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Chung-Yuan Huang, Yu-Shiuan Tsai, Chuen-Tsai Sun, Ji-Lung Hsieh and Chia-Ying Cheng

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Influences of Resource Limitations and Transmission Costs on Epidemic Simulations and Critical Thresholds in Scale-Free Networks

Chung-Yuan Huang
Department of Computer Science and Information Engineering,
Research Center for Emerging Viral Infections,
Chang Gung University,
259 Wen Hwa 1st Road,
Taoyuan 333, Taiwan, Republic of China
gscott@mail.cgu.edu.tw

Yu-Shiuan Tsai
Chuen-Tsai Sun
Ji-Lung Hsieh
Chia-Ying Cheng
Department of Computer Science,
National Chiao Tung University,
1001 Ta Hsueh Road,
Hsinchu 300, Taiwan, Republic of China

Critical thresholds represent one of the most important diffusion indicators of epidemic outbreaks. However, we believe that recent studies have overemphasized ways that the power-law connectivity distribution features of social networks affect epidemic dynamics and critical thresholds. As a result, two important factors have been overlooked: resource limitations and transmission costs associated with social interactions and daily contact. Here we present our results from the simultaneous application of mean-field theory and an agent-based network simulation approach for analyzing the effects of resources and costs on epidemic dynamics and critical thresholds. Our main findings are: (a) a significant critical threshold does exist when resources and costs are taken into consideration, and it has a lower bound whenever contagion events occur in scale-free networks; (b) when transmission costs increase or individual resources decrease, critical contagion thresholds in scale-free networks grow linearly and steady density curves shrink linearly; (c) regardless of whether the resources of individuals obey delta, uniform, or normal distributions, they have the same critical thresholds and epidemic dynamics as long as the average value of usable resources remains the same across different scale-free networks; and (d) the spread of epidemics in scale-free networks remains controllable as long as resources are properly restricted and intervention strategy investments are significantly increased.

Keywords: critical thresholds, computer viruses, contagious diseases, effective spreading rate, complex networks, homogeneous networks, heterogeneous networks

1. Introduction

The presence or absence of a critical threshold as an epidemic spreads through a social network is a central issue currently being addressed by researchers from a range of

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disciplines [1–13]. Pastor-Satorras and Vespignani [5–9] argue that Internet-based computer viruses and contagious diseases in social networks do not have critical thresholds, while others have consistently concluded that, regardless of transmission capability, all contagious diseases have high probabilities of stable spreading and survival in scale-free networks [1–4, 10–14]. A frequently cited example is scale-free networks associated with human sexual contact, which are being analyzed as capable yet vulnerable platforms for the spread of HIV/AIDS.

An important fact remains: new contagious diseases and computer viruses are constantly emerging in different parts of the world, but the majority die almost immediately and an extremely small number survive to achieve epidemic status. In addition to contradicting previous conclusions [1–14], this observation serves as motivation to focus on limitations in daily interaction/communication processes among individuals/computers rather than the topological features of complex networks—the focus of many network-oriented epidemic studies published in the past decade [12, 15, 16]. We believe that two important factors associated with social interactions and computer communications have not been adequately addressed: *resource limitations* and *transmission costs*. While acknowledging the importance of Pastor-Satorras and Vespignani's work on the topological power-law features of scale-free networks (the inspiration for numerous studies on critical thresholds and immunization strategies), we believe a closer inspection of their mathematical analyses and numerical simulations reveals incorrect assumptions that daily interaction/communication processes are *cost-free*, and that the impacts of resource limitations and transmission costs are minimal. Such assumptions are beneficial in terms of mathematical equations and hypotheses, and therefore suitable for studying Internet viruses spread via email messages with large numbers of recipient addresses. However, they are unrealistic and inaccurate when applied to contagious diseases and innovative concepts spread via social interactions and daily human contacts.

Resources consumed by individuals during the process of spreading a contagious disease have five properties: (a) they are visible (e.g., seminal fluid, physical power) or invisible (e.g., time, energy, communication bandwidth); (b) individual resources are finite (e.g., time and energy spent on social interactions per day are finite and follow a normal distribution) and can be temporarily exhausted (e.g., time elapses during interactions with other individuals, daily sexual contacts are limited due to limited consumptive energy); (c) the use of one type of resource entails the consumption of smaller quantities of other types of resources, thereby reducing the total available resource amount (e.g., HIV-positive individuals spend both time and energy during sexual contacts); (d) individual resources can recover or regenerate after a period of time (e.g., energy levels are revived by sleeping); and (e) resources are nonreproducible. Contagious carriers who spend resources on specific recipients cannot reuse the same resources on other recipients; conversely, recipients cannot reuse resources spent on individual carriers.

Adhering to five resource/cost properties, we simultaneously applied mean-field theory and an agent-based network simulation approach to analyze the influences of resources and costs on epidemic dynamics and critical thresholds in scale-free networks. Our results indicate that (a) a significant critical threshold does exist when resources and costs are taken into consideration, and the threshold has a lower bound whenever contagion events occur in scale-free networks; (b) when transmission costs increase or individual resources decrease, critical contagion thresholds in scale-free networks grow linearly and steady density curves shrink linearly; (c) regardless of whether the resources of individuals obey delta, uniform, or normal distributions, they have the same critical thresholds and epidemic dynamics as long as the average value of usable resources remains the same across different scale-free networks; and (d) the spread of epidemics in scale-free networks remains controllable as long as resources are properly restricted and intervention strategy investments are significantly increased. These conclusions can assist epidemiologists, public health professionals, computer scientists, and marketing experts in their efforts to predict epidemic dynamics and critical thresholds and to develop intervention, immunization, and marketing strategies.

2. Complex Networks Applied to Epidemic Simulation Models

Complex networks are commonly used to represent structures for groups of individuals who exhibit interaction or relationship patterns [11–12, 17–19]. As shown in Figure 1 and Table 1, complex networks can be categorized as small-world, scale-free, or random according to basic statistical properties such as (a) local clustering, (b) the small-world phenomenon, and (c) the power-law connectivity distribution. They are popular among researchers who construct computational simulations of virtual societies, contagious diseases, Internet viruses, and the spread of cultural beliefs and influences—all of which are affected by transmission routes.

Generating a Watts and Strogatz [12] small-world network begins with an n-dimension ordered network (with periodic boundary conditions) in which each node is connected to a z quantity of neighbors, usually $z \geq 2n$ (Figure 2(a)) [11, 19]. Each link is randomly rewired to a new node with probability p (Figure 2(b)). Under adverse circumstances, this construction method can break the original ordered network into several isolated subgraphs (Figure 2(d)). Newman and Watts [20] introduced a variation of the original construction method that emphasizes the insertion of long-range shortcuts instead of rewiring links. In their version, two previously unconnected nodes are randomly selected and connected via a newly added link,

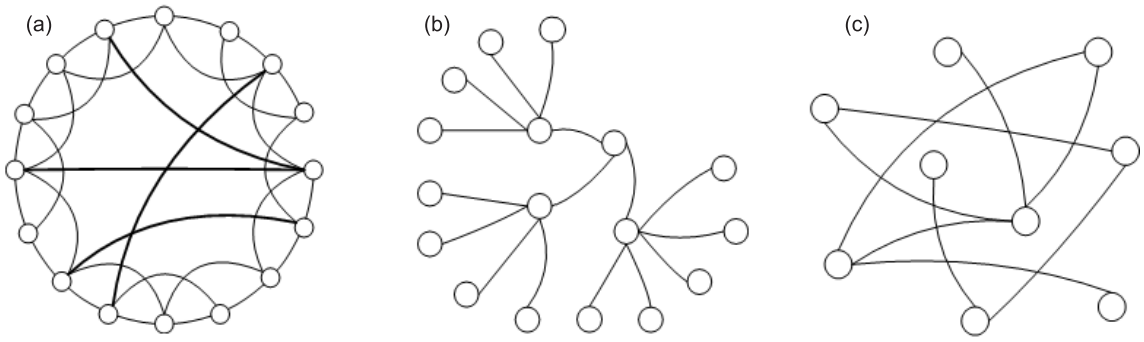

Figure 1. Three types of complex networks. (a) Small-World Network (SWN). (b) Scale-Free Network (SFN). (c) Random Network (RN).

Table 1. Two Complex Network Categories

Category	Network Type	Model	Clustering Coefficient	Degree of Separation	Connectivity Distribution
Homogeneous networks	Small-world	Watts and Strogatz [12]	high	low	normal
	Random	Erdös and Renyi [18]	very low	low	normal
Heterogeneous network	Scale-free	Barabási and Albert [17]	very low	low	power-law

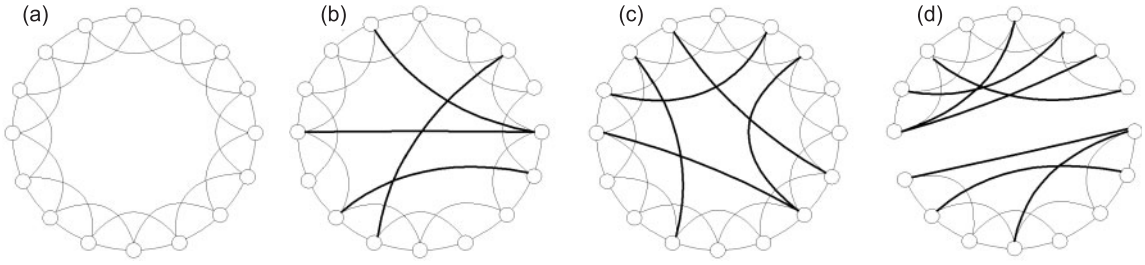

Figure 2. (a) One-dimensional ordered network with each node connected to four adjacent nodes. (b) Watts and Strogatz's [12] small-world network with four rewired shortcuts. (c) Newman and Watts' [20] improved small-world network with five additional shortcuts. (d) Example of a broken network in Watts and Strogatz's [12] small-world network.

with users determining the number of links to be added (Figure 2(c)). Newman and Watts' small-world network thus avoids the problem of network breakage while preserving the positive characteristic of connecting each node in an *n*-dimensional ordered network with 2*n* neighboring nodes. Since both the original and new versions [19] exhibit small-world and local clustering properties, they are considered similar to human daily-contact networks.

Generating a Barabási and Albert [17] scale-free network begins with a small number of nodes designated as z_0 [19]. During each iteration, a new node is introduced and connected to $z \leq z_0$ pre-existing nodes according to a probability based on each node's vertex degree. New nodes are preferentially attached to existing nodes that have large numbers of connections. This type of network exhibits small-world and power-law connectivity distribution properties, implying the existence of a small number of nodes with very large vertex degrees—similar to World Wide Web hyperlinks and human sexual contact networks.

Erdös and Renyi's [18] random networks are generated by adding links between pairs of randomly chosen nodes with certain probabilities [18, 19]. They are capable of exhibiting small-world properties if sufficient numbers of links are added, but with little or no local clustering—an unusual situation in the real world.

Communities, cities, and countries—even the planet—can be defined as individual complex networks consisting of large-scale nodes and links. Each node represents one individual with status-determining attributes (often referred to as node-related local information) such as epidemiological progress, contagiousness, or immunization [13, 21]. Connections between individuals are referred to as links, with different links representing different interpersonal relationships [8]. In HIV/AIDS epidemic simula-

tions they represent sexual relationships, while in Severe acute respiratory syndrome (SARS) epidemic simulations they represent close physical proximity [22,23]. The states of all network nodes change simultaneously during each time step. The state of an individual node is determined by its original state, its linked neighbor's state, and a set of interaction rules.

3. Epidemic Dynamics in Complex Networks

In standard epidemiological models, all individuals (nodes) in a population (complex network) can be roughly classified into a limited number of states, including *Susceptible* (an individual is vulnerable to infection but has not yet been infected), *Infected* (an individual can infect others), and *Removed* (an individual has recovered, died, or otherwise ceased to pose any further threat). Epidemiologists use combinations of these states to represent orders of transition between different epidemiological phases, giving names such as 'SIR' and 'SIS' to their models.

Past epidemiological research has focused on the transmission dynamics and spreading situations of biologically contagious diseases. A growing number of recent studies are focusing on nonbiological and intangible concepts such as computer viruses, cultural influences, rumors, ideas, and beliefs that exist in social networks and on the Internet. In these kinds of spreading scenarios, cultural influences move ideas and beliefs between transmitters and receivers, eventually making the majority of receivers behave in the same manner as the transmitters [21, 24, 25].

In Sections 3.1 and 3.2 we will review research on epidemic dynamics and critical thresholds in homogeneous networks (e.g., Erdös and Renyi's [18] random and Watts and Strogatz's [12] small-world) and heterogeneous networks (e.g., Barabási and Albert's [17] scale-free). Our epidemic model will be analyzed in Section 3.3.

3.1 Epidemic Dynamics and Critical Thresholds in Homogeneous Networks

When simulating epidemic dynamics in complex networks, epidemiologists usually assume that nodes in complex networks run stochastically through an SIS cycle (*S*usceptible $\rightarrow$ *I*nfected $\rightarrow$ *S*usceptible), which does not take into account the possibility of an individual's removal due to death or acquired immunization. The SIS epidemiological model has been adopted widely to study contagious diseases leading to endemic states with a stationary average density of infected individuals. Note that similar analytical results derived from the SIS epidemiological model can be readily extended to the SIR and SIRS models for many contagious diseases [8]. During each time step, each susceptible node is subject to a ν probability contagion rate if it is connected to one or more infected nodes. Infected nodes recover at a probability rate δ, and once again become susceptible. An effective spreading rate λ is defined as $\lambda = \nu/\delta$. Recovery rate δ can be assigned a value of 1, since it only affects the time scale of contagious disease propagation [9].

Pastor-Satorras and Vespignani [5] define $\rho(t)$ as the density of infected nodes at time step t. When time step t becomes infinitely large, ρ can be represented as a steady-state density of infected nodes. Using these definitions, they applied mean-field theory to the SIS epidemiological model, and used Anderson and May's [26] *homogeneous mixing hypothesis* according to the topological features of homogeneous networks to obtain (a) a steady-state density ρ of infected nodes during long time periods (Equation (1)), and (b) the critical threshold λ_c (Equation (2)):

$$\rho = \begin{cases} 0 & \lambda < \lambda_c \\ \dfrac{\lambda - \lambda_c}{\lambda} & \lambda \geq \lambda_c \end{cases} \tag{1}$$

$$\lambda_c = \frac{1}{\langle k \rangle} \tag{2}$$

where $\langle k \rangle = \sum_k k p_k$ is the average vertex degree of the network and p_k the fraction of nodes that have vertex degree k in the network. According to Equations (1) and (2), a positive and nonzero critical threshold λ_c exists in a homogeneous network based on the SIS epidemiological model. A contagion spreads and becomes epidemic if the effective spreading rate exceeds the critical threshold ($\lambda \geq \lambda_c$); otherwise, the contagion dies out. As shown in Figure 3, the SIS epidemiological model separates an infected state from a healthy state at critical threshold λ_c. In summary, the primary prediction in an SIS epidemiological model in a homogeneous network is the presence of a positive critical threshold, proportional to the inverse of the average number of neighbors of each node, below which epidemics die and endemic states are impossible.

3.2 Epidemic Dynamics and Critical Thresholds in Heterogeneous Networks

Pastor-Satorras and Vespignani [6] relaxed their homogeneity assumption and obtained a critical threshold λ_c in a scale-free network (Equation (3)). Their results indicate that, in scale-free networks with a connectivity exponent of $2 < \gamma \leq 3$ and for which $\langle k^2 \rangle = \sum_k k^2 p_k \rightarrow \infty$ is the limit of a network of infinite size, the critical threshold λ_c is very close to 0 ($\lambda_c \rightarrow 0$),

$$\lambda_c = \frac{\langle k \rangle}{\langle k^2 \rangle}. \tag{3}$$

Asserting that the steady-state density ρ of infected nodes for the SIS epidemiological model in a Barabási and Albert [17] scale-free network can be expressed as a function of the effective spreading rate λ, Pastor-Satorras

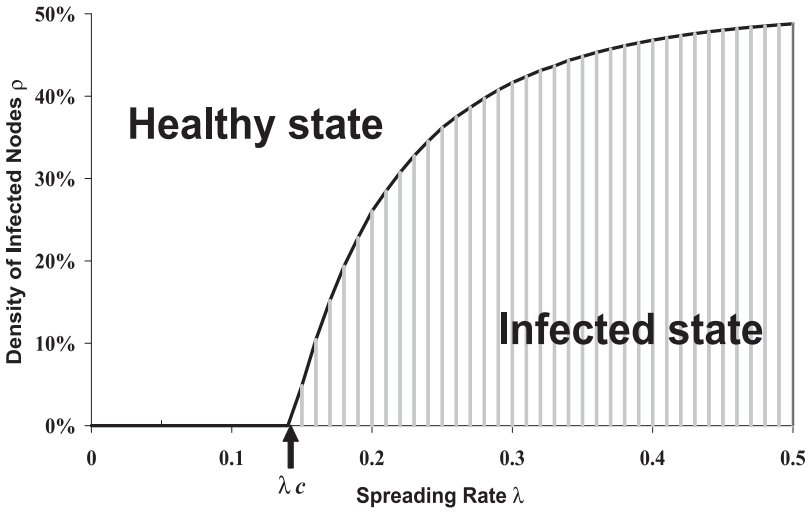

Figure 3. Phase transition diagram for epidemic simulations in homogeneous networks.

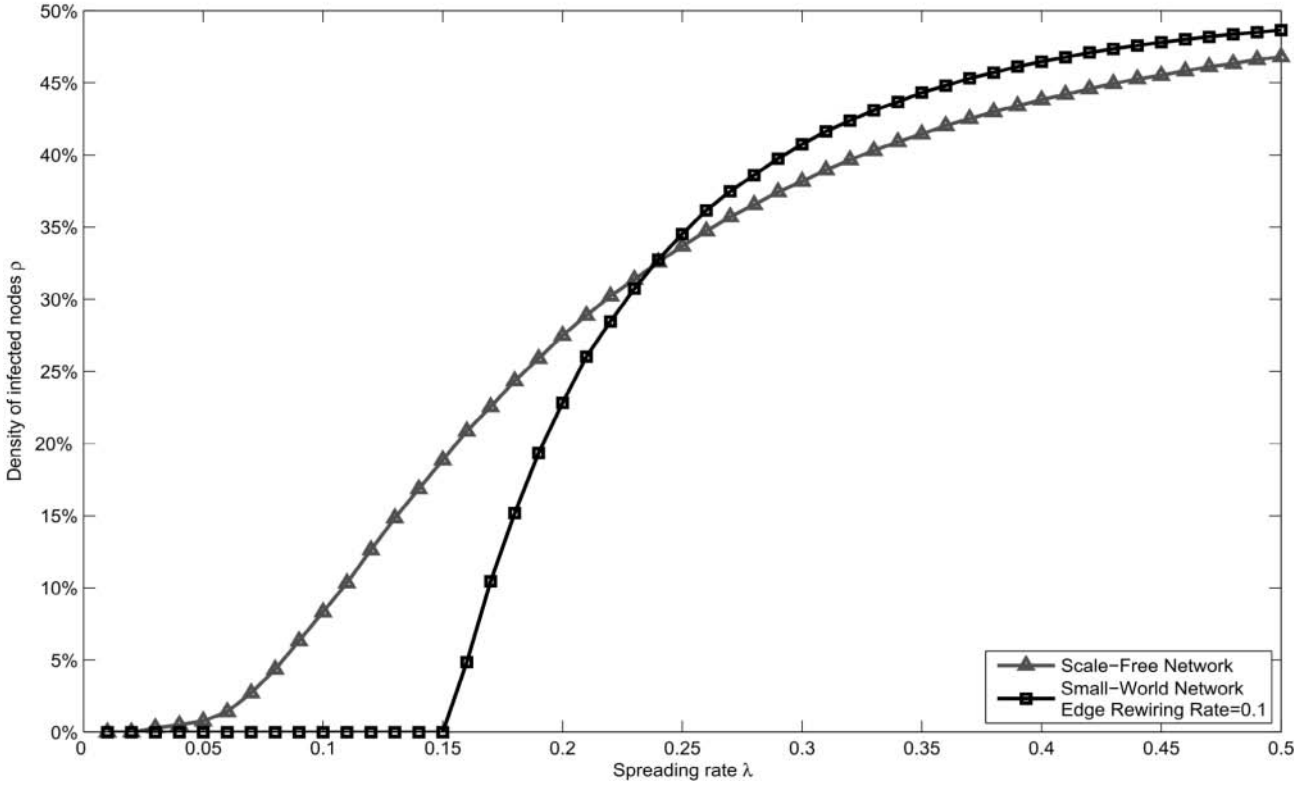

Figure 4. Steady-state density ρ of infected nodes as a function of effective spreading rate λ.

and Vespignani compare it to a theoretical prediction for a homogeneous network. As shown in Figure 4, the steady-state density ρ of infected nodes in a Barabási and Albert scale-free network reaches 0 in a continuous and smooth manner when the effective spreading rate λ decreases, indicating the absence of any critical threshold ($\lambda_c = 0$). In such networks, epidemics can be stably spread and eventually reach a steady state as long as $\lambda > 0$. This explains

why scale-free networks are fragile in epidemiological spreading situations. The Internet, social networks, and the web of human sexual contacts all appear to be scale-free, therefore computer viruses, innovative concepts, and biologically contagious diseases can be stably spread even when initial contagion cases occur in small and limited areas.

For finite-size scale-free networks, Pastor-Satorras and Vespignani [8] introduced the concept of maximum connectivity k_c (dependent on the number of nodes), which has the effects of restoring a boundary in connectivity fluctuations and inducing an effective nonzero critical threshold. According to the definition of maximum connectivity k_c, $\langle k^2 \rangle$ in Equation (3) clearly has a finite value in a finite-size scale-free network. However, in this situation the critical threshold vanishes as network size increases.

These conclusions explain the epidemic outbreak mechanisms of some biologically contagious diseases and computer viruses. According to traditional epidemiological theory, large-scale pandemics only occur when the effective spreading rate exceeds a specific critical threshold. However, Pastor-Satorras and Vespignani claim that contagious diseases can proliferate in scale-free networks regardless of their effective spreading rates. This idea represents a major threat to public health and computer data.

3.3 Epidemic Model Analysis

Our epidemic model is based on the mathematical model proposed by Pastor-Satorras and Vespignani [5]:

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda k \left[1 - \rho_k(t)\right] \theta \left[\left\{\rho_k(t)\right\}\right], \quad (4)$$

where $\rho_k(t) \ll 1$ (neglecting the higher order) is the relative density of infected nodes that have k connections, λ is the infection rate, and $\theta \left[\{\rho_k(t)\}\right]$ is the probability that any given individual will link to an infected individual (with θ assumed to be a function of the partial densities of infected individuals $\left\{\rho_k(t)\right\}$). Equation (4) states that, during each time step, infected individuals who have k connections will recover, yet still infect other individuals according to four parameters: infection rate, connectivity, number of healthy individuals, and probability $\theta \left[\{\rho_k(t)\}\right]$. Pastor-Satorras and Vespignani observed that $\left\{\rho_k(t)\right\}$ is a function of λ in a steady state, and therefore θ becomes a function of λ such that $\theta(\lambda) = \frac{1}{\langle k \rangle} \sum_k k P(k) \rho_k$, where $P(k)$ is the connectivity distribution. When considering the stationary condition $d\rho_k(t)/dt = 0$ within a scale-free network in which $P(k) = 2m^2 k^{-3}$ with minimum degree m, the critical threshold has the property $\lambda_c = \langle k \rangle / \langle k^2 \rangle \to 0$ as $k \to \infty$. Accordingly, for infinite-size networks, either no epidemic threshold exists or the threshold approaches 0.

However, Pastor-Satorras and Vespignani's model (Equation (4)) neglects individual access to energy, time, and other finite resources. To incorporate these costs, we modify their model to

$$\frac{d\rho_k(t)}{dt} = -\rho_k(t) + \lambda S_k \left[1 - \rho_k(t)\right] \theta \left[\left\{\rho_k(t)\right\}\right],$$

$$\text{where } S_k = \min\left(\frac{R}{c}, k\right). \quad (5)$$

The term $S_k = \min(\frac{R}{c}, k)$ (with R representing average resources and c denoting transmission costs) states that each infection is spread proportional to the minimum value of each active node's available resources (R/c) and number of links. We assume the stationary condition $d\rho_k(t)/dt = 0$ and obtain

$$\rho_k = \frac{\lambda S_k \theta(\lambda)}{1 + \lambda S_k \theta(\lambda)} \quad (6)$$

where ρ_k is the steady state of $\rho_k(t)$. Substituting $\theta(\lambda)$ into Equation (6) we get

$$\theta = \frac{1}{\langle k \rangle} \sum_k k P(k) \frac{\lambda S_k \theta}{1 + \lambda S_k \theta}. \quad (7)$$

Note that the right-hand side of Equation (7) is concave at about θ (i.e., the second derivative is no larger than zero), and that $\theta = 0$ is considered a trivial solution. Since it is possible for θ to have a nonsingular solution, we derive the inequality

$$\frac{d}{d\theta} \left(\frac{1}{\langle k \rangle} \sum_k k P(k) \frac{\lambda S_k \theta}{1 + \lambda S_k \theta} \right) \Bigg|_{\theta=0} \geq 1. \quad (8)$$

By differentiating Equation (8) and replacing θ with 0 we get

$$\frac{1}{\langle k \rangle} \sum_k k P(k) \lambda S_k \geq 1 \quad \text{or} \quad \lambda \leq \frac{\langle k \rangle}{\sum_k k P(k) S_k}. \quad (9)$$

Accordingly, the critical threshold λ_c is defined as the maximal λ, resulting in

$$\lambda_c = \frac{\langle k \rangle}{\sum_k k P(k) S_k}. \quad (10)$$

Since $S_k = \min(R/c, k)$, the denominator can be divided into two parts, thereby obtaining

$$\lambda_c = \frac{\langle k \rangle}{\sum_{k \leq \frac{R}{c}} k^2 P(k) + \sum_{k > \frac{R}{c}} \frac{R}{c} k P(k)}. \quad (11)$$

According to the first term in the denominator of Equation (11), the variable k is smaller than R/c, so substituting R/c for k makes the first term larger. Similarly, according to the second term the summation is smaller than the entire scope of k, so substituting k for the entire scope also makes the second term larger. As a result,

$$\lambda_c \geq \frac{\langle k \rangle}{\sum\limits_{k \leq \frac{R}{c}} \left(\frac{R}{c}\right)^2 P(k) + \sum\limits_{k} \frac{R}{c} k P(k)}. \qquad (12)$$

By the same method, making another substitution on the left-hand side of the denominator of Equation (12) results in

$$\lambda_c \geq \frac{\langle k \rangle}{\sum\limits_{k} \left(\frac{R}{c}\right)^2 P(k) + \sum\limits_{k} \frac{R}{c} k P(k)}. \qquad (13)$$

Since $\sum\limits_{k} P(k) = 1$, we arrive at

$$\lambda_c \geq \frac{\langle k \rangle}{\left(\frac{R}{c}\right)^2 + \frac{R}{c}\langle k \rangle} = \frac{1}{\left(\frac{\left(\frac{R}{c}\right)^2}{\langle k \rangle}\right) + \frac{R}{c}} \qquad (14)$$

and find that as $\langle k \rangle \rightarrow \infty$, λ_c is at a minimum equal to c/R.

Since λ_c represents the critical threshold at which a contagious disease exceeds control and becomes epidemic, managing its value is the primary concern of epidemiologists and public health officials. In summary, the lower bound of the critical threshold λ_c becomes smaller if the transmission cost c decreases or the average resource R increases. Accordingly, an individual's available resources expand when c/R decreases, thereby increasing the individual's ability to contact almost all other individuals in his or her personal social network. This result supports what we know about immunization: appropriately restricting one's resources increases the critical threshold. Neglecting resources makes R infinitely large, meaning that resources are inexhaustible and that the critical threshold λ_c will continue to approach 0 as long as the average number of links is sufficiently large. The model thus becomes identical to Pastor-Satorras and Vespignani's model in Equation (4), in which a disease has the potential to reach epidemic proportions regardless of the small number of infected nodes.

Most contagion events involving biological diseases and intangible concepts such as cultural influences, rumors, ideas, and beliefs need sufficient resources (e.g., time, energy, or money) for social interactions and daily contacts to occur. Accordingly, controlling the c/R ratio can increase the threshold λ_c and decrease the steady-state density ρ. In contrast, computer viruses can spread very quickly via the Internet because they can be transmitted simultaneously to many sites [5–9]. While the spreading time is short, affected areas can be very large, with disastrous results in terms of lost data, work time, and money [15]. One suggested strategy for controlling computer virus diffusions and network attacks is placing restrictions on upload/download capacities from remote service servers (e.g., a maximum of one gigabyte per day)—in other words, limiting resources so as to increase the critical threshold. A related strategy is charging upload/download fees when users want to exceed daily limitations—that is, raising transmission costs to achieve the same end result.

4. Epidemic Simulations and Results

For simulation runs we constructed a complex network $G(N, E)$ with $|N|$ nodes (representing individuals in social networks) and $|E|$ links (indicating social interactions and daily contacts between two individuals, with those having direct connections labeled *neighbors*). Only *InitialStatus_I* nodes were given *Infected* status at the beginning of each simulation run; all others were designated as *Susceptible*. All epidemic dynamics and critical thresholds discussed in this paper represent average values for 30 runs (95% confidence interval $\pm$ 17.89%). A list of experimental parameters is presented in Table 2.

Also, at the beginning of each time step, usable resources for each node v_i were reset to $R(v_i)$, meaning that all individuals renewed and/or received supplemental resources. In our later experiments, the statistical distribution of individual resources could be delta (fixed value $r_{Constant}$), uniform, normal, or power-law, as long as the average $\langle r \rangle$ value of individual resources satisfied

$$\langle r \rangle = \sum_{i=1}^{N} R(v_i)/N = r_{Constant}. \qquad (15)$$

Nodes randomly interacted with several neighbors during each time step, with node resources and costs being consumed during each interaction. Each node v_i randomly selected from all $Neighbor(v_i)$ nodes interacted with a single neighboring node v_j. Following each interaction and regardless of the result, nodes v_i and v_j had transmission costs $c(v_i)$, $c(v_j)$ where $0 \leq c(v_i) \leq R(v_i)$ and $0 \leq c(v_j) \leq R(v_j)$, deducted from their resources. If $R(v_i) < c(v_i)$ after an interaction, node v_i could not interact with other neighbors because all of its resources were used up. Otherwise the interaction process was repeated, with nodes randomly selecting other neighboring nodes until their resources were exhausted.

At each time step, the epidemiological status of each node was determined by a combination of behavioral rules, original epidemiological status, neighbors' epidemiological status, contagion rate ν, and recovery rate δ. Assume that an infected and contagious node v_a is adjacent to a susceptible and contagion-prone node v_b. When the two nodes come into contact, a contagion rate ν determines whether or not v_b is infected by v_a. At the same

Table 2. Epidemic simulation parameters.

Attribute	Type	Description
ν	Real	Contagion rate. Default range from 0.01 to 0.5 in 0.01 steps when δ = 1.
δ	Real	Reset rate (also called 'recovery rate' in SIS epidemiological model). Default value = 1.0.
λ	Real	Effective spreading rate $\lambda =$ contagion rate ν/recovery rate δ.
Network Type	Symbol	According to *Network Type*, a complex network for the SIS epidemic model can be built in the same manner as Watts and Strogatz's [12] small-world homogeneous network and Barabási and Albert's [17] scale-free network. If *Network Type* = SWN, a small-world network is built; if *Network Type* = SFN, a scale-free network is built. Default value = SFN.
N	Set	Node set of a complex network. $\|N\|$ represents the total number of nodes in a complex network built for a SIS epidemiological model.
E	Set	Link set of a complex network. $\|E\|$ represents the total number of links in a complex network built for a SIS epidemiological model.
Rewiring Rate	Real	Specific parameter for Watts and Strogatz's small-world network. Generating a WS-SWN begins with a one-dimensional regular network with periodic boundary conditions. Each link is randomly rewired to a new node with a *Rewiring Rate* probability. Default value = 0.01.
Time Step Limit	Integer	Total number of time steps during each simulation. Default value = 300.
c	Integer	Transmission costs per interaction event. Default value = 1.
R	Integer	Daily individual economic resources. Default value = 16.
InitialStatus_I	Integer	Initial number of infected nodes at the beginning of the epidemic simulation. Default value = 10% × $\|N\|$.

time, an infected node v_a can recover at a recovery rate δ and once again become susceptible. The effective spreading rate λ is defined as ν/δ. Generally, the recovery rate $\delta = 1$ and the effective spreading rate $\lambda = \nu$. We defined $\rho(t)$ as the density of infected nodes present at time step t; when time step t becomes infinitely large, ρ can be presented as a steady infected node density.

As shown by the curve marked with triangles in Figure 5, the steady density of the SIS epidemiological model based on a scale-free network approached 0 in a continuous and smooth manner as the effective spreading rate was decreased, thereby indicating the lack of a critical threshold in scale-free networks in the absence of transmission costs. The curve marked with squares in the same figure shows that epidemics do have critical thresholds in small-world networks. In addition to its similarity to the curve marked with squares, the curve marked with circles shows that a nonzero, positive, and significant critical threshold exists when epidemics are spread throughout a scale-free network if individual resources and transmission costs are taken into consideration.

A significant increase in critical threshold λ_c was observed as the overall amount of usable resources decreased. For example, in Figure 6 the critical threshold λ_c increased to 0.22 when the usable resources value was set at 8 units at the beginning of each time step. When the usable resources value was set at 40 units at the beginning of each time step, the shape of the density curve marked with squares was very close to that of the scale-free network without transmission costs (Figure 6, solid black line), and the critical threshold λ_c was reduced to 0.09. As shown in Figure 7, a linear correlation was observed between the critical threshold and the ratio of the cost of a single contagion event to total amount of an individual's resources (hereafter referred to as 'the ratio'). As shown in Figure 6, the density curve grew at a slower rate as the ratio increased—in other words, the ratio and density curve had a negative linear correlation when the effective spreading rate exceeded the critical threshold. The simulation results suggest that in scale-free networks, the critical thresholds of epidemics grow linearly and density curves shrink linearly when transmission costs increase or individual resources decrease. A comparison of results from our mathematical analyses and simulation experiments is shown in Figure 8.

The density curves marked with diamonds, crosses, and circles in Figure 9 respectively represent the effects of delta (fixed value = 16), uniform, and normal distributions of individual usable resources on the critical thresholds of epidemics spread in scale-free networks marked by limited individual resources and transmission costs. Data on their statistical distributions and parameters (average value and standard deviation in a normal distribution or number of values and range in a uniform distribution) are presented in Figure 10. The same critical threshold ($\approx$ 0.14) and near-overlapping density curves (i.e., no statistically significant differences, correlation coefficient $c \approx 0.9998$ and chi-square test, $P > 0.05$) were observed for all three scenarios when average individual usable resource values were equal. When those same resources were distributed in a power-law mode (i.e., the majority of individuals had very limited resources while a small number had large amounts) (Figure 11), and in the absence of correlations between the total amount of an individual's usable resources and vertex degree, the resulting dashed density curve grew more slowly than those for the other three distributions (chi-square test, $P < 0.01$), even when they all had the same critical threshold.

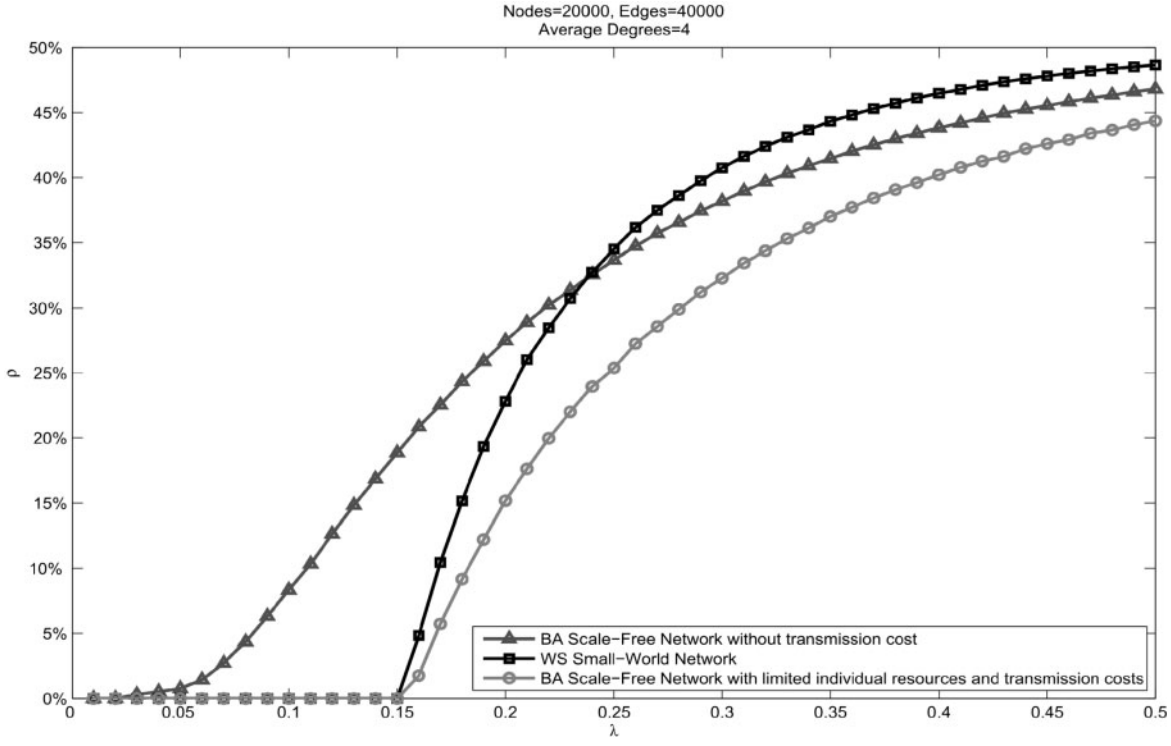

Figure 5. Relationship between effective spreading rate and steady density of the SIS epidemiological model in three types of complex network platforms: small-world, scale-free without transmission costs, and scale-free with limited individual resources and transmission costs.

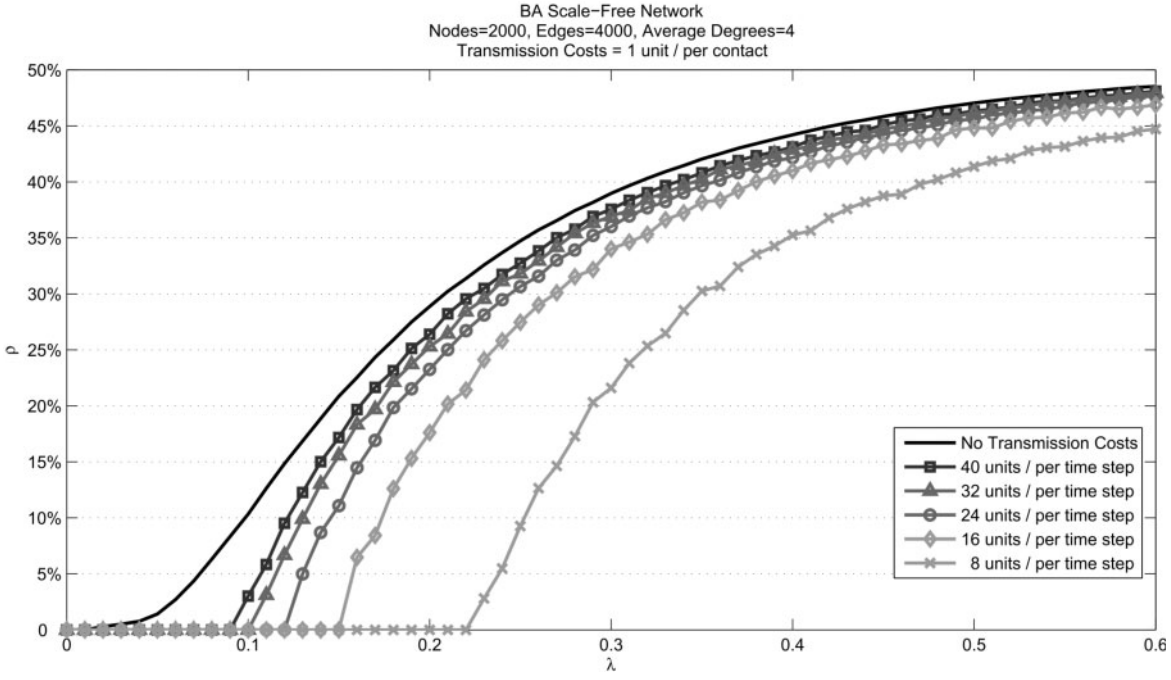

Figure 6. The amount of an individual's resources affects steady-state density curves and critical thresholds.

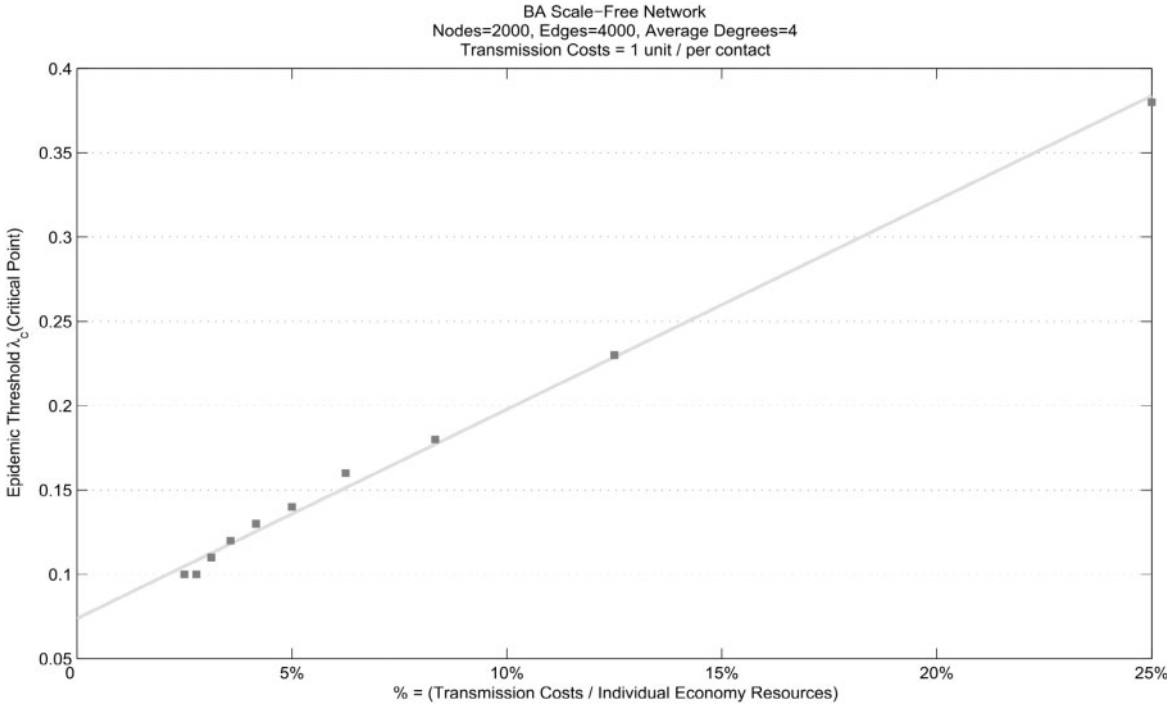

Figure 7. Linear relationship between the ratio of transmission costs to an individual's resources and critical threshold.

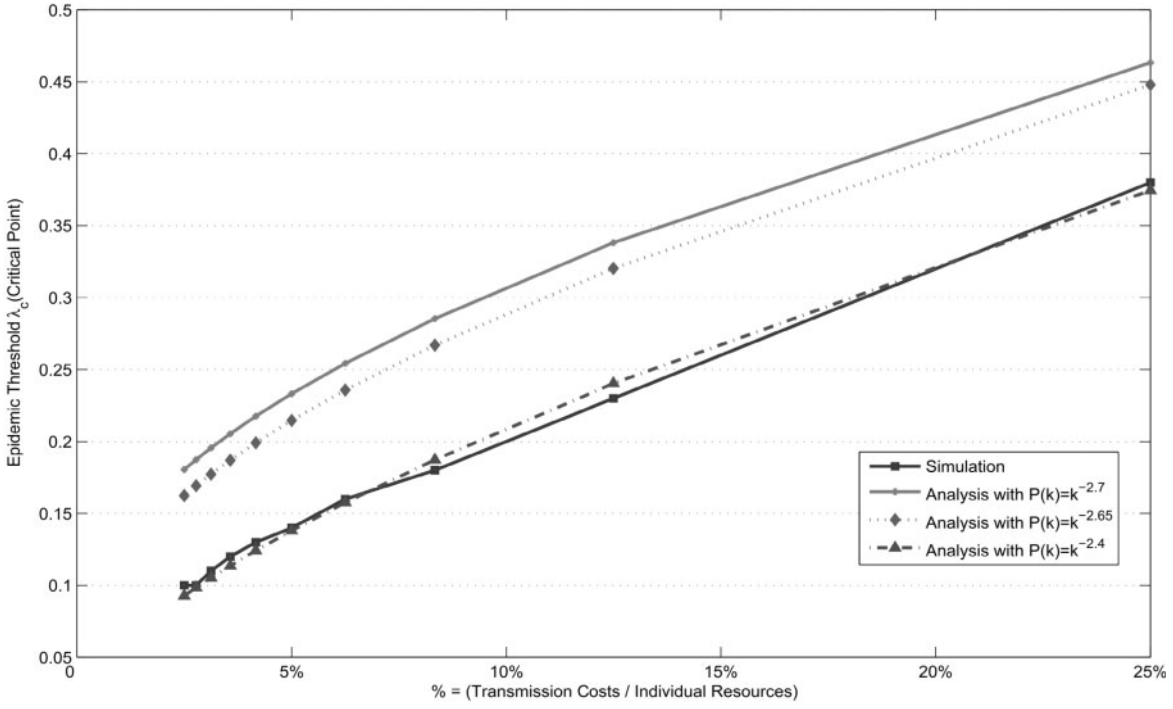

Figure 8. As a function of the *c/R* ratio (transmission costs/individual resources) in Barabási and Albert's [17] scale-free networks, the epidemic threshold λ_c is used to represent results from a simulation and three mathematical analyses.

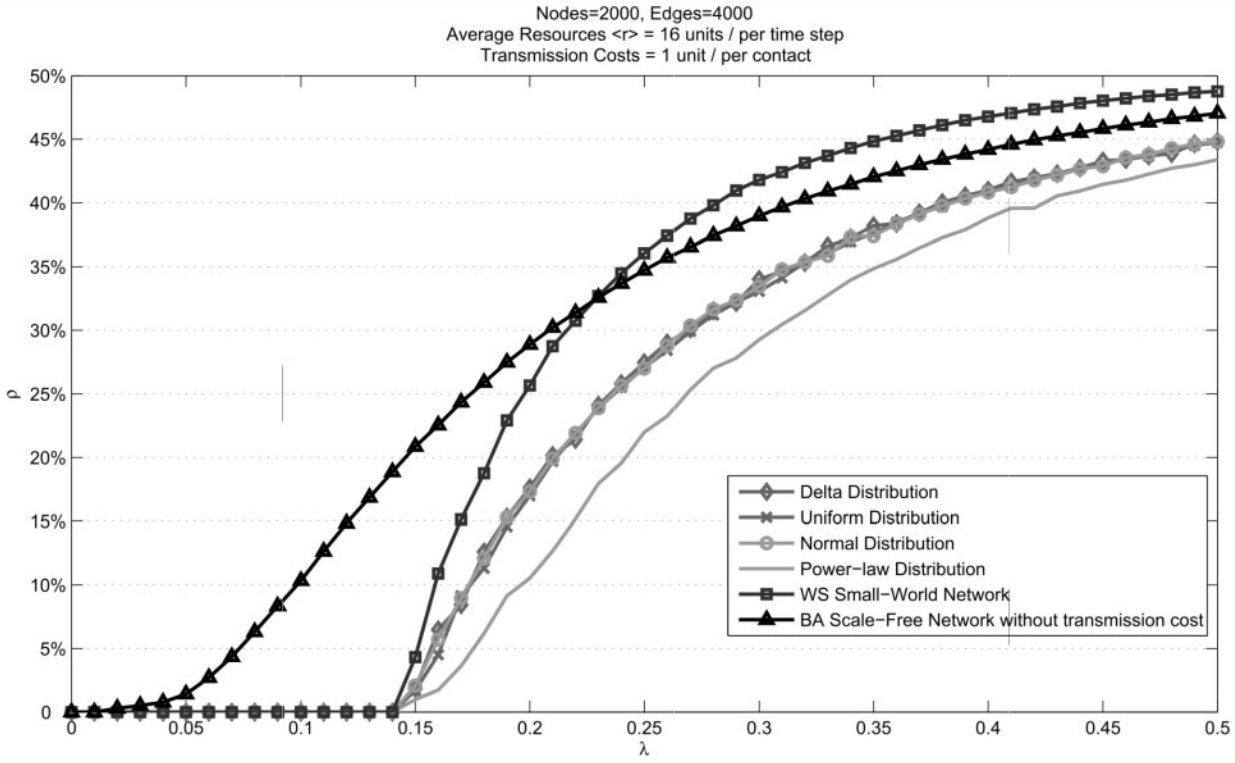

Figure 9. The effects of different statistical distribution types for individual resources on the steady-state density curves and critical thresholds of contagious diseases spread within scale-free networks.

We repeatedly achieved the same results as long as the average usable resource value remained unchanged (Figures 9 and 12 curves marked with diamonds, crosses, and circles). In addition, density curves and critical thresholds were almost identical (i.e., no statistically significant differences, correlation coefficient $c \approx 0.9998$ and chi-square test $P > 0.05$) across different distribution types, regardless of whether the resources were (a) distributed uniformly with a range of 2 or 3 (Figures 10 and 13) or (b) distributed normally with a standard deviation of 2 or 3 (Figures 10 and 13). From the significantly different groups of density curves shown in Figures 9 and 12 (curves marked with diamonds, crosses, and circles versus dashed curve), we conclude that as long as researchers ensure that usable resources do not obey a power-law distribution (Figures 11 and 14), then at the beginning of each time step they can assign usable resources for each individual as the fixed average value $\langle r \rangle$ (Equation (15)) of the statistical distribution derived from a real-world scenario, and do so without affecting simulation results.

Here we will use the 2002–2003 SARS outbreak as a discussion of a practical application of our simulation results. The SARS coronavirus is propagated over short distances and can survive in air for several hours to several days [27, 28]. During the outbreaks, the Singaporean government enacted a body temperature measurement policy, Hong Kong enforced policies such as hand washing and surgical mask usage, and the Taiwan government applied both [27]. These and all other policies can be divided into two categories. The first is aimed at decreasing propagation, with examples including home quarantines for anyone suspected of having contact with infected individuals for a period equal to twice the known SARS latency period [27, 28]. As part of this policy, public health workers take the body temperatures of large numbers of individuals entering public spaces. Anyone with a fever exceeding 37°C is detained and instructed to not come into contact with other individuals. The second type is aimed at increasing propagation costs for infected individuals. Examples include public appeals to wash hands, wear surgical masks, and maintain a high level of cleanliness. Vaccines and anti-virus medicines also belong to this category, as do requests for individuals to avoid public places as much as possible. It is generally believed that the speed with which both types of policies were put into place by the Hong Kong, Taiwan, and Singaporean governments were positive factors in the successful control of the SARS epidemic.

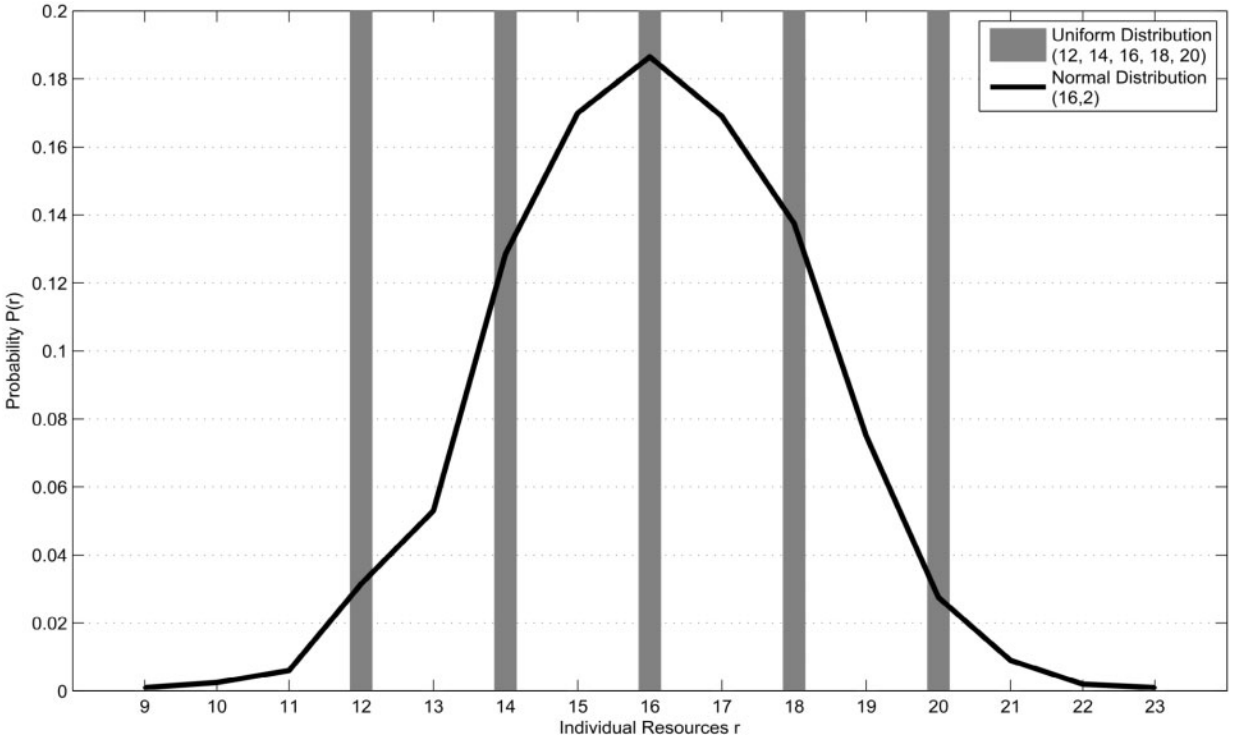

Figure 10. Uniform (n = 5, r = 2) and normal (standard deviation = 2) distributions of individual resources with an average $\langle r \rangle$ value of 16.

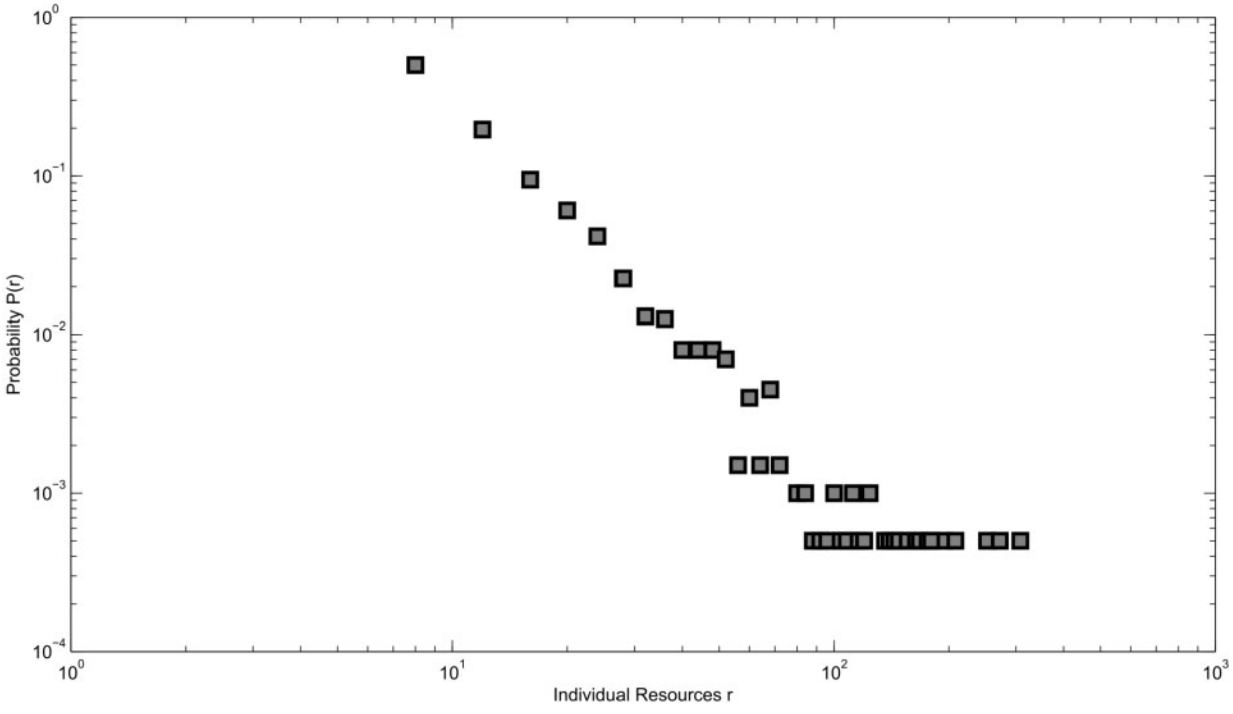

Figure 11. Individual resources in a power-law distribution.

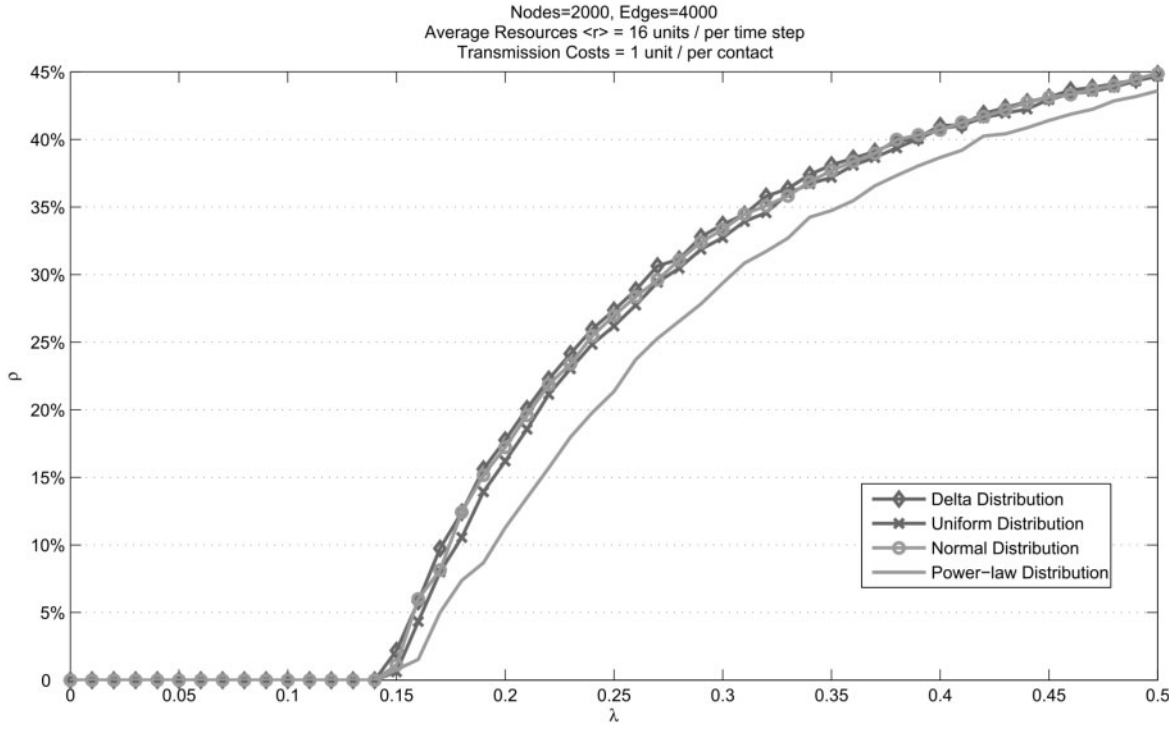

Figure 12. The effects of different statistical distribution types for individual resources on the steady-state density curves and critical thresholds of contagious diseases spread within scale-free networks.

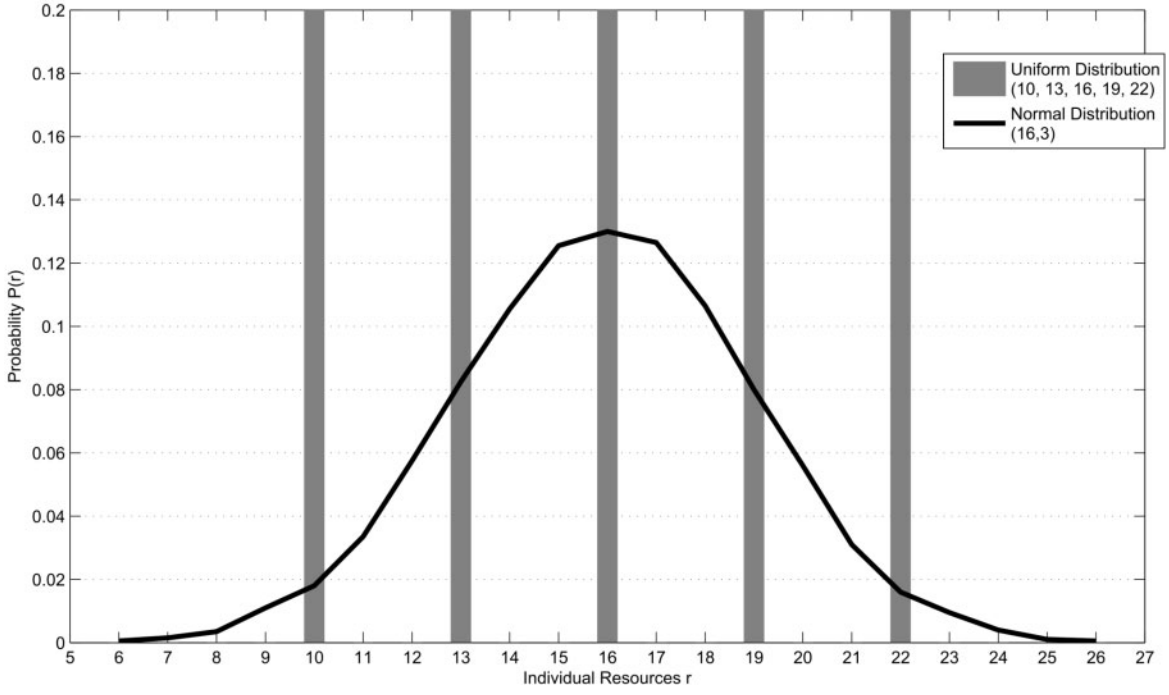

Figure 13. Uniform (n = 5, r = 3) and normal (standard deviation = 3) distributions of individual resources with an average $\langle r \rangle$ value of 16.

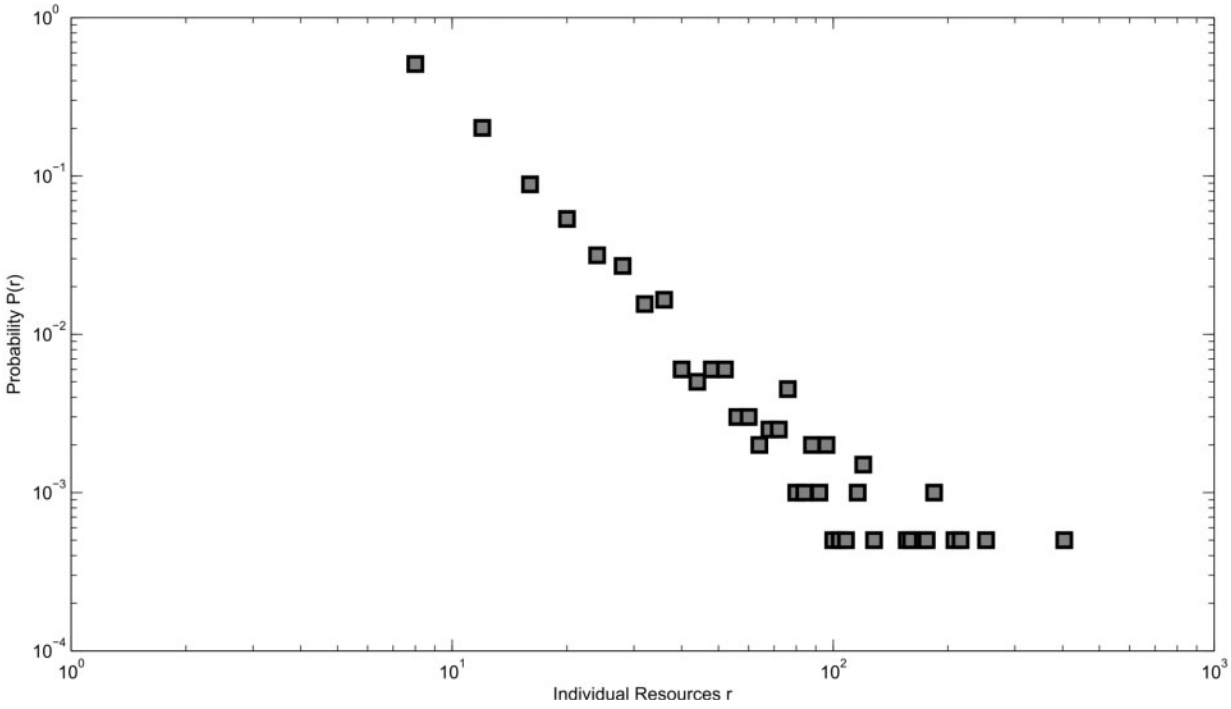

Figure 14. Individual resources in a power-law distribution.

5. Conclusion

Ever since Watts and Strogatz [12] proposed their small-world network model and Barabási and Albert [17] introduced their scale-free network model, epidemiologists, computer scientists, and marketing experts have used complex network theories and computer simulations to analyze the details of contagious diseases, computer viruses, and product diffusion. As part of their attempt to simplify their experiments, researchers have tended to overlook individual resources and transmission costs—both of which exert significant impacts on epidemic dynamics.

In this paper we used definitions for resources and transmission costs from the field of economics, proposed five characteristics of resources, and applied agent-based modeling and network-oriented simulation approaches to construct SIS epidemiological models to investigate how resources and transmission costs influence epidemic dynamics and thresholds in scale-free networks. Our mathematical analysis and simulation results indicate that, when resources and transmission costs are taken into consideration, a critical threshold does in fact exist when a contagion event occurs in a scale-free network.

According to results from our first set of experiments, individual resources, transmission costs, and average vertex degree are among factors exerting significant impacts on critical thresholds; node and link numbers were found to have little impact. Results from our second experimental set provide insight into how the ratio of the single contagion event costs to the total amount of an individual's resources affects density curves and critical thresholds. When transmission costs increase, or when the total amount of an individual's resources decrease, the critical threshold of a contagion event in a scale-free network grows and density is reduced at certain transmission rates. Results from our third set of experiments indicate that, regardless of whether resources obey a delta, uniform, or normal distribution, they all have the same density curve and critical threshold as long as the average resource value remains the same across different networks. This conclusion can make the process of constructing both simple and abstract computer models significantly less complex.

We suggest that our three main conclusions can help epidemiologists and public health professionals understand and analyze core questions of disease epidemics, predict epidemic dynamics and diffusion, and develop effective public health policies and immunization strategies. They may also have utility for computer scientists wanting to develop strategies against contagious viruses (and perhaps computer viruses) at various intrusion levels.

6. Acknowledgements

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Chung-Yuan Huang *is currently an Assistant Professor in the Department of Computer Science and Information Engineering and a member of the Research Center for Emerging Viral Infections at Chang Gung University, Taiwan.*

Yu-Shiuan Tsai *is currently PhD candidate in the Department of Computer Science, National Chiao Tung University.*

Chuen-Tsai Sun *is currently a Professor in the Department of Computer Science, National Chiao Tung University.*

Ji-Lung Hsieh *is currently a PhD candidate in Computer Science at National Chiao-Tung University, Taiwan.*

Chia-Ying Cheng *is currently a PhD candidate in Computer Science at National Chiao-Tung University, Taiwan.*