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Viral Load-Driven Modeling of Epidemic Spread in Networks

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Abstract

This paper studies epidemic transmission in scale-free networks using an SIS model with viral load-dependent infectivity. A network disease model is developed and analyzed via HMF theory, deriving the basic reproduction number and its link to equilibrium stability. Simulations showing how viral load, network heterogeneity, and scale jointly affect transmission. Experiments indicate that: High-er initial viral load increases infection prevalence; larger degree exponent reduces infection due to low-degree node "transmission dead ends"; infection grows with network size in scale-free networks.

Keywords: Complex Networks, Epidemic Transmission, Viral Load Dynamics.

1. Introduction

Epidemics, as a historical challenge threatening human life and health, have per-plexed countless researchers from ancient times to the present. In 1926, Kermack et al. (1927) established the most classic SIR compartment model in the history of epidemiology to explore the transmission characteristics of the London Black Death and the Bombay Plague. In 1932, Kermack et al. (1932) further developed the SIS model and proposed the "threshold theory" to determine whether an infectious disease would continue to spread or gradually die out in the future. The basic reproduction number serves as a threshold in deterministic dynamic models to judge the survival or extinction of an epidemic. When the basic reproduction number is less than 1, the infectious disease will gradually disappear; when it is greater than 1, the disease will continue to spread. As research on infectious diseases has deepened, the types of data related to these diseases have become more diverse. Since the 21st century, the integration of complex networks (such as scale-free networks (Zhang et al., 2024), random networks (Granger et al., 2024), and small-world networks (Fan et al., 2024)) with epidemic models has become a research hotspot.

Whether in deterministic models or stochastic probability models, the relationship between viral load and transmission probability has been widely discussed. It is now generally believed that the higher the viral load an individual carries, the greater the probability of transmission (Marc et al., 2021; Cevik et al., 2021). Therefore, we refer to the infectivity function pro-posed by Becker (Becker, 2017), which measures the infectivity level of an infected individual based on their viral load.

This paper primarily explores the spread of diseases on complex networks using the SIS model. By constructing infectivity rate functions based on parameters such as viral load, the model is analyzed using the mean-field approach. The basic reproduction number is then used to demonstrate the conditions under which a disease will spread or die out. Finally, numerical simulations are conducted to investigate the impact of factors such as viral load on disease transmission.

2. Model Construction

In this paper, we establish a dynamical model for the spread of an SIS compartmental model in a scale-free network. The model assumes: 1. Each node in the network corresponds to an individual, and a connection between two nodes represents mutual contact, allowing for the flow of the virus; otherwise, no flow occurs. 2. The network's topology remains unchanged during the disease spread, and it is assumed that the population's immigration and emigration are in equilibrium, without considering birth and death rates. 3. It is assumed that the disease status of individuals is not influenced by public resource interventions, meaning external measures such as vaccination and quarantine are not considered.

Based on these assumptions, we introduce the concept of an infectiousness function (Becker, 2017), which quantifies the level of infectiousness through the viral load carried by infected individuals. The infectiousness function $x(t)$ represents the viral load carried by an infected individual t units of time after infection. We assume that infected individuals initially have no immunity, and their immune system begins to function T_I days after infection. The viral load follows a deterministic birth-death process (Glass and Becker, 2009), with the specific formula: $\frac{dx(t)}{dt} = \begin{cases} \lambda x(t) & 0 < t < T_I \\ (\lambda - d)x(t) & T_I < t < \infty \end{cases}$, where λ represents the birth rate of the virus, and d represents the death rate of the virus. The expression for the infectiousness function is: $x(t) = \begin{cases} x(0)e^{\lambda t} & 0 < t < T_I \\ x(0)e^{\lambda T_I}e^{(\lambda-d)(t-T_I)} & T_I < t < \infty \end{cases}$, where x_0 represents the initial viral load carried by the infected individual. Furthermore, when the virus spreads for a sufficiently long time, we can derive the escape probability $\theta = e^{-\alpha \int_0^\infty x(t)dt}$, where α represents the degree of contact between individuals, generally a constant (in this paper, $\alpha = 1$ is taken). And $1 - \theta$ can be interpreted as the infection probability β when a susceptible individual comes into contact with an infected individual. At the same time, we refer to the average infectious period of epidemics since the 20th century and the average number of days from diagnosis to full recovery (Cevik et al., 2021; Rafiq et al., 2023; Tenforde et al., 2020), setting the recovery rate in the SIS model as $\mu = 1/10$.

In summary, we derive the calculation formulas for the infection rate and recovery rate during epidemic transmission. Combining the analytical methods of the SIS model on scale-free networks, this paper uses the heterogeneous mean-field method to obtain the dynamical equations for disease spread (Pastor-Satorras and Vespignani, 2001), with the specific formula:

$$\begin{cases} \frac{dI_k(t)}{dt} = (1-\theta)k\Theta(t)\left(1 - I_k(t)\right) - \mu I_k(t) \\ \frac{dS_k(t)}{dt} = \mu I_k(t) - (1-\theta)k\Theta(t)\left(1 - I_k(t)\right) \end{cases} \tag{1}$$

where $\Theta(t) = \sum_k \frac{k \cdot P(k) \cdot I_k(t)}{\langle k \rangle}$ represents the probability that any given edge in the network at time t. is connected to an infected node, and $P(k)$ represents the degree distribution of nodes in the network, $\langle k \rangle$ represents the average degree of the network.

Based on the existence of a positive equilibrium point, the basic reproduction number of the system (1) can be derived as:

$$R_0 = \frac{\beta \left\langle k^2 \right\rangle}{\mu \langle k \rangle} = \frac{(1-\theta)\left\langle k^2 \right\rangle}{\mu \langle k \rangle} \tag{2}$$

3. Model Analytics

Next, we will demonstrate through two theorems that system (1) has a basic reproduction number R_0, and the stability of the model's equilibrium points is related to R_0.

Theorem 3.1 For system (1), we assert:

(1) If $R_0 < 1$, the disease-free equilibrium E_0 is globally asymptotically stable.

If $R_0 > 1$, the disease-free equilibrium is unstable.

(2) If $R_0 > 1$, the endemic equilibrium E^* is globally asymptotically stable.

Proof: From the second equation of system (1), we have

$$\frac{dS_k(t)}{dt} < \mu - \mu S_k(t) \tag{3}$$

Consider the auxiliary system

$$\frac{dS_k(t)}{dt} = \mu - \mu S_k(t) \tag{4}$$

Its unique positive equilibrium is $\hat{S} = 1$. When $t \to +\infty$, for $\forall \varepsilon > 0$, we have $S_k(t) \leq \hat{S} + \varepsilon$. So we obtain

$$\frac{dI_k(t)}{dt} \leqslant k(1-\theta)(\hat{S}+\varepsilon)\Theta(t) - \mu I_k(t) \tag{5}$$

Let

$$V(t) = \sum_{k=1}^{k_{\max}} \frac{kP(k)}{\mu\langle k\rangle} I_k(t) \tag{6}$$

$$\begin{aligned} \frac{dV(t)}{dt} &= \sum_{k=1}^{k_{\max}} \frac{kP(k)}{\mu\langle k\rangle} \left[k(1-\theta)(\hat{S}+\varepsilon)\Theta - \mu I_k(t)\right] \\ &= \Theta\left(R_0(1+\varepsilon) - 1\right) \end{aligned} \tag{7}$$

Since $R_0 < 1$, taking a sufficiently small $\varepsilon > 0$ such that $R_0(1+\varepsilon) < 1$, we have $\frac{dV(t)}{dt} \leq 0$, with equality hold-ing if and only if $I_k = 0$. By LaSalle's invariance principle, we conclude that $\lim_{t\to\infty} I_k(t) = 0$ as $\lim_{t\to\infty} S_k(t) = 1$.

For the stability of the endemic equilibrium when $R_0 > 1$, Wang et al. have proven that as long as there are initially infected nodes in the network, the en-demic equilibrium E^* is globally asymptotically stable when $R_0 > 1$ (Wang and Dai, 2008).

Q.E.D.

4. Numerical Experiments

To verify the theorem, we conducted extensive numerical simulation experiments. In the experiments, we set the number of network nodes to range from $N = 1000$ to $N = 5000$. When constructing Erdős-Rényi (ER) random networks, we used a fixed probability $p = \frac{\langle k\rangle}{N-1}$ to add edges (Rafiq et al., 2023). For scale-free net-works, we employed the uncorrelated configuration model (Catanzaro et al., 2005), where the degree distribution of nodes follows a power-law distribution with exponent $\gamma = 2$, and the maximum degree was set to follow $k_{\max} \sim \sqrt{N}$, with a

minimum degree of $k_{\min} = 3$ (Sottile et al., 2024). In all simulation experiments, the initial proportion of infected nodes was set to $I_0 = 0.1$. To ensure the accuracy of the experiments, each data point obtained during the simulations was averaged over 100 repeated experiments under the same conditions to minimize experimental errors.

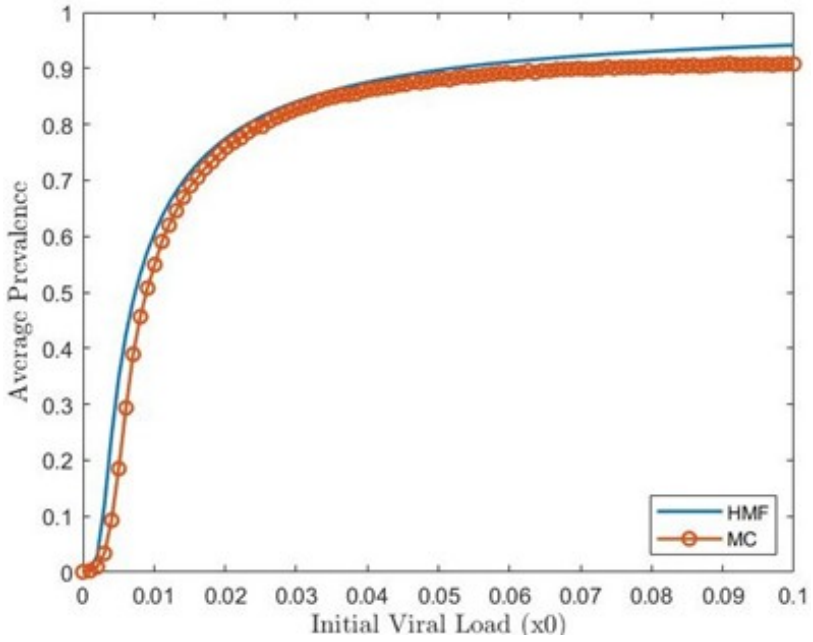

Figure 1: Comparison curves of MC simulation and HMF method. The figure depicts the curve of the infection proportion I in a scale-free network as a function of x_0. Other model parameters were set as $\lambda = 1$, $d = 2$, $T_1 = 1$, and $\mu = 0.1$.

First, we compared Monte Carlo (MC) simulations with theoretical numerical iterations on a scale-free network with $n = 1000$ to validate the effectiveness of the heterogeneous mean-field (HMF) method. Given the characteristics of the HMF analytical approach, this paper calculates the weighted average of the infection proportions of nodes with different degrees based on the degree probability distribution of the network, yielding the curve of the average infection proportion as a function of the initial viral load. The experimental results are shown in Figure 1. From the figure, it can be observed that the MC simulations and the HMF analytical method exhibit the same trend and good consistency in the infection proportion curve as a function of x_0, indicating that the use of the heterogeneous mean-field theoretical approach for system (1) is feasible. However, we also note slight discrepancies be-tween the theoretical and simulation methods, which may arise because the HMF method neglects dynamic correlations between network nodes during disease trans-mission. On the other hand, both methods show a consistent increasing trend in the infection proportion of network nodes as x_0 increases, with the slope of the curve gradually decreasing. This suggests that the higher the initial viral load carried by individuals, the greater the infection proportion of the entire network; however, as the initial viral load x_0 increases, the rate of increase in the infection pro-portion gradually slows down.

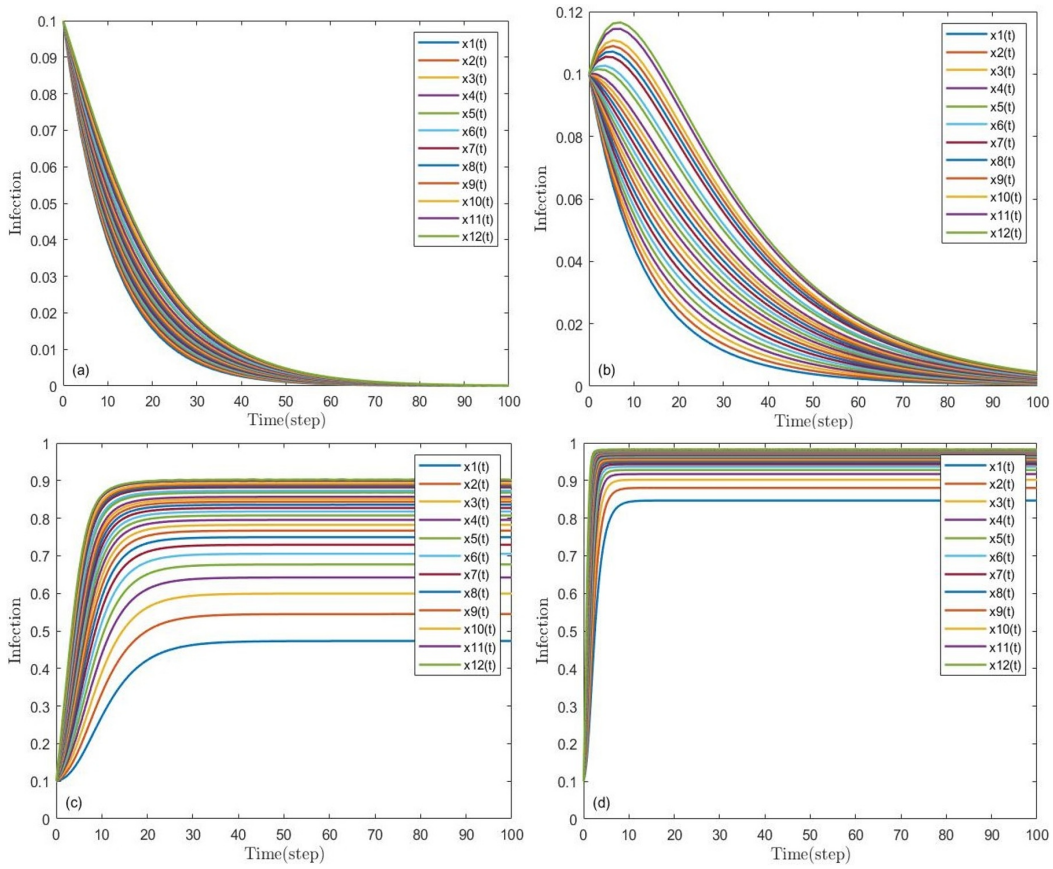

Figure 2: The variation curves of epidemic infection proportions among nodes of different degrees in scale-free networks over time under different basic reproduction numbers. (a) corresponds to a basic reproduction number $R_0 = 0.23$, (b) corresponds to $R_0 = 0.60$, (c) corre-sponds to $R_0 = 4.51 > 1$, and(d) corresponds to $R_0 = 20.67 > 1$. The model adjusts the value of R only by varying x_0, with other parameters set as $\lambda = 1$, $d = 2$, $T_1 = 1$, and $\mu = 0.1$.

To verify the accuracy of the basic reproduction number R_0 and com-pare the infection proportions among nodes of different degrees, we plotted the curves of infection proportions over time for nodes of varying degrees in the network using the heterogeneous mean-field method. We then compared and analyzed the curves under different basic reproduction numbers, with the results presented in Figure 2. From the figure, we observe that when $R_0 < 1$ and is relatively small, the infection proportions for nodes of all degrees decrease directly to 0, as shown in (a). When $R_0 < 1$ but is close to 1, the infection proportions for some high-degree nodes initially increase slightly before gradually decreasing to 0, as shown in (b). The figure also demonstrates that as long as $R_0 < 1$ and sufficient time elapses, the infection proportions for all nodes in the network eventually drop to 0, which aligns with the definition of the basic reproduction number (Kermack et al., 1932). This indicates that the disease will gradually die out, and all individuals in the network will recover. When $R_0 > 1$, we observe that the infection proportions for nodes of different degrees stabilize at

different values after a period of time, and as R_0 increases, these values gradually approach 1. This confirms the existence of an endemic state in the network, where these stable values collectively form the endemic equilibrium point, indicating that the disease will persist in the network.

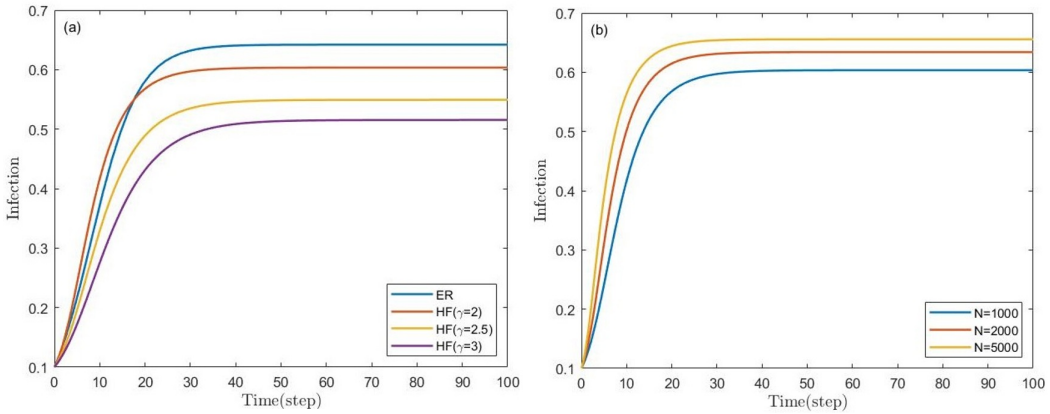

Figure 3: Analyzes the impact of network topology and scale on epidemic transmission. (a) shows the curves of epidemic infection proportions over time in ER random networks and scale-free net-works with different degree exponents. (b) displays the curves of epidemic infection proportions over time under different network scales. Other parameters set as $I_0 = 0.1$, $\lambda = 1$, $d = 2$, $T_1 = 1$, $x_0 = 0.01$ and $\mu = 0.1$.

This section investigates the impact of network topology and scale on disease transmission dynamics. we simulated the curves of infection proportions over time for epidemics in ER random networks with an average degree $\langle k \rangle = 7$ and scale-free networks with degree distributions following k^{-2}, $k^{-2.5}$, k^{-3} and scales. The results, illustrated in Figure 3, reveal several key observations. In scale-free networks, higher degree exponents lead to lower infection proportions, with these networks consistently exhibiting reduced infection rates compared to ER networks. Additionally, larger network sizes correlate with increased infection proportions under identical structural conditions. These findings can be attributed to the distinct transmission mechanisms in each network type. Scale-free networks with smaller degree exponents contain more high-degree nodes that facilitate early-stage transmission but also create "transmission dead ends" due to numerous low-degree nodes. Conversely, networks with larger degree exponents rely on medium-degree nodes with weaker transmission capacity, resulting in lower overall infection rates. ER networks demonstrate more uniform transmission patterns due to their homogeneous degree distribution, ultimately achieving higher infection proportions than scale-free networks. Furthermore, expanding the scale of scale-free networks elevates the absolute number of high-degree nodes, enhancing transmission efficiency while reducing "transmission dead ends" and consequently increasing infection proportions. Future work will test the method on real-world datasets to assess scalability.

5. Conclusion

This paper constructs a dynamical system for disease transmission in networks based on the SIS model, primarily investigating the impact of viral load on disease spread. The model is analytically

examined using heterogeneous mean-field theory. Numerical simulation reveal that a higher initial viral load leads to a larger infection proportion in the network, but the growth rate slows as the viral load increases; the infection proportion in scale-free networks decreases as the degree exponent increas-es, and due to the presence of "transmission dead ends" among low-degree nodes, the infection proportion is lower than that in degree-distribution-uniform ER random networks; in scale-free networks, an increase in the number of nodes raises the abso-lute number of high-degree nodes, leading to an increase in the infection proportion.

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