

Geographical effects on epidemic spreading in scale-free networks

Xin-Jian Xu,¹ Wen-Xu Wang,^{1,2} Tao Zhou,^{1,2} and Guanrong Chen^{1,*}

¹*Department of Electronic Engineering, City University of Hong Kong,
83 Tat Chee Avenue, Kowloon, Hong Kong SAR, China*

²*Nonlinear Science Center and Department of Modern Physics,
University of Science and Technology of China, Hefei Anhui 230026, China*

(Dated: July 3, 2006)

Many real networks are embedded in a metric space: the interactions among individuals depend on their spatial distances and usually take place among their nearest neighbors. In this paper, we introduce a modified susceptible-infected-susceptible (SIS) model to study geographical effects on the spread of diseases by assuming that the probability of a healthy individual infected by an infectious one is inversely proportional to the Euclidean distance between them. It is found that geography plays a more important role than hubs in disease spreading: the more geographically constrained the network is, the more highly the epidemic prevails.

PACS numbers: 89.75.Hc, 87.23.Ge, 05.70.Ln, 87.19.Xx

Accurately modelling epidemic spreading is an important topic in understanding the impact of diseases and the development of effective strategies for their control and containment [1]. The classical mathematical approach for describing disease spreading either ignores the population structure or treats population as distributed in a uniform medium. However, it has been argued in the past few years that many social, biological, and communication systems possess two universal characters, the small-world effect [2] and the scale-free property [3], which can be described by complex networks whose nodes represent individuals and links represent the interactions among them [4]. In view of the wide occurrence of complex networks in nature, it is interesting to study the effects of topological structures on the dynamics of epidemic spreading. Pioneering works [5, 6, 7, 8, 9] have given some valuable insights: for homogeneous networks (e.g., regular, random, and small-world networks), there are critical thresholds of the spreading rate below which infectious diseases will eventually die out; on the contrary, even infections with low spreading rates will prevail over the entire population in heterogeneous networks (e.g., scale-free networks). This radically changes many conclusions drawn from classic epidemic modelling. Furthermore, it has been observed that the heterogeneity of a population network in which the disease spreads may have noticeable effects on the evolution of the epidemic as well as the corresponding immunization strategies [8, 9, 10, 11].

In many real networks, however, individuals are often embedded in a Euclidean geographical space and the interactions among them usually depend on their spatial distances and take place among their nearest neighbors [12, 13, 14, 15]. For instance, the number of long-range links and the number of edges connected to a single node are limited by the spatial embedding, particularly in planar networks. Also, it has been proved that the characteristic distance plays a crucial role in the dynamics taking place on these networks [16, 17, 18, 19, 20, 21]. Thus, it is natural to study complex networks with geographical properties. Rozenfeld *et al.* con-

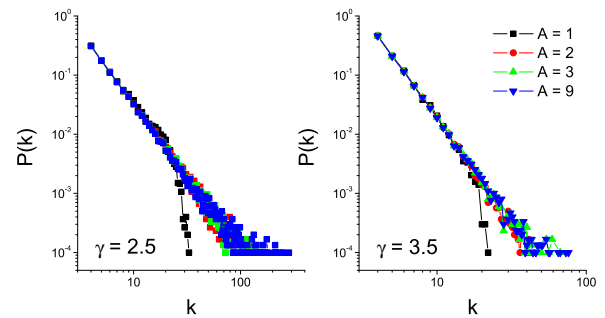


FIG. 1: (color online) Degree distribution of the LESFN model with $N = 10000$ for $\gamma = 2.5$ and 3.5 . The territory parameter A controls the influence of the geographical distance on the network structure.

sidered that the spatial distance can affect the connection between nodes and proposed a lattice-embedded scale-free network (LESFN) model [17]. Based on a natural principle of minimizing the total length of links in the system, a scale-free network can be embedded in a Euclidean space. Since distributions of individuals in social networks always depend on their spatial locations, the study of the influence of geographical structures on dynamical processes is of great importance.

In this paper, we present a modified Susceptible-Infected-Susceptible (SIS) model on the LESFN to investigate how the geographical structure affects the dynamical process of epidemic spreading. Here, we assume that the time scales governing the dynamics is much smaller than those characterizing the network evolution. Thus, the static network is suitable to use for discussing the problem under investigation. In contrast to the assumption that the infection probability is identical across successive contacts, we define the probability of a healthy individual i infected by an infectious one j to be inversely proportional to the Euclidean distance between them. Based on computer simulations, we found that when the network connectivity is less local, it will be more robust to disease spreading, regardless of the heterogeneous distribution of nodes.

*Electronic address: gchen@ee.cityu.edu.hk

The LESFN is generated as follows [17, 18]: (i) a lattice of size $N = L \times L$ with periodic boundary conditions is assumed, upon which the network will be embedded; (ii) for each site of the lattice, a preset degree k is assigned taken from a scale-free distribution, $P(k) \sim k^{-\gamma}$, $m < k < K$; (iii) a node (say i , with degree k_i) is picked randomly and connected to its closest neighbors, until its degree quatum k_i is realized or until all sites up to a distance have been explored

$$d(k_i) = A\sqrt{k_i}, \quad (1)$$

Duplicate connections are prohibited. Here, $d(k_i)$ is the spatial distance on a Euclidean plane denoting the characteristic radius of the region that node i can almost freely to reach the others; (iv) this process is repeated throughout all the sites on the lattice. Following this method, networks with $\gamma > 2$ can be successfully embedded up to a (Euclidean) distance $d(k)$ which can be made as large as desired upon the change of the territory parameter A . The model turns out to be a randomly connected scale-free network when $A \rightarrow \infty$ [22]. Typical networks with $\gamma = 2.5$ and 3.5 resulting from the embedding method are illustrated in Fig. 1. In the case of $N = 10000$, the power-low degree distributions of the LESFNs achieve their natural cutoff lengths for $A = 2, 3$ and 9 , while they end at some finite-size cutoff lengths for $A = 1$.

In order to study geographical effects on the spread of diseases, we introduce a modified SIS model. In this model, an individual is described by a single dynamical variable adopting one of the two stages: *susceptible* and *infected*. Considering the geography, we assume that the probability of a healthy individual i infected by an infectious one j is inversely proportional to the Euclidean distance between them, defined by

$$\lambda_{ij} = \frac{1}{d_{ij}^\alpha}, \quad (2)$$

where α is a tunable parameter. This is motivated by the following idea: human beings are located in territories and they interact more frequently with their nearest neighbors than those far away. The transmission of a disease is described in an effective way with the following rules: a susceptible individual at time t will pass to the infected state with the rate λ at time $t + 1$ if he is connected to infected individuals. Infected individuals at time t will pass to the susceptible state again with the unite rate at time $t + 1$. Individuals run randomly through the cycle, *susceptible* \rightarrow *infected* \rightarrow *susceptible*.

In the present work, we have performed Monte-Carlo (MC) simulations of the model with synchronously updating on the network. Initially, the number of infected nodes is 1% of the size of the network. The total sampling times are 10000 (MC time steps). After appropriate relaxation times, the systems is stabilized to a steady state. Simulations were implemented on the network model averaging over 500 different realizations. Given a network, an important observable is the prevalence ρ , which is the time average of the fraction of infected individuals in the steady state (averaging over 1000 time steps in this context).

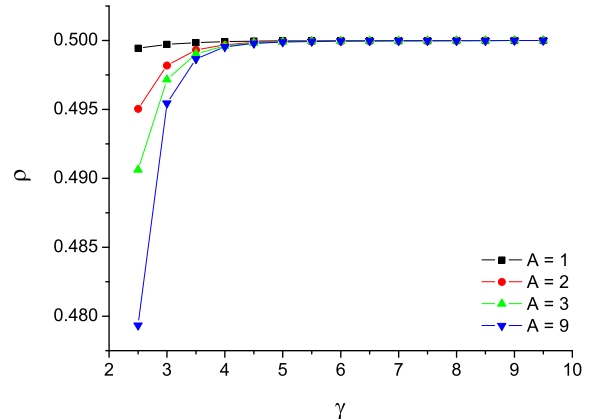


FIG. 2: (color online) Density of infected individuals in the stationary state ρ vs. the scale-free degree exponent γ for the LESFNs with different values of A . The results are obtained for $\alpha = 2$ and on networks of size $N = 10000$.

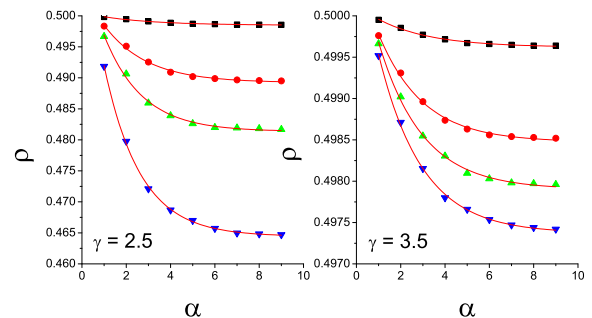


FIG. 3: (color online) Density of infected individuals ρ vs. the tunable parameter α for the LESFNs with different values of A : $A = 1$ (squares), 2 (circles), 3 (up triangles), and 9 (down triangles), respectively. The network size is $N = 10000$.

Figure 2 shows the persistence of infected individuals ρ versus the scale-free degree exponent γ for the LESFNs with different values of A when α is fixed at 2. As γ increases, all the curves approach to an asymptotic value of $\rho = 0.5$, independent of the geography of networks. The larger the parameter A , the quicker the prevalence ρ is close to the asymptotic value. This implies that the scale-free degree exponent has a slight influence on the spread of diseases when networks are more geographical constrained (smaller A) in comparison with the case of more scale-free region (larger A). It has been suggested [23] that there is a threshold $\gamma_c = 3$ which separates the two different dynamical behaviors of disease spreading, so we will focus on the values of γ at 2.5 and 3.5, respectively, to study two typical cases.

In Fig. 3, we plot the densities of infected individuals ρ versus the tunable parameter α for the LESFNs with $\gamma = 2.5$ and 3.5 , respectively. As α becomes larger, the prevalence ρ

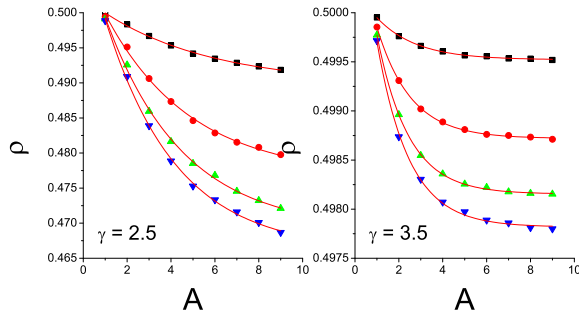


FIG. 4: (color online) Density of infected individuals ρ vs. the territory parameter A of the modified SIS model with different values α : $\alpha = 1$ (squares), 2 (circles), 3 (up triangles), and 4 (down triangles), respectively. The network size is $N = 10000$.

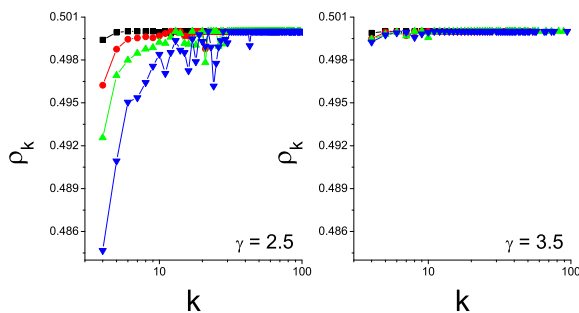


FIG. 5: (color online) The density ρ_k , defined as the fraction of nodes with connectivity k that are infected, in LESFNs of size $N = 10000$ and the territory parameters $A = 1$ (squares), 2 (circles), 3 (up triangles), and 9 (down triangles), respectively.

decreases, and all the curves approach to stable values finally. The solid lines fits to the form $\rho = B_0 + Be^{-\alpha/\alpha_0}$, implying that there is a relation of the first-order exponential decay between ρ and α . According to the definition of the spreading probability of our model (see Eq. (2)), one can easily find that the larger the parameter α , the smaller the spreading rate λ is. This results in a small fraction of infected nodes in the network. In order to understand how the geographical structure affects the epidemic dynamics, we plot the prevalence ρ as a function of the territory parameter A in Fig. 4. It shows that as A increases, the density ρ decreases. In other words, when

networks are more geographically constrained, i.e., more locally interconnected, they tend to have larger epidemic prevalence. This is different from the results observed on Barabási-Albert scale-free networks, where nodes with large degrees (called “hubs”) accelerate the spreading process and induce significant epidemic prevalence. The solid line fits to the form $\rho = C_0 + Ce^{-A/A_0}$. Similar to Fig. 3, there is also a relation of the first-order exponential decay between ρ and A .

We also provide an illustration for the behavior of the probability ρ_k that a node with given connectivity k is infected. The range of an edge is the length of the shortest paths between the nodes it connected in the absence of itself [24, 25]. If an edge’s range is l , the shortest cycle it lies on is of length $l + 1$. Thus the distribution of range in a network sketches the distribution of shortest cycles. It has been demonstrated numerically that when the spatial constraint is stronger, the LESFN has more small-order cycles [21]. In this case, the nodes are more likely to meet which speeds disease spreading. As shown in Fig. 5, in the case of $\gamma = 2.5$, there is a heterogeneous behavior of ρ_k , especially for $A = 9$, i.e., the higher a node degree, the larger the probability ρ_k . However, when a node’s degree is larger than a certain value, this feature vanishes. This implies that even a geographical network tends to a scale-free random graph, where the hub effect on the spreading dynamics is still limited, i.e., a node’s potential infectivity is not strictly equal to its degree due to geographical effects. This effect is more stronger for $\gamma = 3.5$, that is, all the curves are nearly linearly independent of the value of the territory parameter.

In conclusion, we have studied geographical effects on the spreading phenomena in lattice-embedded scale-free networks, in which a territory parameter A controls the influence of the geography on the network structure and therefore on the epidemic dynamics. We studied the modified SIS model in which the probability of a healthy individual infected by an infectious one is inversely proportional to the Euclidean distance between them. Our main finding is that when the network is more geographically constrained, i.e., with heavier local connections, the epidemic prevalence will be more significant. This indicates that networks with more local connections have a higher risk to disease spreading. On the contrary, while the network is more scale-free, it will be more robust to disease spreading, regardless of the heterogeneous connectivity of the network.

G. Chen acknowledges the Hong Kong Research Grants Council for the CERG Grant CityU 1114/05E. T. Zhou acknowledges the Natural Science Foundation of China for the Grant No. 70471033.

[1] R.M. Anderson and R.M. May, *Infectious Diseases in Humans* (Oxford University Press, Oxford, 1992).
 [2] D.J. Watts and S.H. Strogatz, *Nature* **393**, 440 (1998).
 [3] A.-L. Barabási and R. Albert, *Science* **286**, 509 (1999).
 [4] R. Albert and A.-L. Barabási, *Rev. Mod. Phys.* **74**, 47 (2002); S.N. Dorogovtsev and J.F.F. Mendes, *Adv. Phys.* **51**, 1079 (2002); M.E.J. Newman, *SIAM Rev.* **45**, 167 (2003).

[5] C. Moore and M.E.J. Newman, *Phys. Rev. E* **61**, 5678 (2000); *ibid* **62**, 7059 (2000).
 [6] R.M. May and A.L. Lloyd, *Phys. Rev. E* **64**, 066112 (2001).
 [7] M. Kuperman and G. Abramson, *Phys. Rev. Lett.* **86**, 2909 (2001).
 [8] R. Pastor-Satorras and A. Vespignani, *Phys. Rev. Lett.* **86**, 3200 (2001); *Phys. Rev. E* **63**, 066117 (2001); *ibid* **65**, 036104

- (2002).
- [9] Y. Moreno, R. Pastor-Satorras, and A. Vespignani, *Eur. Phys. J. B* **26**, 521 (2002).
- [10] R. Cohen, S. Havlin, and D. ben-Avraham, *Phys. Rev. Lett.* **91**, 247901 (2003).
- [11] M. Barthélemy, A. Barrat, R. Pastor-Satorras, and A. Vespignani, *Phys. Rev. Lett.* **92**, 178101 (2004); *J. Theor. Biol.* **235**, 275 (2005).
- [12] R. Durrett, *SIAM Review* **41**, 677 (1999).
- [13] S.-H. Yook, H. Jeong, and A.-L. Barabási, *Proc. Natl. Acad. Sci. USA* **99**, 13382 (2002).
- [14] G. Nemeth and G. Vattay, *Phys. Rev. E* **67**, 036110 (2003).
- [15] M.T. Gastner and M.E.J. Newman, *Eur. Phys. J. B* **49**, 247 (2006).
- [16] S.S. Manna and P. Sen, *Phys. Rev. E* **66**, 066114 (2002).
- [17] A.F. Rozenfeld, R. Cohen, D. ben-Avraham, and S. Havlin, *Phys. Rev. Lett.* **89**, 218701 (2002).
- [18] D. ben-Avraham, A.F. Rozenfeld, R. Cohen, and S. Havlin, *Physica A* **330**, 107 (2003).
- [19] Y.-B. Xie, T. Zhou, W.-J. Bai, G. Chen, W.-K. Xiao, and B.-H. Wang, [arXiv:physics/0605054](https://arxiv.org/abs/physics/0605054).
- [20] C.P. Warren, L.M. Sander, and I. M. Sokolov, *Phys. Rev. E* **66**, 056105 (2002).
- [21] L. Huang, L. Yang, and K. Yang, *Phys. Rev. E* **73**, 036102 (2006).
- [22] M.E.J. Newman, S.H. Strogatz, and D.J. Watts, *Phys. Rev. E* **64**, 026118 (2001).
- [23] A. Vazquez, *Phys. Rev. Lett.* **96**, 038702 (2006); [arXiv:q-bio.PE/0603010](https://arxiv.org/abs/q-bio.PE/0603010).
- [24] D.J. Watts, *Small Worlds: The Dynamics of Networks Between Order and Randomness* (Princeton University Press, New Jersey, 1999).
- [25] S.A. Pandit and R.E. Amritkar, *Phys. Rev. E* **60**, R1119 (1999).