# EPIDEMIC SPREAD MODELING IN HETEROGENEOUS NETWORKS Integrating Barabasi-Albert Network and Sirs Model with Methodological Exploration and Future Prospects

by

***Zhibin LUO[a], Haiyan LI[b*], and Haibin LI[a]***

[a] College of Science, Inner Mongolia University of Technology, Hohhot, China
[b] Department of Endocrinology, Affiliated Hospital of Inner Mongolia Medical University, Hohhot, China

Original scientific paper
https://doi.org/10.2298/TSCI2602899L

*This paper integrates the Barabasi-Albert network model and the Susceptible-infectious-recovered-susceptible (SIRS) model to explore epidemic spread modeling in heterogeneous networks. Notwithstanding the simplifying assumptions inherent in the model and the absence of empirical verification, the proposed approach provides a theoretical foundation for comprehending multi-scale epidemic transmission. The Barabasi-Albert subnetworks and SIRS-based simulation method are described in detail, and they can be used to create a realistic network environment for further analysis. Subsequent research endeavors will center on the implementation of this framework in relation to authentic data and the integration of more intricate elements, such as human behavior and environmental factors.*

Keywords: *Barabasi-Albert network, SIRS model, complex network theory, epidemic modeling*

## Introduction

In the contemporary era of accelerated economic globalization and increasing population mobility, the global propagation of infectious diseases has reached a distressingly rapid pace. Notable examples of this phenomenon include the SARS outbreak of 2002-2003 and the H1N1 influenza pandemic, which have vividly demonstrated the high vulnerability of populations with extensive interconnections to novel pathogens [1]. The etiology of diseases is typically localized in a specific region before rapidly disseminating worldwide, initially affecting densely populated urban areas and subsequently spreading to peripheral regions [2]. For instance, the cholera epidemic of the 19$^{th}$ century, which originated in India, rapidly disseminated globally. A parallel can be drawn to the SARS epidemic that emerged in Guangdong Province, China, from 2002 to 2003. That epidemic rapidly propagated to major cities such as Beijing and Guangzhou, ultimately impacting 37 countries [3].

Urban areas, characterized by their high population density and extensive network of inter-city transportation connections, function as pivotal nodes in the disease transmission network. These urban hubs not only facilitate the rapid local spread of diseases but also serve as important gateways for the dissemination of diseases to surrounding areas [2, 3]. Consequently, the construction of models and the undertaking of exhaustive research on the propa-

* Corresponding author, e-mail: 13614810768@163.com

gation of diseases within such interconnected urban networks is of paramount importance for global public health.

The utilization of mathematical models in the depiction of epidemic transmission patterns has a long-standing history [4-6]. The utilization of mathematical models in the field of epidemiology can be traced back to the 18th century research on smallpox [1]. In the early 20th century, Kermack and McKendrick [2, 7] introduced the SIR model. This model posited a compartmental perspective on epidemic spread, inspiring the development of derivative models such as SEIR, SIRS, and SIRV. However, these early models frequently assumed homogeneous mixing among individuals, which resulted in the neglect of the complex structures that exist in real-world social networks [8].

In the domain of modern epidemiology, complex network theory has emerged as a robust framework for modeling social and biological interactions [9]. The study of complex networks can be traced back to the seminal work of Erdos and Renyi on random graphs [10]. Subsequently, Watts and Strogatz [11] expanded upon this concept, applying it to small-world networks. In a similar vein, Barabasi and Albert proposed scale-free networks [12]. The Barabasi-Albert (BA) network model is particularly pertinent to the field of epidemic modeling. This is due to the fact that the model contains hubs, which are analogous to the *super-spreaders* observed in real-life epidemic situations [13].

The objective of this paper is to establish a methodological foundation for multicity epidemic analysis. The text commences with an exposition of the BA network structure, followed by a delineation of the SIRS model. The following discussion will elucidate the methodology by which BA subnetworks can be utilized to represent individual urban centers and the manner in which their interconnections can simulate population mobility between cities. Furthermore, a simulation method based on the SIRS model will be discussed. It is imperative to acknowledge the significance of incorporating network heterogeneity in order to accurately model realistic epidemic dynamics. However, it should be noted that although the proposed models and methods have certain theoretical significance, the BA network's construction rules and the SIRS model's assumptions may not fully reflect the complexity of real-world scenarios. To illustrate, the preferential attachment rule in the BA network may be overly simplistic in its representation of the complex relationships between cities in reality. Similarly, the SIRS model may not fully account for all the influencing factors in real-life epidemic situations. Future research endeavors must address these limitations to enhance the accuracy and practicality of the models.

## The Brabasi-Albert network

### *Network structure*

The development of the BA network is characterized by an iterative growth process. The process begins with the formation of a modest initial network comprising m nodes. As the network evolves, new nodes are added sequentially. The attachment of each newly introduced node to existing nodes is governed by a probability that is directly proportional to the degree of the existing nodes. This mechanism is known as preferential attachment [12].

Mathematically, this process gives rise to a power-law degree distribution:

$$P(k) \sim k^{-\gamma}$$

where $\gamma > 0$. This distribution suggests the coexistence of a small number of high-degree *hub* nodes and a large number of low-degree nodes [9, 10]. In the context of epidemiology, these

hub nodes are of particular significance. It has been posited that these entities may act as *super-spreaders*, a term used to describe the propagation of a disease from one infected individual to a disproportionately large number of other nodes within a network [13].

This characteristic distinguishes the BA network from other types of networks [14-16]. To illustrate, in the context of random networks, the degree distribution typically exhibits a Poisson-like pattern. In such networks, the probability of connection between nodes is relatively uniform, and the presence of distinct hub nodes is not observed. Conversely, small-world networks prioritize the minimization of average path lengths between nodes. Although these networks manifest certain distinctive characteristics with regard to information diffusion and disease propagation, they do not demonstrate the heavy-tailed degree distribution that is hallmark of scale-free networks, such as the BA network.

### *Comparison with other models*

*Random Networks (Erdos–Renyi).* Random networks are predicated on the assumption that the probability of an edge existing between any two nodes is uniform. This simplicity results in a paucity of hub structures. Consequently, the propagation dynamics of diseases in random networks differ considerably from those in BA networks. In random networks, the propagation of an epidemic is more uniformly dispersed, and the impact of individual nodes on the overall diffusion is comparatively more balanced. Nevertheless, in actual, real-world scenarios in which the presence of super-spreaders (hub nodes) can significantly influence epidemic development, the random network model may not accurately reflect the true nature of epidemic spread [10].

*Small-World Networks (Watts-Strogatz).* The concept of small-world networks is predicated on the observation that these networks are characterized by short average path lengths. The consequence of this property is that information or diseases can spread quickly across the network. However, a distinguishing feature of scale-free networks is the presence of a heavy-tailed degree distribution, which is absent in this case. This suggests that, in contrast to BA networks, there is an absence of dominant hub nodes in small-world networks. Despite the capacity of small-world networks to elucidate certain phenomena associated with rapid transmission over brief distances, they may lack the comprehensive capability to entirely justify the function of highly connected nodes in expediting epidemic propagation [11].

*Scale-Free Networks (Barabasi-Albert).* A distinguishing characteristic of scale-free networks, such as the BA network, is the presence of highly connected nodes that follow a power-law distribution. This property is of significant importance in the context of epidemic spread. In the context of large-scale Bayesian networks, the epidemic threshold frequently approaches zero. This finding suggests that even a limited number of initial infections has the potential to trigger a large-scale epidemic, thereby underscoring the rapid and far-reaching nature of disease propagation within such networks [13]. It is imperative to comprehend these discrepancies to ascertain the most suitable network model when investigating epidemic dynamics in various real-world scenarios.

## The SIRS model

### *Model description*

The SIRS model is a compartmental model that categorizes individuals within a population into three distinct states: susceptible, $S$, infectious, $I$, and recovered, $R$. In this model, the transition of individuals between these states is governed by specific rates.

Susceptible individuals are those who are vulnerable to the disease and can contract it upon coming into contact with infectious individuals. The rate at which this occurs is denoted as the infection rate $\beta$. When a susceptible individual interacts with an infectious one, there is a probability determined by $\beta$ that the susceptible individual will become infected.

Infected individuals, on the other hand, are carriers of the disease. They can spread the infection to susceptible individuals in their vicinity. However, over time, infected individuals recover at a rate $\alpha$. Once they recover, they enter the recovered state, $R$.

An important aspect of the SIRS model is that recovered individuals do not necessarily have lifelong immunity. At a rate $\gamma$, they can lose their immunity and revert back to the susceptible state. This feature makes the SIRS model suitable for representing diseases where immunity wanes over time, such as certain seasonal flu strains.

The dynamics of the SIRS model can be mathematically described by the following system of differential equations:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = -\beta SI + \gamma R, \quad \frac{\mathrm{d}I}{\mathrm{d}t} = \beta SI - \alpha I, \quad \frac{\mathrm{d}R}{\mathrm{d}t} = \alpha I - \gamma R \tag{1}$$

where $S$, $I$, and $R$ are the number of individuals in the susceptible, infectious, and recovered states, respectively, and $t$ – the time. These equations provide a quantitative way to analyze how the proportion of individuals in each state changes over time as the epidemic progresses.

### *Application in epidemic studies*

*Temporary immunity.* A considerable number of prevalent infectious diseases do not engender lifelong immunity in those who recover. For instance, the common cold virus can infect an individual multiple times due to the gradual dissipation of immunity acquired from prior infections. The SIRS model is an appropriate framework for the analysis of immune-related transitions. By adjusting the value of the rate $\gamma$, researchers can model how quickly immunity is lost and how this affects the overall epidemic dynamics. When set to a relatively high value, $\gamma$ suggests that individuals who have recovered lose their immunity rapidly, which results in a larger pool of susceptible individuals over time. This phenomenon potentially increases the likelihood of future outbreaks.

*Public health policy.* The SIRS model is a valuable tool for policymakers. By observing the alterations in the infection curve (which represents the number of infectious individuals over time) under varying values of the infection rate, $\beta$, and recovery rate, $\alpha$, policymakers can estimate the effectiveness of various public health interventions. For instance, social distancing measures aim to reduce the value of the parameter $b$ by minimizing contact between individuals. The implementation of social distancing is supported by evidence derived from simulations utilizing the SIRS model, which demonstrate that a substantial decrease in the parameter $\beta$ results in a notable flattening of the infection curve. Conversely, vaccination programs can be modeled by adjusting the rates in the SIRS framework. Vaccination can be conceptualized as a strategy to diminish the population of susceptible individuals (by transitioning them to a vaccinated, protected state, which can be integrated as an extension of the SIRS model) or augment the recovery rate, $\alpha$, if vaccinated individuals exhibit a more expeditious recovery from the disease.

*Combined models.* The SIRS model can be extended into various variants to incorporate additional factors relevant to epidemic spread. For instance, the SEIRS model incorporates a latent state, $E$, to account for the period between infection and the onset of infectious-

ness. This is of particular importance for diseases with a substantial incubation period, such as the novel coronavirus (SARS-CoV-2). Another extension is the SIRV model, which incorporates vaccination. In this model, a new category is introduced for vaccinated individuals, and the model parameters are adjusted to reflect the impact of vaccination on the spread of the disease. Furthermore, the development of models that incorporate behavioral changes within the population is a potential avenue for further research. As an epidemic progresses, there is a possibility that individuals will modify their behavior. This modification may take the form of a reduction in non-essential travel or an increase in hand-washing frequency. The modeling of these behavioral changes can be achieved by adjusting the values of $\beta$, $\alpha$, or $\gamma$ in the SIRS framework. This adjustment allows for a more realistic representation of real-world epidemic scenarios.

## Epidemic simulation method

### *Creation of Barabasi-Albert subnetworks*

In order to accurately simulate a realistic urban system, it is essential to generate multiple BA subnetworks of varying scales. The process commences with the formation of a small, connected network comprising m nodes. This initial network serves as the foundation upon which the larger subnetworks are built.

Subsequently, new nodes are iteratively incorporated into the network. For each new node addition, a crucial step is executed based on the preferential attachment rule. This rule stipulates that the probability of a new node connecting to an existing node is directly proportional to the degree of that existing node. In practical terms, nodes with a higher number of connections (*i.e.*, higher-degree nodes) are more likely to attract new connections. This phenomenon is analogous to real-world scenarios in which well-connected entities, such as large cities with extensive transportation and social networks, tend to attract a greater number of new connections or interactions.

The iterative addition of nodes persists until each subnetwork attains the desired size. The formation of heterogeneous networks is achieved by creating subnetworks with varying numbers of nodes. This heterogeneity is a pivotal element, as it more accurately reflects the intricacies and variations inherent in actual urban centers. For instance, certain cities may possess a substantial population and exhibit a high degree of connectivity, while others may be smaller and less connected. The resulting BA subnetworks can serve as representations of these differences, thereby providing a more realistic basis for epidemic simulation.

### *Interconnection of subnetworks*

Following the successful creation of the BA subnetworks, the subsequent critical step involves the interconnection of these networks to simulate the relationships between disparate cities. The interconnection process has been meticulously designed to emulate the authentic interactions that transpire between urban centers in the real world.

For each subnetwork, a node is selected at random. This selected node is then connected to nodes in other subnetworks, following a preferential attachment strategy. In practical terms, this phenomenon is analogous to the stronger connections that some cities establish with other cities due to factors such as population flow, economic activities, and cultural exchanges. For instance, major economic hubs such as New York and London have extensive connections with numerous other cities around the world through international flights, trade routes, and business partnerships.

This connection method ensures that the resulting comprehensive network captures the complex relationships between cities. Consequently, it offers an optimal platform for conducting epidemic simulations. This interconnected network facilitates a more realistic modeling of the spread of an epidemic, taking into account the potential for disease transmission within a single city (represented by a subnetwork) and between different cities (through the connections between subnetworks). As illustrated in fig. 1, the interconnected network structure can be conceptualized. This visualization facilitates comprehension of the spatial and structural characteristics of the network, which are imperative for the analysis of epidemic spread patterns.

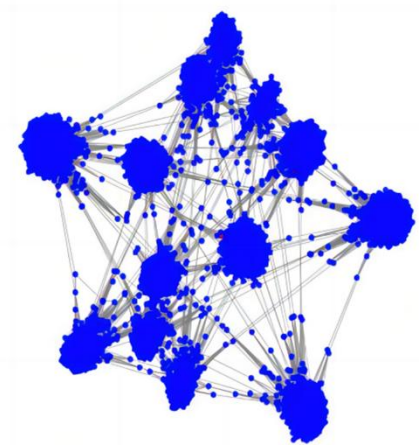

**Figure 1. Simulated community network of Wuhan**

### *The SIRS model simulation*

Within the context of the simulation, a predetermined percentage of nodes within the entire network, which is interconnected, are arbitrarily assigned as initially infected. These infected nodes function as the primary origin of the epidemic's propagation within the network. The remaining nodes are designated as *susceptible*, indicating their vulnerability to infection.

At each time step of the simulation, the states of the nodes are updated in accordance with the rules of the SIRS model. When a susceptible node comes into contact with an infected node, there is a probability, determined by the infection rate, $\beta$, that the susceptible node will become infected. Conversely, infected nodes exhibit a probability of recovery at each time step, determined by the recovery rate, $\alpha$. Upon the recovery of an infected node, it transitions to a recovered state. However, it should be noted that recovered nodes are not necessarily immune for life. According to the specified rate, $\gamma$, the subjects can undergo a loss of immunity and revert to a state of susceptibility.

During the simulation, it is imperative to collect data on the number of nodes in each state (susceptible, infectious, and recovered) at every time step. This data collection facilitates a thorough examination of the epidemic's propagation patterns. By observing the temporal dynamics of infected nodes, researchers can ascertain the epidemic peak, its rate of spread, and its rate of subsidence. Furthermore, an analysis of the fluctuations in the number of susceptible and recovered nodes offers insights into the epidemic overall dynamics, including the development of herd immunity and the potential for future outbreaks. This data-driven approach facilitates a more profound comprehension of the epidemic and can inform the development of effective intervention strategies.

## Conclusions

This article has established a substantial foundation for the study of epidemic spread in heterogeneous networks. Integration of the BA network model and the SIRS model, along with the refinement of the epidemic simulation method, has yielded a substantial theoretical framework and a pragmatic approach.

The BA network structural characteristics, including its power-law degree distribution and the presence of hub nodes, offer a crucial perspective on the rapid spread of diseases

in a networked population. It has been demonstrated that these hubs, akin to *super-spreaders* in the real world, have the capacity to exert a substantial influence on the progression of an epidemic. Conversely, the SIRS model effectively captures the dynamic states of individuals within the population, accounting for the loss of immunity over time, a common phenomenon in many infectious diseases.

The detailed simulation method delineated in this paper facilitates the creation of a realistic network environment. The generation of BA subnetworks with different scales and their interconnection based on preferential attachment rules has been demonstrated to mimic the complexity of real-world urban systems. The SIRS model can be applied to this network to analyze the factors that affect epidemic spread, such as the initial infection rate, recovery rate, and the rate of immunity loss.

However, it is imperative to acknowledge the limitations of this study. The BA network model offers a simplified approach to the formation of real-world networks, while the assumptions inherent in the SIRS model may not fully capture the complexities inherent in actual epidemic situations. To illustrate, the preferential attachment rule in the BA network may not take into account all the factors that influence node connections in real-world social or urban networks. The SIRS model is predicated on the assumption that the constant rates of infection, recovery, and immunity loss accurately represent the complex and time-varying nature of these processes in reality. However, this may not be the case.

Future research endeavors should prioritize the identification of these discrepancies and seek to develop effective interventions that address these gaps. Firstly, it is imperative to apply this framework to real-world data. The utilization of authentic epidemiological data, network connectivity data, and other pertinent information enables the validation and refinement of models, thereby enhancing their accuracy and applicability. Secondly, exploring more sophisticated models that incorporate additional factors is crucial. Human behavior, including shifts in social distancing measures over time, and environmental factors, such as weather conditions that influence virus survival, can exert a substantial influence on the propagation of epidemics. The incorporation of these factors into the model will result in a more comprehensive understanding of epidemic dynamics and better-informed decision-making in public health.

In summary, while this study signifies a significant advancement in the comprehension of epidemic propagation in heterogeneous networks, there is ample opportunity for refinement and expansion. By addressing the limitations and expanding the scope of research, we can enhance our ability to predict and control epidemics more effectively in the future.

## References

[1] Dietz, K., Heesterbeek, J. A. P., Daniel Bernoulli's Epidemiological Model Revisited, *Mathematical Biosciences*, *180* (2002), 1, pp. 1-21
[2] Kermack, W. O., McKendrick, A. G., Contributions to the Mathematical Theory of Epidemics – II. The Problem of Endemicity, *Bulletin of Mathematical Biology*, *53* (1991), 1, pp. 57-87
[3] Furushima, D., *et al.*, Estimation of the Basic Reproduction Numbers of Novel Influenza a (H1N1) pdm09 in Elementary Schools Using the SIR Model, TONURSJ-11-64, *The Open Nursing Journal*, *11* (2017), pp. 64-72
[4] Zhang, Y., *et al.*, Unraveling Atherosclerosis: A Nonlinear Coupled Mathematical Model with Dual Free Boundaries − Solution and Numerical Insights. *Journal of Applied and Computational Mechanics*, *11* (2025), 4, pp. 1162-1171
[5] Alshomrani, N. A., *et al.*, Homotopy Perturbation Method for Solving a Nonlinear System for an Epidemic0 *Advances in Differential Equations and Control Processes*, *31* (2024), 3, pp. 347-355

[6] Alsubaie, A. A., *et al.*, Adomian′s Method for Solving a Nonlinear Epidemic Model, *Advances in Differential Equations and Control Processes*, *31* (2024), 1, pp. 95-107
[7] Basnarkov, L., *et al.*, Non-Markovian SIR Epidemic Spreading Model of COVID-19, *Chaos, Solitons & Fractals*, *160* (2022), 112286
[8] Ahmed, M., *et al.*, Bifurcation Analysis and Optimal Control of Discrete SIR Model for COVID-19, *Chaos, Solitons & Fractals*, *174* (2023), 113899
[9] Yagasaki, K., Nonintegrability of the SEIR Epidemic Model, *Physica D: Nonlinear Phenomena*, *453* (2023), 133820
[10] Erdos, P., Renyi, A., On Random Graphs I, *Publicationes Mathematicae*, *6* (1959), pp. 290-297
[11] Watts, D. J., Strogatz, S. H., Collective Dynamics of 'Small-World' Networks, *Nature*, *393* (1998), 6684, pp. 440-442
[12] Barabasi, A. L., Albert, R., Emergence of Scaling in Random Networks, *Science*, *286* (1999), 5439, pp. 509-512
[13] Kitsak, M., *et al.*, Identification of Influential Spreaders in Complex Networks, *Nature Physics*, *6* (2010), 11, pp. 888-893
[14] He, J.-H., Transforming Frontiers: The Next Decade of Differential Equations and Control Processes, *Advances in Differential Equations and Control Processes*, *32* (2025), 1, 2589
[15] Li, H. B., *et al.*, Correlation Analysis Based on Neural Network Copula Function, *Thermal Science, 27* (2023) , 3A, pp. 2081-2089
[16] Nosonovsky, M., Aglikov, A. S. Triboinformatics: Machine Learning Methods for Frictional Instabilities, *Facta Universitatis: Series Mechanical Engineering, 22* (2024), 3, pp. 423-433

Paper submitted: December 15, 2024
Paper revised: March 21, 2025
Paper accepted: March 23, 2025