Hindawi
Complexity
Volume 2023, Article ID 4591403, 10 pages
https://doi.org/10.1155/2023/4591403

Research Article

Epidemic Dynamics in Temporal Clustered Networks with Local-World Structure

Wenjun Jing,[1] **Juping Zhang**,[2] **and Xiaoqin Zhang**[1]

[1]*School of Statistics, Shanxi University of Finance and Economics, Taiyuan, Shanxi 030006, China*
[2]*Complex Systems Research Center, Shanxi University, Taiyuan, Shanxi 030006, China*

Correspondence should be addressed to Wenjun Jing; jingwj@sxufe.edu.cn

Received 3 November 2022; Revised 17 December 2022; Accepted 11 January 2023; Published 30 January 2023

Academic Editor: Siew Ann Cheong

Population demography can change the network structure, which further plays an important role in the spreading of infectious disease. In this paper, we study the epidemic dynamics in temporal clustered networks where the local-world structure and clustering are incorporated into the attachment mechanism of new nodes. It is found that increasing the local-world size of new nodes has little influence on the clustering coefficient but increases the degree heterogeneity of networks. Besides, when the network evolves faster, increasing the local-world size of new nodes leads to a faster initial growth rate and a larger steady density of infectious nodes, while it has small impacts on the steady density of infectious disease when the network evolves slowly. Furthermore, if the average degree is fixed, increasing the probability of triad formation p enlarges the clustering coefficient of a network, which reduces the initial growth rate and steady density of infectious nodes in the network. This work could provide a theoretical foundation for the control of infectious disease.

1. Introduction

Infectious diseases, such as H1N1 influenza and the ongoing coronavirus disease 2019 (COVID-19), pose a significant health and economic burden on society. Mathematical models have been used as a powerful tool to mitigate the impacts of infectious diseases as they can provide valuable insights into the transmission mechanism of diseases and then guide policymakers implementing effective prevention and control measures. More recently, researchers have devoted a great deal of effort to mathematically model the spread of COVID-19 and seek for effective non-pharmaceutical interventions, such as social distancing, isolation, and intercity travel restrictions, to control the local outbreak of COVID-19 [1–5]. Besides, Li et al. used the signed networks to study the influence of structural balance on epidemic transmission [6]. Sun et al. investigated the effects of resource diffusion on epidemic propagation in multilayer networks with simplicial complexes, showing that increasing the resource diffusion on 2-simplexes can suppress the epidemic spreading [7]. It is also discovered that information transmission induced by the 2-simplex can suppress epidemic outbreak to a certain extent [8]. Also, a review of different phenomena on higher order networks was given by Majhi et al. [9]. All these studies have demonstrated that the population contact pattern which can be described as a network plays an important role in the epidemic spreading.

The population demography can change the network structure which further affects the disease spreading process on networks. Kamp introduced an SID model with HIV as a case study to investigate the interplay between epidemic spreading and the dynamics of network structure [10]. Jin et al. established an SIS heterogeneous mean-field model with a constant recruitment rate and derived the global dynamics of the model where the new members link to existing nodes randomly [11]. Piccardi et al. integrated the birth and death process into the SIRS model on heterogeneous networks and disentangled the underlying causes of the different outcomes obtained for SIR and SIRS processes by applying an age-degree model [12]. Based on conditional Markov chain and pairwise approximation, Luo et al.

studied an SIS pairwise model on networks, finding that demographics of individuals can induce the extinction of an epidemic [13]. Jing et al. studied the effects of different moment closure methods [14], adaptive behaviors of new members [15], and the distribution of infection age [16] on epidemic spreading in networks with demographics. Leung et al. provided a new framework using digital proxies of population mobility and mixing to monitor viral transmissibility and effectiveness of social distancing interventions in the ongoing COVID-19 pandemic [17].

Currently, most research in network epidemic models with demographics has assumed that a new node links to existing nodes according to random or preferential attachment mechanism when it enters the network. In fact, there has been evidence to indicate that the preferential attachment mechanism works on a local world of each node [18, 19]. Using a local preferential attachment mechanism, Li and Chen constructed a local-world evolving network whose distribution is a transition between that of an exponential network and of a power-law scaling network [18]. Zhang et al. proposed a local-world evolving network model with changeable local-world size and tunable clustering by including a triad formation step in the local-world evolving network model [20]. Wen and Duan introduced extended links to mimic the weak relations between nodes in different local worlds, obtaining a local-world evolving model which exhibits scale-free behavior as well as the small-world property [21]. Wang et al. presented a local preferential attachment model where a local area consists of a node and all its neighbors [22]. They derived the stable degree distributions and clustering-degree correlations of the network. Besides, the epidemic spreading behavior [23], rumor spreading process [24], and cascading failures [25] in local-world evolving networks are studied. It shows that the attachment mechanism of new members has a significant impact on the network structure and the propagation process on networks.

Many real-world networks exhibit nonnegligible clustering which can have an important impact on epidemic dynamics. However, there is no clear consensus on the role of clustering. For example, it shows that the effect of clustering on epidemic spreading in Watts–Strogatz networks is stronger than the effect of clustering on epidemic spreading in Facebook network [26]. Considering the local-world structure and clustering characteristics, we will investigate the effects of attachment mechanism of new members on the network structure and epidemic dynamics in this paper.

The rest of this paper is organized as follows. In Section 2, we introduce some basic characteristics of networks. Then, the evolving network model and epidemic spreading process on networks are given in Section 3. In Section 4, numerical simulations are conducted to explore the impacts of mechanisms that new nodes connect existing nodes on the network structure and epidemic dynamics. Finally, some conclusions and discussions about this work are given in Section 5.

2. Basic Characteristics of Networks

Let $G(V, E)$ be an unweighted, undirected network with V representing the set of nodes and $E \in V \times V$ representing the set of links between nodes in the network. For a finite network with N nodes, the set E can be encoded in the adjacency matrix whose entry g_{ij} is 1 if there is a link between i and j, and 0 otherwise. A finite network is determined uniquely by its adjacency matrix.

Degree distribution is the most important characteristic of the network. The degree of one node is the number of links connecting it. Then, the degree distribution is given by $p_k = N_k/N$, where N_k is the number of nodes with degree k. Some widely used networks include regular random, Poisson, bimodal, and scale-free networks.

Clustering, one frequently used statistical property of characterizing networks, measures the probability that two neighbors of a randomly chosen node share a link to form a triangle [27]. Specifically, the clustering coefficient of node i is defined as the ratio of the number of triangles and triples:

$$c_i = \frac{2T_i}{k_i(k_i - 1)}, \tag{1}$$

where T_i and $k_i(k_i - 1)/2$ are the total number of existing links and the number of all possible links between the neighbors of node i, respectively. Furthermore, the degree-dependent clustering coefficient of node with degree k can be defined as the average clustering coefficient of nodes with degree k, i.e.,

$$c(k) = \frac{1}{N_k} \sum_{i \in \mathscr{Y}(k)} c_i, \tag{2}$$

where $\mathscr{Y}(k)$ is the set of nodes with degree k. Then, the clustering coefficient of the network is defined as the weighted average of degree-dependent clustering coefficient, which is also the average of clustering coefficient of nodes:

$$\overline{c} = \frac{1}{N} \sum_{i=1}^{N} c_i = \sum_{k=1}^{k_{\max}} p_k c(k), \tag{3}$$

where $k_{\max}$ is the maximum degree of nodes.

3. The Model

3.1. The Evolving Network Model. The local-world evolving network was first proposed by Li and Chen [18]. It captures the localization of real-life networks. Then, Zhang et al. presented expanded local-world evolving network with tunable clustering by including a triad formation (TF) step [20]. Considering the local-world structure and clustering of networks, we propose the generating algorithm of the dynamic clustered network with demographics. It is summarized as follows:

(i) Initial condition: the initial network consists of N nodes and has a Poisson degree distribution with average degree $\langle k \rangle$.

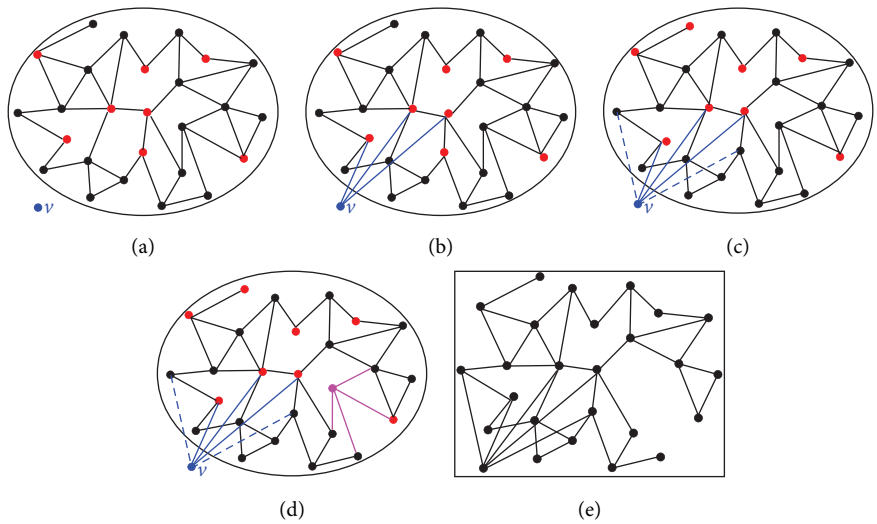

Figure 1: Sketch of the network evolving process with the local-world size $M = 8$ and the value $m = 3$. In (a), M nodes (the red nodes) are randomly selected as the local world of the new node v (the blue node). In (b), m nodes in the local world are chosen to be connected with v by blue lines according to the preferential attachment. In (c), for the m nodes in (b), a randomly chosen neighbor is linked to v (by blue dashed line) with probability p. In (d), one randomly selected node as well as its links (denoted by pink color) is deleted from the network. Then, the network after one step, i.e., adding one node and then deleting one node, is shown in (e).

(ii) Network evolving: At each time step t, we fist add a new node v to the network and then delete a randomly selected node as well as its links from the network. The attachment mechanism between the new node v and existing nodes is as follows:

(1) Local-world establishment: randomly select M nodes from the network, referred to as the "local world" of v.

(2) Local preferential attachment (LPA): The new node v connects to m different nodes in its local world according to the preferential attachment. Thus, the probability $\Pi_{\text{Local}}(k_i)$ that node v is connected to an existing node i in the local world of node v depends on the degree k_i of node i:

$$\Pi_{\text{Local}}(k_i) = \frac{M}{N}\frac{k_i}{\sum_{l\in\text{Local}} k_l}. \quad (4)$$

(3) Triad formation (TF): When the new node v connects an existing node i, we then implement a TF step with probability p. Specifically, if a link between v and i was added in the previous LPA step, then one more link between v and a randomly chosen neighbor of i is added. If all neighbors of i have already been connected to v, do one more LPA step instead.

A sketch of the network evolving process is shown in Figure 1. After T steps (the designed total iteration steps which are large enough to reach a stationary network), the algorithm results in a connected network with N nodes and average degree is $\langle k\rangle = m(1+p)$. Obviously, the network size keeps a constant and it has $M \leqslant N$ and $0 \leqslant p \leqslant 1$. There are two limiting cases for the evolving network: $M = m$, $p = 0$ and $M = N$, $p = 0$. When $M = m$, $p = 0$, the stationary degree distribution is Poisson with mean m, while when $M = N$, $p = 0$, the stationary degree distribution is "stretched exponential," namely, $p_k \sim k^{-(3/4)}e^{-(2/\sqrt{k})}$ [28]. Besides, the parameter p allows us to include the clustering effect into network by building triads.

3.2. Epidemic Spreading on Evolving Networks. We consider an SIS epidemic spreading on evolving networks, where the initial network is the stationary network obtained by the generating algorithm in Section 3.1. During the epidemic, nodes can either be susceptible (S) or infectious (I). The susceptible nodes can be infected by their infectious neighbors with a per-contact transmission rate λ if they are connected to one or more infected nodes, while the infectious one can be cured at a recovery rate γ. When an infectious node recovers, it turns to susceptible and can be infected again. Meanwhile, existing nodes together with all their links can leave the network due to death, and new nodes enter the network due to birth. Here, the mechanism that nodes enter or leave the network is identical to that in Section 3.1. Thus, the population size is a constant. Given a short time interval δ_t, the algorithm of epidemic spreading on evolving networks can be summarized as follows:

(i) Initialization: An undirected and unweighted network with N nodes is created and set to the initial network by performing the network generating algorithm in Section 3.1. Then, $I(0)$ randomly selected nodes in the network are initially infected and the other nodes are susceptible.

(ii) Epidemic process: Given the state of each node at time t, the states of nodes at time $t + \delta t$ are updated as follows. Each infected node recovers and becomes susceptible again with probability $\gamma\delta_t$. Each

susceptible node, such as node i, turns into infectious with probability λn_i, where n_i is the number of infectious neighbors of i.

(iii) Network evolving: At time t, D_t (extracted from the binomial distribution $B(N, \mu\delta_t)$) randomly selected nodes as well as all their links are eliminated from the network. Then, D_t nodes are added into the network sequentially where links are created according to the mechanism in Section 3.1.

To sum up, we can simulate the epidemic spreading on dynamic clustered networks by iterating the epidemic process and networking evolving process until there is no infectious node in the network or the number of infectious nodes reaches a steady state. During the iteration, record the number of infectious nodes in each time step.

3.3. The Heterogeneous Pairwise Model on Evolving Networks. In line with the above algorithms, we construct the heterogeneous pairwise model on evolving networks in this section. The notations of the model follow those established in [29], i.e., using square brackets [ ] to denote the expected numbers of nodes, pairs, or triples of a particular type. Specifically, let $[A_k]$ be the number of nodes in state A and with degree k, and $[A]$ be the number of nodes in state A. $[A_kB_l]$ represents the number of pairs with one node in state A having degree k and with the other node in state B having degree l. $[A_kB_lC_m]$ stands for the number of triples of type $A_k - B_l - C_m$. If a disease state of a variable has no subscript, it implicitly contains the sum over all possible degrees, such as $[A] = \sum_k [A_k]$ and $[A_kB] = \sum_l [A_kB_l]$. Let Π_{A_k} denote the probability that a link of new nodes is connected to an existing node in state A with degree k. It depends on the values of M and p. Furthermore, according to the attachment mechanism, the degree distribution of new nodes is

$$\pi_k = \begin{cases} \binom{m}{k-m} p^{k-m}(1-p)^{2m-k}, & m \leqslant k \leqslant 2m, \\ 0, & \text{others.} \end{cases} \tag{5}$$

Using the notations above, the SIS pairwise model on evolving networks can be written as

$$\begin{aligned} \frac{\mathrm{d}[S_k]}{\mathrm{d}t} &= \gamma[I_k] - \lambda \sum_m [S_k I_m] + \mu N \pi_k - \mu[S_k] - \mu\left(k[S_k] - (k+1)[S_{k+1}]\right) \\ &\quad + \mu m N (1+p)\left(\Pi_{S_{k-1}} - \Pi_{S_k}\right), \end{aligned} \tag{6a}$$

$$\begin{aligned} \frac{d[I_k]}{dt} &= \lambda \sum_m [S_k I_m] - (\mu+\gamma)[I_k] - \mu\left(k[I_k] - (k+1)[I_{k+1}]\right) \\ &\quad + \mu m N (1+p)\left(\Pi_{I_{k-1}} - \Pi_{I_k}\right), \end{aligned} \tag{6b}$$

$$\begin{aligned} \frac{\mathrm{d}[S_kS_l]}{\mathrm{d}t} &= \gamma\left([S_kI_l] + [I_kS_l]\right) - \lambda \sum_m \left([S_kS_lI_m] + [I_mS_kS_l]\right) + \mu N \pi_k k \Pi_{S_{l-1}} + \mu N \pi_l l \Pi_{S_{k-1}} \\ &\quad + \mu m N (1+p)\left(\Pi_{S_{k-1}} \frac{[S_{k-1}S_l]}{[S_{k-1}]} - \Pi_{S_k} \frac{[S_kS_l]}{[S_k]} + \Pi_{S_{l-1}} \frac{[S_kS_{l-1}]}{[S_{l-1}]} - \Pi_{S_l} \frac{[S_kS_l]}{[S_l]}\right) \\ &\quad + \mu\left(k[S_{k+1}S_l] + l[S_kS_{l+1}] - (k+l)[S_kS_l]\right), \end{aligned} \tag{6c}$$

$$\begin{aligned} \frac{\mathrm{d}[S_kI_l]}{\mathrm{d}t} &= \lambda \sum_m \left([S_kS_lI_m] - [I_mS_kI_l]\right) - \lambda[S_kI_l] - \gamma[S_kI_l] + \gamma[I_kI_l] + \mu N \pi_k k \Pi_{I_{l-1}} \\ &\quad + \mu m N (1+p)\left(\Pi_{S_{k-1}} \frac{[S_{k-1}I_l]}{[S_{k-1}]} - \Pi_{S_k} \frac{[S_kI_l]}{[S_k]} + \Pi_{I_{l-1}} \frac{[S_kI_{l-1}]}{[I_{l-1}]} - \Pi_{I_l} \frac{[S_kI_l]}{[I_l]}\right) \\ &\quad + \mu\left(k[S_{k+1}I_l] + l[S_kI_{l+1}] - (k+l)[S_kI_l]\right), \end{aligned} \tag{6d}$$

$$\begin{aligned} \frac{d[I_kI_l]}{dt} &= \lambda \sum_m \left([I_kS_lI_m] + [I_mS_kI_l]\right) + \lambda\left([S_kI_l] + [I_kS_l]\right) - 2\gamma[I_kI_l] \\ &\quad + \mu m N (1+p)\left(\Pi_{I_{k-1}} \frac{[I_{k-1}I_l]}{[I_{k-1}]} - \Pi_{I_k} \frac{[I_kI_l]}{[I_k]} + \Pi_{I_{l-1}} \frac{[I_kI_{l-1}]}{[I_{l-1}]} - \Pi_{I_l} \frac{[I_kI_l]}{[I_l]}\right) \\ &\quad + \mu\left(k[I_{k+1}I_l] + l[I_kI_{l+1}] - (k+l)[I_kI_l]\right). \end{aligned} \tag{6e}$$

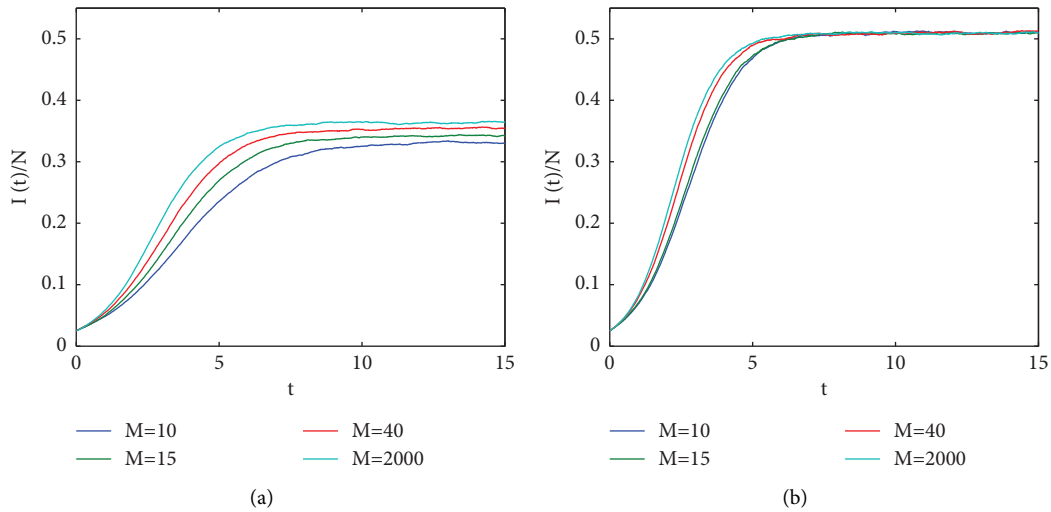

Figure 2: Time dependence of the densities of infectious nodes for different values of M. The lines are averaged over 200 stochastic simulations according to the algorithm of epidemic spreading on evolving networks in Section 3.2, with the parameter values being $N = 2000$, $\lambda = 0.3$, $\gamma = 1$, $m = 8$, $p = 0$, (a) $\mu = 0.5$, and (b) $\mu = 0.1$.

In order to close the above equations, we need to give the expression of Π_{A_k} and an approximation of triples in terms of nodes and pairs. The expression of Π_{A_k} depends on the values of M and p, which are classified as the following three cases:

(1) $M = m$, $p = 0$: In this special case, newcomers connect to all nodes in their local world. It means that the LPA is reduced to uniform attachment, that is, new nodes connect to existing nodes randomly. Therefore, it has

$$\Pi_{S_k} = \frac{k[S_k]}{\sum_h (h[S_h] + h[I_h])},$$
$$\Pi_{I_k} = \frac{k[I_k]}{\sum_h (h[S_h] + h[I_h])}. \quad (7)$$

Moreover, the network clustering coefficient approached 0 such that the triples can be approximated by the triple closure in [30]:

$$[A_k B_l C_m] \approx \frac{l-1}{l} \frac{[A_k B_l][B_l C_m]}{[B_l]}. \quad (8)$$

Substituting equations (7) and (8) into equation (6) yields a closed system.

(2) $M = N$, $p = 0$: In this limiting case, the network clustering coefficient approached 0 and new nodes connect existing nodes exactly the same as global preferential attachment mechanism. Therefore, it has

$$\Pi_{S_k} = \frac{k[S_k]}{\sum_h (h[S_h] + h[I_h])},$$
$$\Pi_{I_k} = \frac{k[I_k]}{\sum_h (h[S_h] + h[I_h])}. \quad (9)$$

Substituting equations (8) and (9) into equation (6) gives a closed system.

(3) $m < M < N$: In this general case, the explicit expressions of Π_{S_k} and Π_{I_k} are much more complicated and unable to be derived since they depend on the values of M and p. Thus, we do not give the exact formula of the model here. There are two remarks about the model.

Remark 1. It is assumed in [18] that $\sum_{l \in \text{Local}} k_l = \langle k \rangle M$, finding that the local-world growing network also has a power-law degree distribution when $m \ll M$. Similarly, the formulas of Π_{S_k} and Π_{I_k} are exactly the same as in equation (9) under this assumption. This reveals the main reason that as M increases, the epidemic curves are closer to those when $M = N$ as shown in Figure 2.

Remark 2. When $p \neq 0$, there is small but non-negligible clustering in the network. Let φ be the network clustering coefficient; then, the triples can be approximated by the triple closure with clustering in [31]:

$$[A_k B_l C_m] \approx \frac{l-1}{l} \frac{[A_k B_l][B_l C_m]}{[B_l]} \left(1 - \phi \frac{nN}{km} \frac{[A_k C_m]}{[A_k B_m]}\right). \quad (10)$$

4. Numerical Simulations

4.1. Degree Distribution and Clustering Coefficient of Networks. We will investigate the degree distribution and clustering coefficient of networks by stochastic simulations. In these simulations, the initial network size is $N = 2000$ and each pair of nodes has a link independently of any others with probability $(m(p+1)/N - 1)$. Thus, the initial network has a Poisson degree distribution with mean $m(p+1)$. The

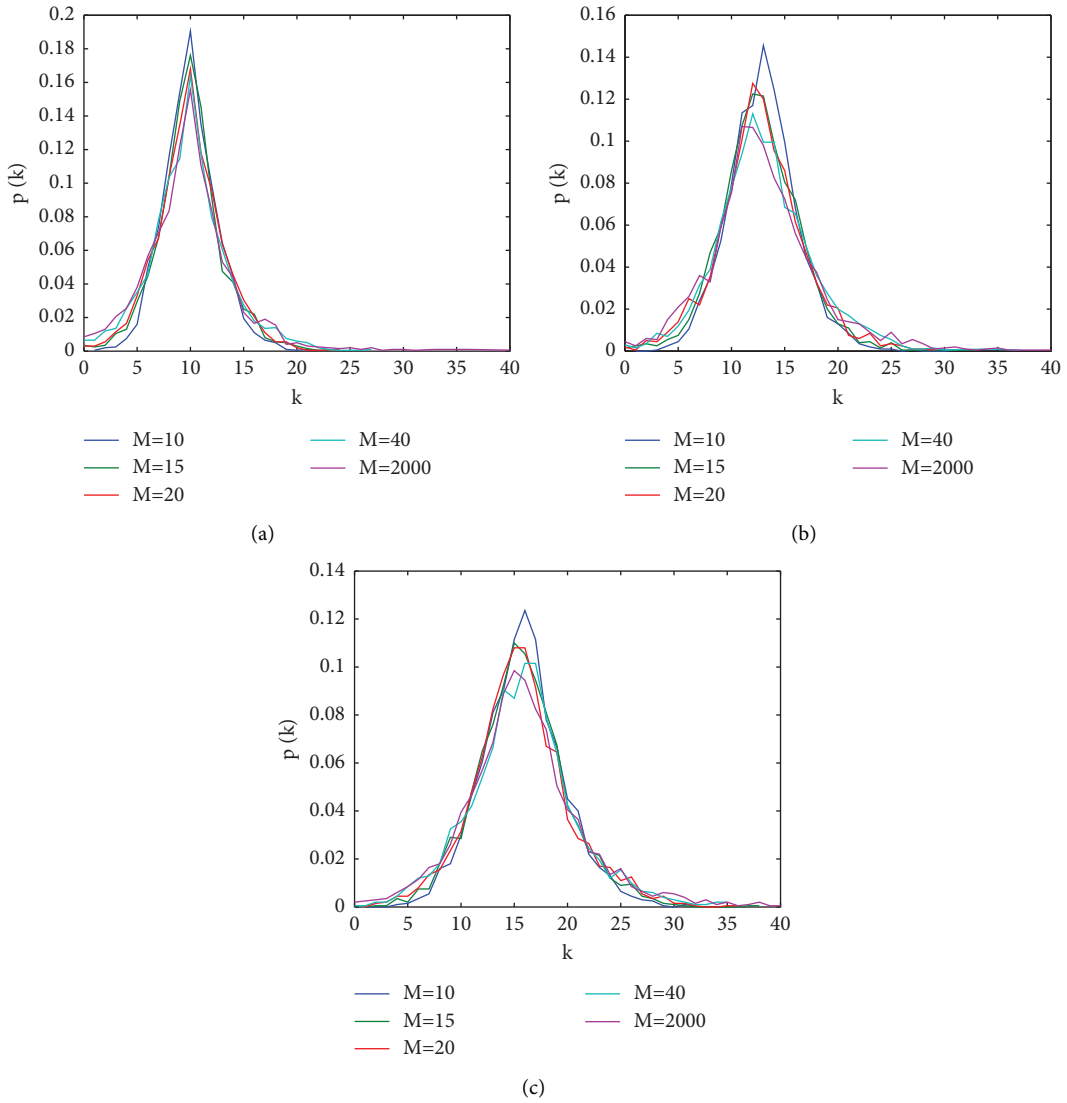

FIGURE 3: Stationary degree distribution of networks for different values of M. Results are obtained by stochastic simulations according to the network generating algorithm in Section 3.1, where the initial network has a Poisson degree distribution with mean $m(p+1)$. The parameters are $m = 10$, $T = 20000$, (a) $p = 0$, (b) $p = 0.3$, and (c) $p = 0.6$.

simulation lasts 20000 time steps according to the network generating algorithm described in Section 3.1.

Figure 3 gives the stationary degree distribution of networks under different values of M and p. It demonstrates that the local-world size M and the probability of triad formation p have a direct impact on the network structure. It can be seen from Figure 3(a) that when $p = 0$, the larger the value of M, the flatter and wider the degree distribution. This means that increasing the local-world size reinforces the degree heterogeneity for networks. However, as both the values of p and M increase, this impact disappears as shown in Figure 3(c). If the values of p and M are large enough, new nodes link to existing nodes with approximately a global preferential attachment mechanism, that is, the local preferential attachment selection is not effective. Thus, there is relatively little change in the degree distribution of networks.

Figure 4 shows the evolution of clustering coefficients for different values of M and p. It suggests that the local-world size M has little effect on the network clustering for a fixed value of p. However, for fixed M, the network clustering coefficient is approximately 0 if $p = 0$, whereas it converges rapidly to a stable value if $p > 0$. Furthermore, compared with the growing network without deletion of nodes [20], it can be found that node removal dramatically decreases the degree distribution and clustering in networks. But the network still has nonnegligible clustering where the clustering coefficient is increasing with p in our model. Thus, the

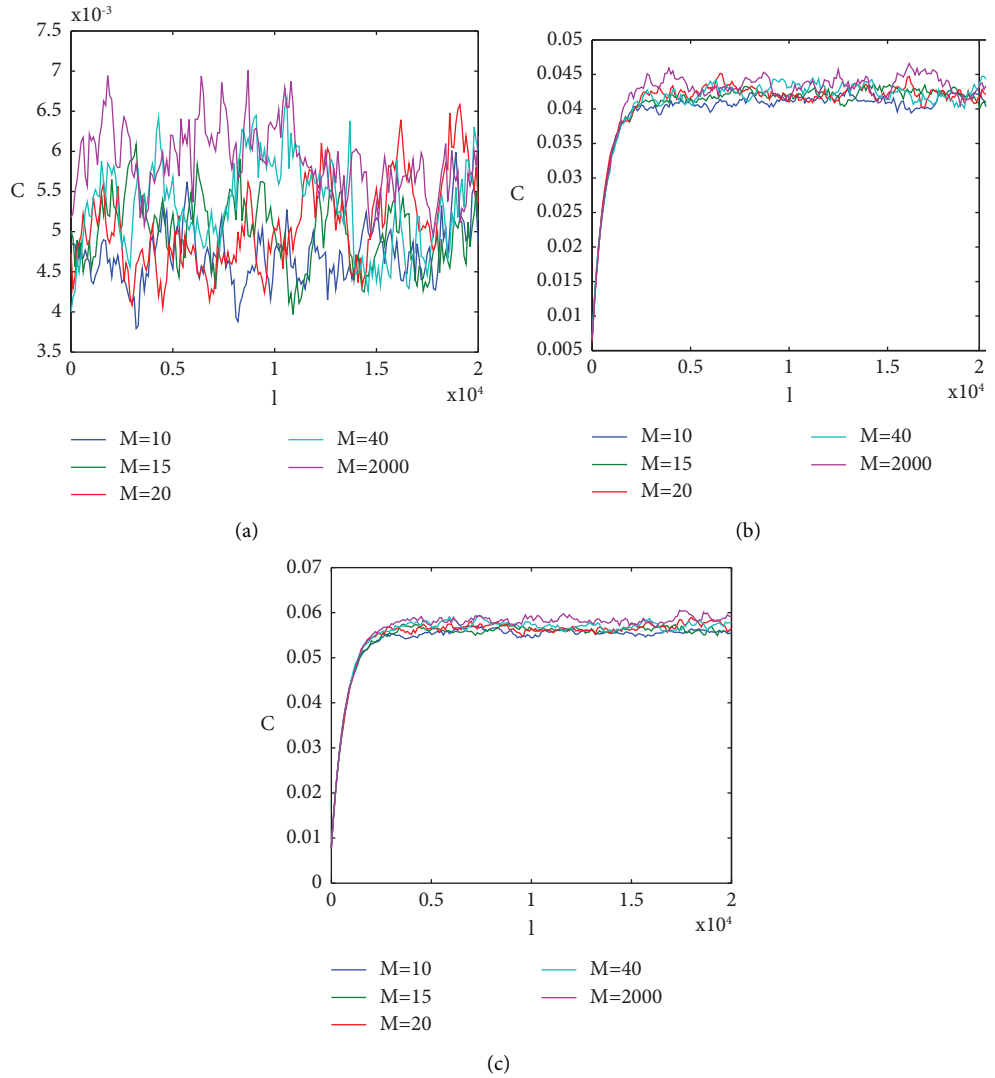

Figure 4: Clustering coefficients of the network during the evolution of the network for different values of M. The network evolves as the generating algorithm in Section 3.1, where the initial network has a Poisson degree distribution with mean $m(p+1)$. The parameters are $m = 10$, (a) $p = 0$, (b) $p = 0.3$, and (c) $p = 0.6$.

clustering coefficient can be tuned by changing the probability of triad formation.

4.2. Epidemic Dynamics. Figure 5 provides a comparison of stochastic simulations to numerical simulations of pairwise model for two limiting cases $M = m$ and $M = N$, which correspond to uniform attachment and global preferential attachment, respectively. One can see that the agreement is very good in both cases. Besides, when new nodes connect existing nodes according to global preferential attachment, the epidemic has a faster initial growth rate and a larger steady density of infectious nodes compared to the case of uniform attachment. This phenomenon is more significant in Figure 5(a) where the average degree of the network is smaller. It indicates that the attachment of new nodes can significantly affect disease dynamics, and decreasing the degree heterogeneity of networks is helpful in mitigating epidemics.

Figure 2 investigates the effects of local-world size M on epidemic dynamics. In Figure 2(a) where μ is large, higher values of M correspond to faster initial growth rate and larger steady density of infectious nodes. However, when μ is small as shown in Figure 2(b), the value of M has only a relatively small effect on the initial growth rate and does not affect the steady density of infectious nodes. Besides, it can be seen that a larger value of μ leads to a higher steady density of infectious nodes, and the epidemic reaches the steady state quicker by comparing Figures 2(a) and 2(b). This is due to the fact that the network evolves faster such that M has a more significant effect on the network distribution as μ increases. Specifically, infectious nodes are removed faster from the network and susceptible nodes enter the network instead. Meanwhile, more infectious nodes leave the network before they infect others, reducing the average infection period. All these factors contribute to the decline in epidemic spreading. In contrast, when μ is small, the

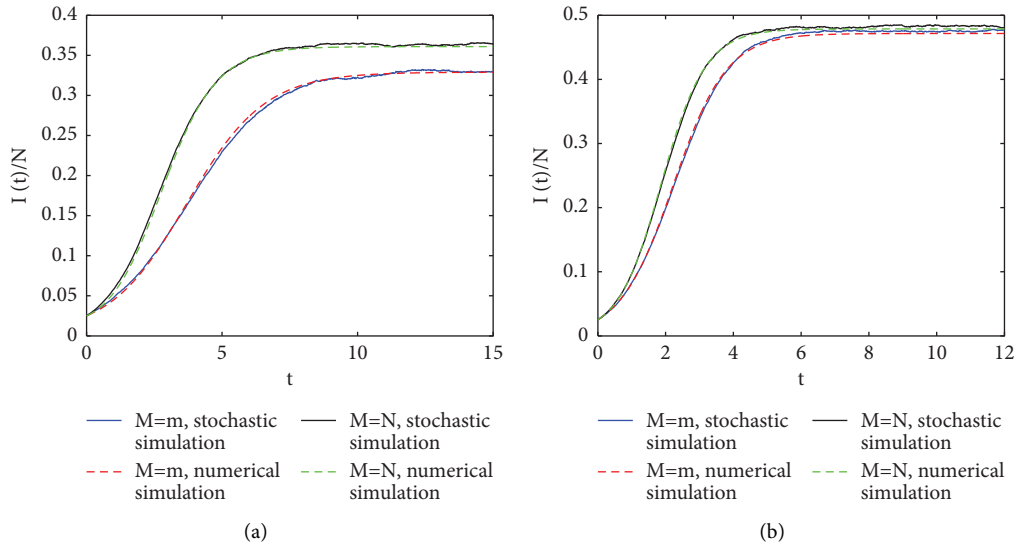

Figure 5: A comparison of stochastic simulations and model predictions of infection dynamics for two limiting cases $M = m$ and $M = N$. The parameter values are $\lambda = 0.3$; $\gamma = 1$; $N = 2000$; (a) $m = 8$, $p = 0$; and (b) $m = 10$, $p = 0$. Red and green lines correspond to numerical solutions of pairwise model ((6a)–(6e)) with the linking mechanism being (7) and (8), respectively. Dashed lines present the average of 250 stochastic simulations according to the algorithm of epidemic spreading on evolving networks in Section 3.2.

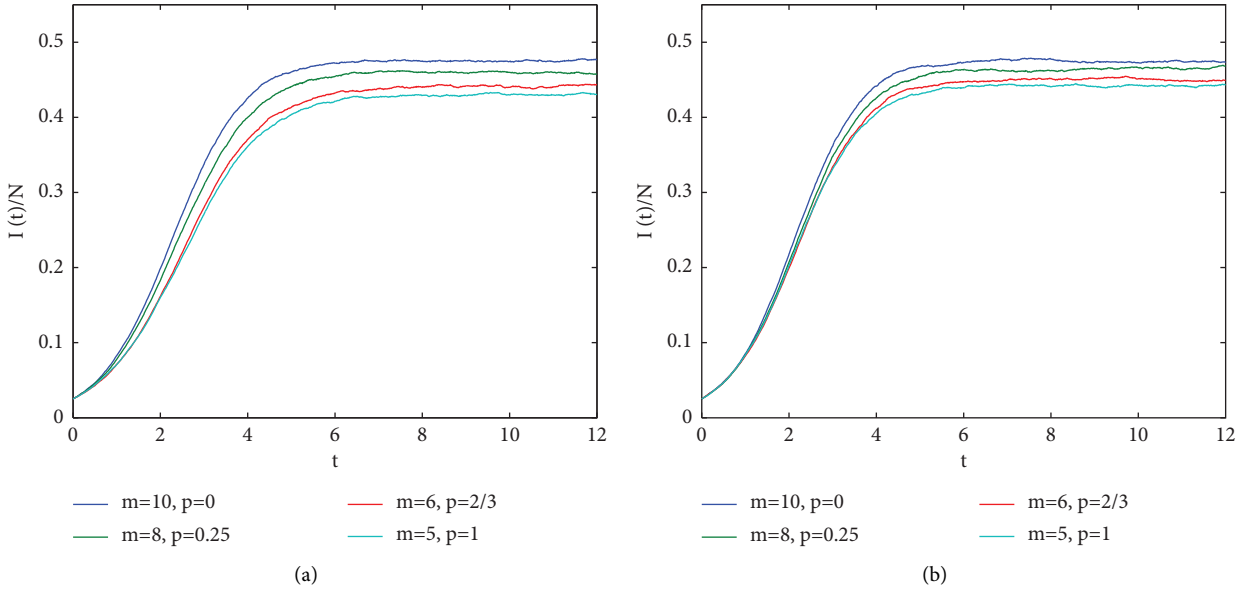

Figure 6: Continued.

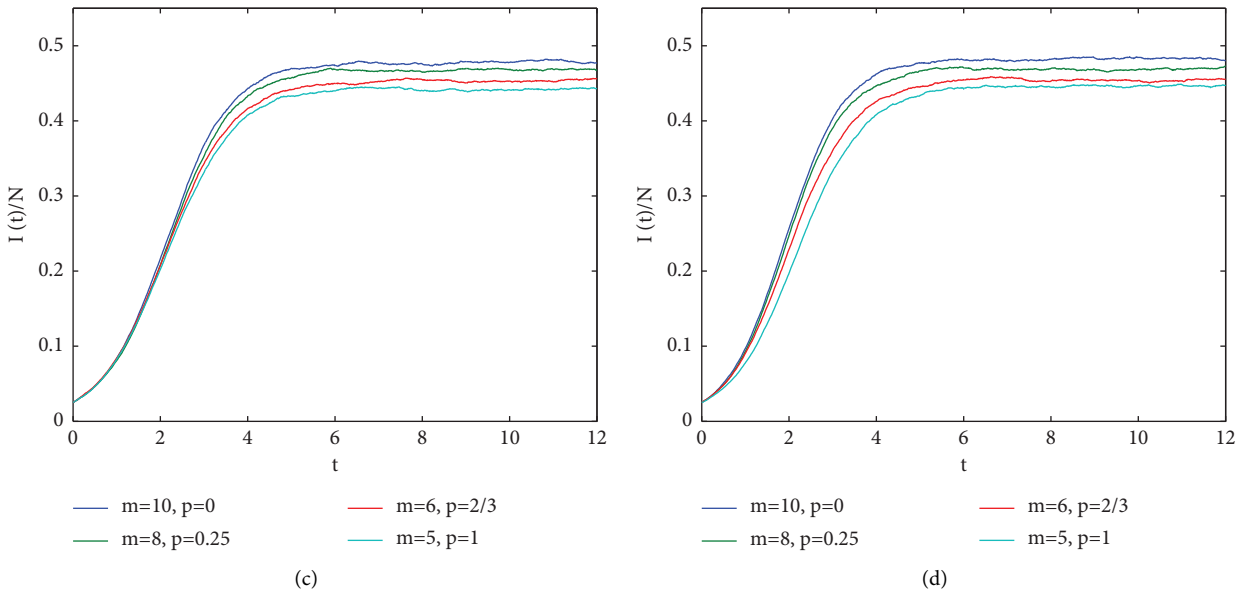

FIGURE 6: Time dependence of the densities of infectious nodes based on the average of 200 stochastic simulations for different values of m and p. The lines are averaged over 200 stochastic simulations according to the algorithm of epidemic spreading on evolving networks in Section 3.2, and the parameter values are $\lambda = 0.3$, $\mu = 0.5$, $\gamma = 1$, $N = 2000$, (a) $M = m$, (b) $M = 20$, (c) $M = 40$, and (d) $M = N$.

network evolves slowly and the network structure is relatively stable, such that the local-world size has little influence on the steady density of infectious diseases.

Figure 6 gives the simulation results based on the average of 200 stochastic simulations for different values of M and p. For comparison, M and p are determined such that the networks have the same average degree, i.e., $m(1 + p) = 10$. It shows that the initial growth rate and steady density of infectious nodes decrease with increasing p values for fixed M and average degree. On the other hand, combining with the result noted in Figure 4 that larger values of p lead to a larger clustering coefficient of the network, it implies that clustering in networks is capable of suppressing infection of an epidemic. This is consistent with the previous work in static clustered networks [29, 32].

5. Conclusion and Discussion

The present study has explored the interplay between epidemic spreading and network evolution. In our model, new nodes fist connect m existing nodes according the local preferential attachment mechanism and then connect to one randomly chosen neighbor of these nodes with a given probability p. Such network evolution mechanism incorporates both the local-world structure and clustering into networks. Simulation results illustrate that the attachment mechanism of new nodes can influence the degree distribution, clustering, and epidemic dynamics in networks. It shows that increasing the local-world size leads to a stronger heterogeneity in the network degree distribution but has little impact on the clustering in networks. Furthermore, it corresponds to a faster initial growth rate as well as a larger steady density of infectious nodes when the network evolves faster. In contrast, the local-world size has a small impact on the steady density of infectious nodes if the network evolves slowly. On the other hand, for a given average degree and local-world size, increasing the probability of triad formation leads to a larger clustering coefficient of the network which further suppresses the infection of an epidemic. In conclusion, controlling the attachment mechanism of new nodes could be an effective measure to mitigate an epidemic.

A limitation of our study is that the probability that an edge emanating from new nodes connects a susceptible or an infectious node in networks cannot be calculated explicitly. Besides, the high dimensionality of the model poses a challenge for theoretical analysis of epidemic dynamics. How to derive an explicit formula for the probability (Π_{S_k} and Π_{I_k}) and investigate the steady states as well as their stability of the model needs more rigorous mathematical techniques. This remains an open question.

Data Availability

The data used to support the findings of this study are available from the corresponding author upon request.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

Acknowledgments

This study was supported by the National Natural Science Foundation of China (nos. 12101373 and 11971278), the Natural Science Foundation of Shanxi Province (no. 20210302124261), and the Scientific and Technological Innovation Programs of Higher Education Institutions in Shanxi (no. 2020L0246).

References

[1] M. te Vrugt, J. Bickmann, and R. Wittkowski, "Effects of social distancing and isolation on epidemic spreading modeled via dynamical density functional theory," *Nature Communications*, vol. 11, no. 1, p. 5576, 2020.

[2] C. Xu, Y. Pei, S. Liu, and J. Lei, "Effectiveness of non-pharmaceutical interventions against local transmission of COVID-19: an individual-based modelling study," *Infectious Disease Modelling*, vol. 6, pp. 848–858, 2021.

[3] M. T. Li, G. Q. Sun, J. Sun et al., "Analysis of COVID-19 transmission in Shanxi Province with discrete time imported cases," *Mathematical Biosciences and Engineering*, vol. 17, no. 4, pp. 3710–3720, 2020.

[4] M. T. Li, J. Cui, J. Zhang, and G. Q. Sun, "Transmission analysis of COVID-19 with discrete time imported cases: tianjin and Chongqing as cases," *Infectious Disease Modelling*, vol. 6, pp. 618–631, 2021.

[5] Y. Wei, J. Wang, W. Song, C. Xiu, L. Ma, and T. Pei, "Spread of COVID-19 in China: analysis from a city-based epidemic and mobility model," *Cities*, vol. 110, Article ID 103010, 2021.

[6] H. J. Li, W. Xu, S. Song, W. X. Wang, and M. Perc, "The dynamics of epidemic spreading on signed networks," *Chaos, Solitons and Fractals*, vol. 151, Article ID 111294, 2021.

[7] Q. Sun, Z. Wang, D. Zhao, C. Xia, and M. Perc, "Diffusion of resources and their impact on epidemic spreading in multilayer networks with simplicial complexes," *Chaos, Solitons and Fractals*, vol. 164, Article ID 112734, 2022.

[8] J. Fan, Q. Yin, C. Xia, and M. Perc, "Epidemics on multilayer simplicial complexes," *Proceedings of the Royal Society A*, vol. 478, Article ID 20220059, 2022.

[9] S. Majhi, M. Perc, and D. Ghosh, "Dynamics on higher-order networks: a review," *Journal of The Royal Society Interface*, vol. 19, Article ID 20220043, 2022.

[10] C. Kamp, "Untangling the interplay between epidemic spread and transmission network dynamic," *PLoS Computational Biology*, vol. 6, Article ID 1000984, 2010.

[11] Z. Jin, G. Q. Sun, and H. Sun, "Epidemic models for complex networks with demographics," *Mathematical Biosciences and Engineering*, vol. 11, no. 6, pp. 1295–1317, 2014.

[12] C. Piccardi, A. Colombo, and R. Casagrandi, "Connectivity interplays with age in shaping contagion over networks with vital dynamics," *Physical Review E: Statistical, Nonlinear, and Soft Matter Physics*, vol. 91, Article ID 22809, 2015.

[13] X. Luo, L. Chang, and Z. Jin, "Demographics induce extinction of disease in an SIS model based on conditional Markov chain," *Journal of Biological Systems*, vol. 25, no. 01, pp. 145–171, 2017.

[14] W. Jing, Z. Jin, and J. Zhang, "Low-dimensional SIR epidemic models with demographics on heterogeneous networks," *Journal of Systems Science and Complexity*, vol. 31, no. 5, pp. 1103–1127, 2018.

[15] W. Jing, Z. Jin, and X. L. Peng, "Adaptive SIS epidemic models on heterogeneous networks with demographics and risk perception," *Journal of Biological Systems*, vol. 26, no. 02, pp. 247–273, 2018.

[16] W. Jing, J. Jin, and J. Zhang, "An SIR pairwise epidemic model with infection age and demography," *Journal of Biological Dynamics*, vol. 12, no. 1, pp. 486–508, 2018.

[17] K. Leung, J. T. Wu, and G. M. Leung, "Real-time tracking and prediction of covid-19 infection using digital proxies of population mobility and mixing," *Nature Communications*, vol. 12, no. 1, p. 1501, 2021.

[18] X. Li and G. Chen, "A local-world evolving network model," *Physica A: Statistical Mechanics and Its Applications*, vol. 328, no. 2, pp. 274–286, 2003.

[19] X. Li, Y. Y. Ying Jin, and G. Chen, "Complexity and synchronization of the world trade web," *Physica A: Statistical Mechanics and Its Applications*, vol. 328, no. 2, pp. 287–296, 2003.

[20] Z. Zhang, L. Rong, B. Wang, S. Zhou, and J. Guan, "Local-world evolving networks with tunable clustering," *Physica A: Statistical Mechanics and Its Applications*, vol. 380, pp. 639–650, 2007.

[21] G. Wen and Z. Duan, "Dynamics behaviors of weighted local-world evolving networks with extended links," *International Journal of Modern Physics C*, vol. 20, no. 11, pp. 1719–1735, 2009.

[22] L. N. Wang, J. L. Guo, H. X. Yang, and T. Zhou, "Local preferential attachment model for hierarchical networks," *Physica A: Statistical Mechanics and Its Applications*, vol. 388, no. 8, pp. 1713–1720, 2009.

[23] C. Xia, Z. Liu, Z. Chen, S. Sun, and Z. Yuan, "Epidemic spreading behavior in local-world evolving networks," *Progress in Natural Science*, vol. 18, no. 6, pp. 763–768, 2008.

[24] "Rumor spreading in local-world evolving network," *Communications in Computer and Information Science*, vol. 227, pp. 693–699, 2011.

[25] "Cascading failures in local-world evolving networks," *Journal of Zhejiang University - Science*, vol. 10, pp. 1336–1340, 2008.

[26] I. Z. Kiss, J. C. Miller, and P. L. Simon, *Mathematics of Epidemics on Networks: From Exact to Approximate Models*, Springer, New York, NY, USA, 2017.

[27] Strogatz, "Collective dynamics of 'small-world' networks," *Nature*, vol. 393, no. 6684, pp. 440–442, 1998.

[28] C. Moore, G. Ghoshal, and M. E. J. Newman, "Exact solutions for models of evolving networks with addition and deletion of nodes," *Physical Review E: Statistical, Nonlinear, and Soft Matter Physics*, vol. 74, Article ID 36121, 2006.

[29] M. J. Keeling, "The effects of local spatial structure on epidemiological invasions," *Proceedings of the Royal Society of London, Series B: Biological Sciences*, vol. 266, no. 1421, pp. 859–867, 1999.

[30] K. T. D. Keeling and M. J. Keeling, "Modeling dynamic and network heterogeneities in the spread of sexually transmitted diseases," *Proceedings of the National Academy of Sciences*, vol. 99, no. 20, Article ID 13330, 2002.

[31] "Insights from unifying modern approximations to infections on networks," *Journal of The Royal Society Interface*, vol. 8, no. 54, pp. 67–73, 2010.

[32] "Modelling disease spread through random and regular contacts in clustered populations," *Theoretical Population Biology*, vol. 73, no. 1, pp. 104–111, 2008.