 0$ was set. Initially, one node is assumed to be infected, while the other nodes are susceptible. The fractions $\rho_S (t), \rho_R (t), \rho_E$, and $\rho_I$ are obtained, where $\rho_S = \rho_{Rc} + \rho_{Rg} + \rho_{Rc} + \rho_{Rg}$, and the dot represents the time derivative. $\rho_C$ and $\rho_R$ represent the proportions of newly confirmed cases and the accumulated confirmed cases, respectively. The three densities are shown in Fig. 2(a). The contagion spreads rapidly during the early stage and eventually reaches a steady state. As shown in Fig. 2(b) with $k_6 = 0.09$, when the quarantine system is functioning, the fraction $\rho_C$ initially increases rapidly, then slowly increases with some fluctuations, and finally reaches a steady state. Resurgent behavior is observed in $\rho_C$. Further, it is noted that for the system with no quarantine strategy, the absorbing state of the infectious node completely disappears on reaching the 150th day, whereas for the K-quarantine system, it reaches the 400th day. The proportions of the accumulated confirmed cases for (a) and (b) are close; however, the proportion of remaining susceptible people is extremely small for (a), but it is more than 20% for (b). On the other hand, the fraction of asymptomatic infected patients appears to be about 30% for (a), but it is approximately 10% for (b). This is because asymptomatic patients can be detected when they are in quarantine owing to the infection of their neighbors.

Figure 2(c) depicts the case in which the infection rate $k_1$ suddenly increases to $k_1 = 0.41$ at $t = 108$, owing to the change of virus species from S or V to GH clade [29]. There exists another significant peak of $\rho_C$ around $t = 130$, and the infection rate increases dramatically. Following this, the system reaches a steady state. The density of $\rho_C$ in the steady state increased by 22.72% compared to that of the case (b). However, no such dramatic change is observed in the empirical data. Figure 2(d) depicts the case in which the quarantine system is overloaded and does not act at a certain time (e.g., $t = 108$). Then, $\rho_C$ instantaneously exhibits resurgent behavior, and $\rho_R$ rapidly increases and reaches a steady state, as in the SIR model.

Next, the K-quarantine model is simulated on a scale-free network in Fig. 2(e) and on an empirical social network in Fig. 2(f). It seems that the contagion pattern is insensitive to network structural type, because the overall patterns of (e) and (f) are similar to that of (b), even though the absolute values of the accumulated number of confirmed cases are different. This seems to be counterintuitive because a scale-free network contains a few super-spreaders. Thus, if they are infected, many susceptible nodes linked to them could be infected, and the proportion of infectious nodes would increase drastically. However, the pattern of increase
Fig. 1. Flowchart for the K-quarantine model. The states under quarantine are represented by squares, while others are represented by stadiums.

Fig. 2. Plot of the densities $\rho_S(t)$, $\rho_L(t)$, $\rho_R(t)$, and $\rho_C(t)$, where $\rho_C$ denotes $\rho_R + \rho_I + \rho_S$ as a function of $t$ for the Susceptible-Exposed-Infected-Recovered (SEIR) model. These represent the proportions of susceptible individuals, recovered individuals without noticing, newly confirmed cases, and accumulated confirmed cases, respectively. The rates are taken as $k_1 = 0.11$, $k_2 = 0.33$, and $k_3 = 0.11$. (a) Simulations are performed on ER random networks without the K-quarantine measures. System size $N = 2.1 \times 10^4$, the mean degree $\langle d \rangle = 10$, and $k_6 = 0$ are set. (b) Similar plot to (a), but under the K-quarantine strategy $k_6 = 0.09$. (c) Similar plot to (b), but the rate $k_1$ changes suddenly at $t = 108$ to $k_1 = 0.41$. This change is caused by a new type of coronavirus, GH clade [29]. (d) Similar plot to (b), but the rate $k_6 = 0$ at $t = 108$. This change is considered to occur because the quarantine system no longer functions owing to overloading. (e) and (f) Similar plots to (b), but simulations are performed on scale-free networks with degree exponent $\lambda = 2.5$ [18] and on an empirical social network [30], respectively. For (g), $N = 21403$ and (d) = 7.8. Owing to this smaller mean degree, the contagion rate is lower. (g) and (h) Similar plots to (b), but on modular networks. The network is composed of $N_m$ modules and each module contains $N_n$ nodes and has the mean degree of intra-module edge $\langle d_{\text{intra}} \rangle = 10$. Those modules are connected through $L_m$ inter-modular links. For (g), $N_m = 10$, $N_n = 10^4$, and $L_m = 200$ are set. For (h), $N_m = 10^1$, $N_n = 10^2$, and $L_m = 10^4$ are set.
is similar because the K-quarantine measure is relatively effective in contact tracing [14]. Information related to the times and places of visit by an infectious person is collected, and the people at these places and times are traced within a short period using various methods. These people self-quarantine themselves or are quarantined at an isolated place, even for the contagion by a super-spreader. This containment still works effectively. Therefore, the overall contagion pattern is slightly sensitive to the type of network structure (either heterogeneous or homogeneous).

The scale of the second wave, however, is moderately dependent on the type of network structure. Comparing (b) and (e) in Fig. 2, we find that the peak of the second wave is slightly higher for a scale-free network (e) than that of (b) for a random network, which may be caused by the epidemic of super-spreaders. The ratio of the peak height of the second wave to that of the first wave is approximately 27% for a scale-free network, which is comparable to approximately 11% for a random network. Note that for the empirical network (coauthorship network) (f), the mean degree is smaller than that of (a)-(e). Thus, the accumulated fraction of confirmed cases in the steady state was considerably smaller. Nevertheless, the peak of the second wave is higher.

For (g)-(h), simulations are performed on modular networks [41,47]. The modular networks are composed of $N_m$ modules, each of which contains $N_h$ nodes. Thus, the total number of nodes in the system is $N_m N_h$. Nodes within each module are connected to each other randomly with mean degree $(d_{\text{intra}}) = 10$. To make the modules connected, $\ell_m$ pairs of modules are selected randomly, each of the pairs is connected by $\ell_p$ links by selecting $\ell_p$ nodes from each module. Thus, the total number of inter-modular edges is $L_m = \ell_m \ell_p$. Specific those numbers are listed in the caption of Fig. 2(g)-(h).

Figure 3(a) and (b) depict the cases in which the system is locked-down for 60 days. The lockdown can be realized by either social distancing or restriction of transportation [5,6,8]. In (a), the lockdown is implemented by deleting the fractions of links (indicated in the legend) randomly selected on the 30th day. After 60 days, those links are recovered. When the fractions are below 50%, the lockdown effect is almost negligible. On the other hand, when the fraction is 90%, then the epidemic spread is highly suppressed. In the intermediate range, a resurgent behavior appears. However, such behaviors fluctuate depending on the density of infectious nodes on the 30th day. Thus, in (b), we consider the case that the starting day of lockdown measure is determined by the fraction of accumulated confirmed cases, called the lockdown threshold. Once the lockdown comes into force, 70% of links are deleted and they are recovered after 60 days. Depending on the threshold value, the time of the resurgent peak is determined. In short, while the lockdown measure during the 60 days is effective during some intervals, the outbreak eventually occurs. Note that the fraction, for instance, 50%, does not indicate 50% of a real system. The value is meaningful only in the simulated model network.

In Fig. 3(c), a small number of the nodes in state $S$ every day change their state to $L$. This change is considered to occur when individuals from abroad become new sources of the epidemic. Because in this case, no root is found explicitly and implicitly in the

**Fig. 3.** Similar plots to Fig. 2(b). (a) But, at $t = 30$, some fractions of links (indicated in legend) are artificially deleted. This change is considered to occur when a global lockdown is functioning. Depending on the fraction, diverse temporal patterns of $r_C$ appear. (See the details in the main text.) (b) Similar plot to (a) but the lockdown is functioning when the fraction of accumulated confirmed cases reaches a threshold value given in the legend. Then 70% links are deleted at random. (c) Similar plot to Fig. 2(b), but a small fraction of the nodes in state $S$ randomly selected every day are forced to change their state to the latent state from the specified day. This is caused by the transmission of the disease by people from abroad. (d) Similar plot to Fig. 2(b), but a large fraction of the nodes in state $S$ instantaneously change state to $L$ on a single occasion [43]. This change reflects the transmission of the disease by close contact among people participating in a large street demonstration.
trail of disease transmission, the pattern of spread may somewhat differ from the previous patterns. In Fig. 3(d), a large fraction of the nodes in state $S$ instantaneously change state to $L$. This change is considered to occur by the transmission of disease among people participating in a large street demonstration at $t = 209$ owing to their close contact and shouting.

In Fig. 4, the simulation results are compared with the empirical data of South Korea, starting from January 20, 2020, and accumulated as of September 9th, 2020. It is observed that the increasing behavior of the accumulated number of confirmed cases from the model during the early stage is well-fitted to the empirical data with the rates assumed herein. However, there is some difference during the intermediate stage, which may be due to the unexpected social event (a festival opening in a club) that was held shortly after reducing the level of social distancing. In the latter region, the number of confirmed cases abruptly increases owing to the large demonstration on the main street near the city hall in Seoul. Among over 10,000 people participating in the demonstration, a non-negligible portion of them did not wear masks. Therefore, the disease transmission would be high. The model proposed herein cannot reproduce the output of such a large-scale perturbation. Instead, some portion (80%) of the remaining susceptible nodes were changed to nodes in the latent state, under the assumption that those portions of people are infected in high-risk areas. With the passage of time, the surge decreases owing to the K-quarantine measures.

In Fig. 5, three time-dependent reproduction numbers $R(t)$ are plotted from the empirical and simulation data. $R(t)$ can be measured using two methods. Method (i), used in (a), utilizes the number of daily confirmed cases $[48]$. $R(t)$ is obtained as the ratio of the number of new infectious patients $k_i$ generated at time step $t$ to the total number of infectious patients during all preceding time steps, that is,

$$R(t) = \sum_{s=1}^{t} k_i w_s,$$

where $w_s$ is the weight that a person infected at a given time remains in the infectious state after $s$ time steps. Moreover, the ratio is averaged over a time window of size $t = 7$ (days) ending at time $t$. Accordingly, the curve exhibits low noise.

In (b), method (ii) is used to estimate $R(t)$ from simulation data with and without a quarantine process. The formula $k_1 (d(t))/k_3$ is used to obtain $R(t)$. While $k_1$ and $k_3$ are fixed, the mean number of susceptible neighbors of each infectious node at a given time $d(t)$ is variable. Owing to the different methodologies, there exists a time delay between the estimated $R(t)$ curves. To make them overlap, $R(t)$ obtained by method (ii) is shifted by 12 days to the left in the early stage.

While $R(t)$ curves calculated from two different methods are close to that from empirical data in the early stage and $t > 105$, they are not in agreement with each other in the intermediate region $62 < t < 105$. This deviation may be caused by an increase in the level of social distancing by the Korean government. Note that $R(t)$ decreases to zero as the system reaches a steady state, in which a new infection rarely occurs, even though there exist a nonzero fraction of nodes in the infectious state. This case occurs when the infectious nodes are surrounded by the recovered nodes. This state may be called a herd-immunized state.

6. Discussion and conclusion

This paper proposed an epidemic contagion model on complex networks to investigate the effects of contact tracing and local quarantine on the spread of an epidemic disease, COVID-19, in South Korea. Contact tracing and quarantine are essential factors of the “K-quarantine measure” in South Korea. Under this measure, information on the spatial and temporal trajectories of infected individuals is collected not only by self-statement but also by using mobile phone data. This information is used to find other individuals who were present at the same place and time, who are then
requested to undergo a diagnostic test. If infected, they are quarantined. The process is then repeated. Thus, contact tracing and quarantine are effectively implemented in South Korea, and a large outbreak is prevented. 

In this study, the contact tracing and quarantine were modeled by disconnecting links to an infected node. As the node was released from quarantine following a given period, the links were reinstated. This process was repeated as new individuals became infected. The model contained seven parameters, which were determined based on empirical data. It was demonstrated that after determining these parameter values, the proposed model reasonably produced the empirical data of the accumulated and daily numbers of confirmed cases. However, owing to the limitation of the system size, the proportion of accumulated confirmed cases already reached a steady state at \( t \approx 230 \) days in the performed simulations. Thus, it can be concluded that this simple network model is useful for predicting the pattern of epidemic spread for the near future. Moreover, a large number of asymptomatic infected patients were detected, as they were quarantined with infected people. In contrast, in the K-quarantine measure, the level of social distancing is adjusted on a timely basis, depending on the number of daily confirmed cases. Accordingly, the infection rate and other parameter values must be updated in the model. However, establishing a general relationship between the level of social distance and the infection rate remains an important future task.

Simulations were performed on several types of networks, including random, scale-free, and modular structures. The patterns of epidemic spreading on each type of network were compared, and it was concluded that the overall pattern of the accumulated number of confirmed cases is insensitive to the network type. This is because, regardless of the number of susceptible nodes infected by an infectious node, they are quarantined. The network structure of the proposed model is static because the links do not consider human mobile behavior. This is because the links represent close contact among family members and people in the same workplace. To represent the contagion through loose contacts, a proportion of susceptible nodes were randomly selected. These were then regarded as newly infected nodes through loose contacts. This is a simple approach. Further, the possible consequences of the breakdown of local quarantine measures and social distancing were also investigated.

Notably, the time-dependent reproduction number \( R(t) \) was considered. It was found that the theoretically obtained \( R(t) \)'s value is in good agreement with the empirical data in the early epidemic stage. It decreases to zero as the system reaches a steady state in a finite system. However, \( R(t) \) still changes in the real system, because the pandemic is still ongoing as of January 2022.

Vaccination strategy is indeed an important issue, particularly when vaccines cannot be sufficiently supplied to the majority of the population. Many researches have been performed to improve the vaccination strategy, for instance, [49]. In the K-quarantine measure, because the infected individuals are traced, at first glance, it would be reasonable to vaccinate the susceptible people around infected people in the network. However, the model we considered in the paper is constructed on static (not temporal) networks. Thus, this vaccination strategy would not be sufficiently effective in real systems.

In summary, the K-quarantine measure was modeled on complex networks. In this model, infection and quarantine processes are implemented locally and stochastically, in contrast to the homogeneous method over the system in the numerical method using the compartment equation. With the appropriate choice of parameter values from the empirical data, the model successfully reproduced the patterns of the accumulated and daily numbers of confirmed cases. Thus, it can be used to predict the near future pandemic patterns.

### Declaration of Competing Interest

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

### CRediT authorship contribution statement

**K. Choi:** Conceptualization, Methodology, Software, Investigation, Writing – original draft. **Hoyun Choi:** Conceptualization, Methodology, Software, Investigation, Writing – original draft. **B. Kahng:** Conceptualization, Investigation, Writing – review & editing, Funding acquisition, Supervision.

### Acknowledgments

This research was supported by the NRF, Grant No. NRF-2014R1A3A2069005 (BK). BK thanks Prof. H. B. Kim MD for helpful discussions.

### Appendix A. Simulation algorithm

The probability that the state \( X_i \) of a node \( i \) changes the state \( A \) from \( B \) in unit time is called the transition rate \( r_{X_iA \rightarrow B} \). For example, in the K-quarantine model, the probability that a node in the susceptible state \( S \) moves to the latency state \( L \) is as follows:

\[
r_{X,S \rightarrow L} = k_1 \sum_j A_{ij} (\delta(X_j, L) + \delta(X_j, L))
\]

where \( A_{ij} \) is the adjacency matrix of the network, and \( \delta(X_j, L) \) is the Kronecker delta.

When the K-quarantine model is simulated through the discrete-time approach method, the state of each node is changed independently depending on the reactions in Fig. 1. On the other hand, the time interval \( \tau \) is constant [50]. It is possible that node \( i \) in state \( A \) changes to \( B_1, \ldots, B_M \), and the transition rate of node \( i \) is expressed as

\[
r_i = \sum_{a=1}^{M} r_{X_iA \rightarrow B_a}.
\]

Assume that the reactions follow the Poisson process. Then, for a given time interval \( \tau \), the probabilities \( p_i \) and \( p_{X_iA \rightarrow B_a} \) that the state of the node \( i \) changes to another state and to a specific state \( B_a \), respectively, are obtained as follows:

\[
p_i = 1 - e^{-r_i \tau}, \quad p_{X_iA \rightarrow B_a} = \frac{r_{X_iA \rightarrow B_a}}{r_i} p_i.
\]

Therefore, a random number \( u \) is chosen from the uniform random distribution following \( (0 : 1) \). If \( u > p_i \), the state of node \( i \) is not changed; else, the state of node \( i \) may be changed to \( B_\beta \) as follows:

\[
\sum_{a=1}^{\beta-1} r_{X_iA \rightarrow B_a} < u \leq \sum_{a=1}^{\beta} r_{X_iA \rightarrow B_a}.
\]

Following the update rule of the states of all nodes in parallel, this increases the time by \( \tau \).

The Gillespie algorithm [19] was also employed for the numerical simulation of the K-quarantine model. Because this algorithm adjusts the time interval \( \tau \) according to the transition probability, it is widely used to simulate stochastic epidemic models in real time [20,51]. The cumulative transition rate \( r \) is the probability that at least one reaction occurs per unit time,

\[
r = \sum_{i=1}^{N} r_i,
\]
It is well known that the time interval $\tau$ in which at least one reaction occurs follows an exponential distribution with a mean of $1/r$.

$$P(\tau) = re^{-\tau r}$$  \hspace{1cm} (15)

Two random numbers $u_1$ and $u_2$ were chosen from the uniform random distribution following $(0:1)$, respectively. Then, the time interval $\tau = \frac{1}{r} \ln \frac{1}{u_1}$ was found, and the node $j$ and state $B_j$ were obtained as follows:

$$\sum_{i=1}^{j} \sum_{\beta=1}^{\beta} I_{X_i A \rightarrow B_j} < u_2 \leq \sum_{i=1}^{j} \sum_{\beta=1}^{\beta} I_{X_i A \rightarrow B_j}$$  \hspace{1cm} (16)

Following the change of the state of node $j$ to state $B_j$, the time increases by $\tau$.

Appendix B. Simulated annealing

The simulated annealing method was used to determine the quarantine ratio $k_0$. The obtained value of $k_0$ minimizes the difference between the number of accumulated confirmed cases obtained by simulations and the empirical data of South Korea. A target function $E$, often referred to as “energy,” was proposed as follows:

$$E = \frac{1}{T} \sum_{t=1}^{T} (N(t) - \tilde{N}(t))^2.$$  \hspace{1cm} (17)

where $N(t)$ and $\tilde{N}(t)$ are the accumulated numbers of confirmed cases from the simulations and empirical data, respectively, as shown in Fig. 4. $T$ is the time interval of the target function to be minimized. The simulated annealing algorithm is based on the Monte Carlo algorithm, which is often used in equilibrium statistical mechanics. Thus, it uses the Boltzmann factor $e^{-\Delta E/T_c}$ where $T_c$ denotes the temperature. The algorithm is executed as follows:

**Algorithm 1 Simulated Annealing.**

1. Set temperature $T_c \gg 1$, energy $E = \infty$
2. Randomly choose $k_0 = k_0^{(0)}$
3. while $T_c \geq T_{c_{min}}$ do
   a. sufficiently small $T_{c_{min}}$
   b. Perturbate $k_0$
4. for $j = 1, 2, \ldots, \max$ do
   a. $k_0^{(j)} = k_0^{(j-1)} + \Delta k_0$
   b. Simulate $N(t) (k_0 = k_0^{(j)})$
6. calculate $E^{(j)}$
7. $k_0 = k_0^{(j)} \leftarrow k_0^{(j)}$ if $E^{(j)} < E^{(j)}$ with prob max$(1, e^{-\Delta E/T_c})$
8. $\Delta E = E^{(j)} - E^{(j-1)}$
9. end for
10. $T_c \leftarrow T_c/2$
11. end while

The algorithm perturbs $k_0$ randomly and then checks whether this perturbed value produces an energy value lower than the previous one. If it does, then $k_0$ is replaced by the perturbed one. Otherwise, the updated $k_0$ is taken with the probability following the Boltzmann factor $e^{-\Delta E/T_c}$ in line 8. This method of updating is similar to the Metropolis algorithm often used in equilibrium thermodynamic systems, which enables the parameter value to converge to the empirical value.

The simulated annealing algorithm was initiated with a rather high temperature. This choice enables the search for a global minimum by jumping over the local minima in the configuration space. To more precisely obtain a global minimum, the search was repeated $\max$ times at a given $T_0$, and the temperature was decreased. As these steps were repeated, the rate $k_0$ converged. An optimal value of $k_0 = 0.09$ was obtained. As shown in Fig. 4, using this value, the accumulated number of confirmed cases was obtained. This number was found to be close to that of the empirical data. Moreover, the time-dependent reproduction number $R(t)$ was obtained as shown in Fig. 5.

**References**

[1] Sanche S, Lin YT, Xu C, Romero-Severson E, Hengartner N, Re K. High contagiousness and rapid spread of severe acute respiratory syndrome coronavirus 2. Emerg Infect Dis 2020;26:1470. doi:10.3201/eid2607.200282.

[2] Liu Y, Gayle AA, Wilder-Smith A, Rocklov J. The reproductive number of COVID-19 is higher compared to SARS coronavirus. J Travel Med 2020;27:1–4. doi:10.1093/jtm/laaa021.

[3] Wu JT, Leung K, Leung GM. Nowcasting and forecasting the potential domestic and international spread of the 2019-nCoV outbreak originating in Wuhan, China: a modelling study. Lancet 2020;395:689–97. doi:10.1016/S0140-6736(20)30266-9.

[4] Wu JT, Leung K, Bushman M, Kishore N, Niehus R, de Salazar PM, et al. Estimating clinical severity of COVID-19 from the transmission dynamics in Wuhan, China. Nat Med 2020;26:506–10. doi:10.1038/s41591-020-0882-7.

[5] Chinazzi M, Davis JT, Ajelli M, Gozdzewki C, Litvinova M, Merler S, et al. The effect of travel restrictions on the spread of the 2019 novel coronavirus (COVID-19) outbreak. Science 2020;368:395–400. doi:10.1126/science.abb9757.

[6] Kraemer MU, Yang CH, Gutierrez B, Wu CH, Klein B, Piggott DM, et al. The effect of human mobility and control measures on the COVID-19 epidemic in China. Science 2020;368:493–7. doi:10.1126/science.abb3184.

[7] Roux J, Massonnaud C, Colizza V, Cauchemez S, Crépèy P. Impact of national and regional lockdowns on COVID-19 epidemic waves: application to the 2020 spring wave in France. medRxiv 2021. doi:10.1101/2021.04.21.21253876.

[8] Mahteshvari P, Albert R, Network model and analysis of the spread of COVID-19 with social distancing. Appl Netw Sci 2020;5:1–13. doi:10.1007/s41109-020-00344-5.

[9] Aleta A, Martin-Corral D, Piontti A, Ajelli M, Litvinova M, Chinazzi M, et al. Modelling the impact of testing, contact tracing and household quarantine on second waves of COVID-19. Nat Hum 2020;4:964–71. doi:10.1038/s41562-020-0931-9.

[10] Cattuto C, Perra N, Vespignani A, et al. On the use of telecommunication data in epidemiology. PLoS One 2009;4(11):1–7. doi:10.1371/journal.pone.0003012.

[11] Firth JA, Hellewell J, Klepac P, Kissler S, Kucharski AJ, Spurgeon LG. Using a real-world network to model localized COVID-19 control strategies. Nat Med 2020;26:1616–22. doi:10.1038/s41591-020-1036-8.

[12] Erdős P, Rényi A. The evolution of random graphs. Pub Math Inst Hung Acad Sci Ser A 1960;5:17–60. doi:10.1007/BF02196505.

[13] Goh KI, Kahng B, Kim D. Universal behavior of load distribution in scale-free networks. Phys Rev Lett 2001;87:287801. doi:10.1103/PhysRevLett.87.287801.

[14] Chung F, Lu L. Connected components in random graphs with given expected degree sequences. Ann Comb 2002;6:125–45. doi:10.1007/s00026001002580.

[15] Gillespie DT. Exact stochastic simulation of coupled chemical reactions. J Phys Chem 1977;81:2340–61. doi:10.1021/j100540a008.

[16] Vestergaard CL, Génois M. Temporal Gillespie algorithm: fast simulation of contagion processes on time-varying networks. PLoS Comput Biol 2015;11:11–28. doi:10.1371/journal.pcbi.1004579.

[17] Chen TM, Rui J, Wang QP, Zhao ZY, Liu JA, Yin L. A mathematical model for simulating the phase-based transmission of a novel coronavirus. Infect Dis Poverty 2020;9:1–8. doi:10.1186/s40249-020-00640-3.

[18] He S, Peng Y, Sun K. SEIR modeling of the COVID-19 and its dynamics. Nonlinear Dyn 2020;101:1660–85. doi:10.1007/s11071-020-05743-y.

[19] Furukawa NW, Brooks JT, Soibel J. Evidence suggesting transmission of severe acute respiratory syndrome coronavirus 2 while presymptomatic or asymptomatic. Emerg Infect Dis 2020;26:E201959.

[20] Xie C, Cheng S, Wu J, Wu T, Xin H, Wang C. Reconstruction of the full transmission dynamics of COVID-19 in Wuhan. Nature 2020;584:420–4. doi:10.1038/s41586-020-2554-8.

[21] Reyna-Lara A, Soriano-Paños D, Gómez S, Granell C, Matamalas JT, Steinger B, et al. Virus spread versus contact tracing: Two competing contagion processes. PloS Rev Res 2021;3:003163. doi:10.1371/journal.pcrs.003163.

[22] Kucharski AJ, Klepac P, Conlan AJ, Kissler SM, Tang ML, Fry H, et al. Effective isolation of testing, contact tracing, and physical distancing on reducing transmission of SARS-CoV-2 in different settings: a mathematical modelling study. Lancet Infect Dis 2020;20:1511–60. doi:10.1016/S1473-3099(20)30457-6.

[23] Randolph HE, Barreiro LB. Herd immunity: understanding COVID-19. Immunity 2020;52:737–41. doi:10.1016/j.immuni.2020.04.012.
[28] Gani R, Hughes H, Fleming D, Griffin T, Medlock J, Leach S. Potential impact of antiviral drug use during influenza pandemic. Emerg Infect Dis 2005;11:1355–62. doi:10.3201/eid1109.04144.

[29] Korber B, Fischer WM, Gnanalan S, Yoon H, Theiler J, Abfalterer W, et al. Tracking changes in SARS-CoV-2 spike: evidence that D614G increases infectivity of the COVID-19 virus. Cell 2020;182:812–27. doi:10.1016/j.cell.2020.06.043.

[30] Lee D, Goh KI, Kahng B, Kim D. Complete trails of coauthorship network evolution. Phys Rev E 2010;82:026112. doi:10.1103/PhysRevE.82.026112.

[31] Biggerstaff M, Cowling BJ, Cucunubá ZM, Dinh L, Ferguson NM, Gao H, et al. Early insights from statistical and mathematical modeling of key epidemiologic parameters of COVID-19. Emerg Infect Dis 2020;26:E1–E14. doi:10.3201/ eid2611.200174.

[32] Ma S, Zhang J, Zeng M, Yun Q, Guo W, Zheng Y, et al. Epidemiological parameters of coronavirus disease 2019: a pooled analysis of publicly reported individual data of 1155 cases from seven countries. medRxiv 2020. doi:10.1101/2020.03.21.20040329.

[33] Liu Z, Magal P, Seydi O, Webb G. A COVID-19 epidemic model with latency period. Infect Dis Model 2020;5:323–37. doi:10.1016/j.idm.2020.03.003.

[34] Zhang J, Litvinova M, Wang W, Wang Y, Deng X, Chen X, et al. Evolving epidemiology and transmission dynamics of coronavirus disease 2019 outside Hubei province, China: a descriptive and modelling study. Lancet Infecct Dis 2020;20:793–802. doi:10.1016/S1473-3099(20)30320-9.

[35] Zhang J, Litvinova M, Liang Y, Wang Y, Wang W, Zhao S, et al. Changes in contact patterns shape the dynamics of the COVID-19 outbreak in China. Science 2020;368:1481–6. doi:10.1126/science.abb8001.

[36] Nishiura H, Kobayashi T, Miyama T, Suzuki A, Jung SM, Hayashi K, et al. Estimation of the asymptomatic ratio of novel coronavirus infections (COVID-19). Int J Infect Dis 2020;94:154–5. doi:10.1016/j.ijid.2020.03.029.

[37] Mizumoto K, Kagaya K, Zarebski A, Chowell G. Estimating the asymptomatic proportion of coronavirus disease 2019 (COVID-19) cases on board the diamond princess cruise ship, Yokohama, Japan, 2020. Eurosurveillance 2020;25:1–5. doi:10.2807/1560-7917.es.2020.25.20.2000180.

[38] Workman J. The proportion of COVID-19 cases that are asymptomatic in South Korea: comment on Nishiura et al. Int J Infect Dis 2020;96:398. doi:10.1016/j.ijid.2020.02.095.

[39] Zhuang Z, Zhao S, Lin Q, Cao P, Lou Y, Yang L, Yang S, He D, Xiao L. Preliminary estimates of the reproduction number of the coronavirus disease (COVID-19) outbreak in republic of Korea and Italy by 5 march 2020. Int J Infect Dis 2020;95:308–10. doi:10.1016/j.ijid.2020.04.044.

[40] Barrat A, Barthelemy M, Vespignani A. Dynamical processes on complex networks. Cambridge University Press; 2008.

[41] Watts DJ, Muhamad R, Medina DC, Dodds PS. Multiscale, emergent epidemics in a hierarchical metapopulation model. Proc Natl Acad Sci USA 2005;102:11157–62. doi:10.1073/pnas.0501226102.

[42] Colizza V, Barrat A, Barthelemy M, Valleron AJ, Vespignani A. Modeling the worldwide spread of pandemic influenza: baseline case and containment interventions. PLoS Med 2007;4:0095–110. doi:10.1371/journal.pmed.0040013.

[43] Liu WM, Levin SA, Iwasa Y. Influence of nonlinear incidence rates upon the behavior of sirs epidemiological models. J Math Biol 1986;23:187–204. doi:10.1007/BF00276956.

[44] Kuznetsov YA, Piccardi C. Bifurcation analysis of periodic SEIR and SIR epidemic models. J Math Biol 1994;32:109–21. doi:10.1007/BF00163027.

[45] Li MY, Graef JR, Wang L, Karsai J. Global dynamics of a SEIR model with varying total population size. Math Biosci 1999;160:191–213. doi:10.1016/S0025-5564(99)00030-9.

[46] Wang H, Wang Z, Dong Y, Chang R, Xu C, Yu X, et al. Phase-adjusted estimation of the number of coronavirus disease 2019 cases in Wuhan, China. Cell Discov 2020;6:4–11. doi:10.1038/s41421-020-0148-0.

[47] Du Z, Wang L, Cauchemez S, Xu X, Wang X, Cowling BJ, Meyers LA. Risk for transportation of coronavirus disease from Wuhan to other cities in China. Emerg Infect Dis 2020;26:1049–52. doi:10.3201/eid2605.200146.

[48] Cori A, Ferguson NM, Fraser C, Cauchemez S. A new framework and software to estimate time-varying reproduction numbers during epidemics. Am J Epidemiol 2013;178:1505–12. doi:10.1093/aje/kwt133.

[49] Huang J, Wang J, Xia C. Role of vaccine efficacy in the vaccination behavior under myopic update rule on complex networks. Chaos Solitons Fractals 2020;130:109425. doi:10.1016/j.chaos.2019.109425.

[50] Fennell PG, Melnik S, Gleeson JP. Limitations of discrete-time approaches to continuous-time contagion dynamics. Phys Rev E 2016;94:052125. doi:10.1103/PhysRevE.94.052125.

[51] Ferreira SC, Castellano C, Pastor-Satorras R. Epidemic thresholds of the susceptible-infected-susceptible model on networks: a comparison of numerical and theoretical results. Phys Rev E 2012;86:041125. doi:10.1103/PhysRevE.86.041125.