Integrated systemic inflammatory response syndrome epidemic model in scale-free networks*

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(Received 11 October 2010; revised manuscript received 30 March 2011)

Based on the scale-free network, an integrated systemic inflammatory response syndrome model with artificial immunity, a feedback mechanism, crowd density and the moving activities of an individual can be built. The effects of these factors on the spreading process are investigated through the model. The research results show that the artificial immunity can reduce the stable infection ratio and enhance the spreading threshold of the system. The feedback mechanism can only reduce the stable infection ratio of system, but cannot affect the spreading threshold of the system. The bigger the crowd density is, the higher the infection ratio of the system is and the smaller the spreading threshold is. In addition, the simulations show that the individual movement can enhance the stable infection ratio of the system only under the condition that the spreading rate is high, however, individual movement will reduce the stable infection ratio of the system.

Keywords: scale-free networks, systemic inflammatory response syndrome model, analog simulation

PACS: 05.45.–a, 05.10.–a, 02.60.Cb **DOI:** 10.1088/1674-1056/20/9/090503

1. Introduction

In 1998, Watts and Strogatz (WS) published an article entitled "Collective dynamics of small-world networks".[1] The WS small-world network model was proposed in the article. The model reveals the small-world property of realistic systems. In 1999, Barabási and Albert (BA) published an article entitled "The emergence of scaling in random networks".[2] The famous BA scale-free model was proposed in the article. The model reveals the scale-free property of realistic systems. Many studies have shown that almost all realistic networks have small-world and scale-free properties.[3−6] The two excellent articles have motivated many scholars' interest in the research of complex networks.

Almost all practical systems have network structures. In addition, there are many similar statistical features in the complex networks of practical systems in different domains. So the complex network theory has become an indispensable tool to study complex systems. It helps us to understand the fundamental laws in realistic complex systems.[7−15]

As the spreading phenomenon is universal and closely related to our daily life, one of the main goals in the study of complex networks is to reveal and understand their spreading behaviour. In recent years, scholars have done a lot of research in this domain and obtained plenty of results. However, the research mainly focuses on the effect of network structure on the spreading process.[14−27] The effect of non-structural factors on the spreading process has rarely been studied. In fact, many non-structural factors can affect the spreading process significantly. In this paper, the effects of some typical non-structural factors (artificial immunization, feedback mechanism, crowd density and individual moving) on the spreading process in scale-free networks will be studied. The laws will be investigated by building a new systemic inflammatory response syndrome epidemic model.

2. Building model

In a realistic system, artificial immunization is one of the most important measures for disease prevention and control. The method turns a suscepti-

*Project supported by the Natural Science Foundation of the Education Department of Guizhou Province, China (Grant No. 20090133) and International Cooperative Foundation of Guizhou Province, China (Grant No. 20117007).

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ble individual into an immune individual by direct immunization.[28−32]

In a realistic system, when a disease is spreading and the number of infections is increasing, people will reduce contact with others on their own initiative and the number of infections will eventually be reduced. However, when the number of infections starts to decrease, people will relax their vigilance and the number of infections may again increase. Obviously, adaptive feedback has an effect on the virus spreading. In order to reveal the influence of the feedback, the ideas of feedback are added to the classical epidemic model. Suppose that all individuals in the network know the situation of the disease spreading. That is to say, the individual knows the proportion of the infected individuals and actively disconnects his connection with his neighbors at a certain developmental stage of the disease spreading.[29,30]

When an epidemic disease is spreading, emphasis should be placed on the control of the disease spreading in a region where the population density is high. In addition, frequently moving people should be restricted. In order to reveal the influence of the crowd density and the moving activity of the individual on the disease spreading, we suppose that the crowd is distributed sparsely in the network with a specified percentage. The crowd density d is defined to be the proportion of the number of individuals to the number of nodes in the network. The moving process follows the following rules. At each time step, an infective individual can move to a selected location randomly. If the target location has been occupied by other individual, their positions are exchanged.[31]

In the SIRS epidemic model, the individuals can only exist in three different states, which are the susceptible state (S), the infective state (I) and the recovered state (R). A susceptible individual can be infected and becomes an infective individual, an infective individual can be cured and becomes an immunity individual (recovered state), an immunity individual can lose his immunity and becomes a susceptible individual again. The process can be simply described as S→I→R→S. Suppose that there are only a small number of individuals being infected at the initial moment. Let $s_k(t)$, $\rho_k(t)$ and $r_k(t)$ respectively denote the ratios of the susceptible, the infective and the recovered nodes with degree k at time t. Let $\rho(t)$ be the average ratio of all infective nodes at time t. We assume the infection probability is α, the cure probability is β, the losing immunity probability is γ, the artificial immunity probability is δ and the disconnect probability is μ. Meanwhile, the crowd density is assumed to be d. According to the mean-field theory, the dynamic behaviour of the model with the artificial immunity, the feedback mechanism and the crowd density can be described by

$$\begin{cases} \dfrac{\partial s_k}{\partial t} = -\alpha(kd)(s_k - \mu\rho)\Theta(t) \\ \qquad\qquad +\gamma(1 - s_k - \rho_k) - \delta s_k, \\ \dfrac{\partial \rho_k}{\partial t} = \alpha(kd)(s_k - \mu\rho)\Theta(t) - \beta\rho_k, \end{cases} \tag{1}$$

where $\Theta(t)$ denotes the probability that any given link points to an infected individual.

For the scale-free network with a degree of no-correlation, we denote the degree distribution by $P(k)$. When the system is in the steady state, let $\Theta = \Theta(\infty)$, $\rho_\infty = \rho(\infty)$, we can obtain

$$\rho_k(\infty) = \frac{\alpha(kd)\Theta(\gamma - \gamma\mu\rho_\infty - \delta\mu\rho_\infty)}{\alpha(kd)(\beta+\gamma)\Theta + \beta(\gamma+\delta)}. \tag{2}$$

So

$$\rho_\infty = \sum_k P(k)\frac{\alpha(kd)\Theta(\gamma - \gamma\mu\rho_\infty - \delta\mu\rho_\infty)}{\alpha(kd)(\beta+\gamma)\Theta + \beta(\gamma+\delta)}, \tag{3}$$

$$\Theta = \frac{1}{\langle k\rangle}\sum_k kP(k)\frac{\alpha(kd)\Theta(\gamma - \gamma\mu\rho_\infty - \delta\mu\rho_\infty)}{\alpha(kd)(\beta+\gamma)\Theta + \beta(\gamma+\delta)}. \tag{4}$$

From the condition that Eq. (4) has a nonzero solution, we can obtain

$$\lambda_{\rm c} = \frac{\alpha}{\beta} = \frac{\langle k\rangle}{\langle k^2\rangle}\left(\frac{1}{d} + \frac{\delta}{d\gamma}\right), \tag{5}$$

where $\lambda_{\rm c}$ is the epidemic threshold of the system. For the BA scale-free network, let m be the minimum number of connections at every node, then $P(k) = 2m^2/k^3$. If the size of the network is infinity ($N \to \infty$), then $\langle k\rangle = 2m$, $\langle k^2\rangle \to \infty$. According to Eq. (5), we know $\lambda_{\rm c} \to 0$.

Integrating Eq. (3) and taking the limit $N \to \infty$ yields

$$\rho_\infty = \frac{\dfrac{2md\alpha\gamma\Theta}{\beta(\gamma+\delta)} - \dfrac{2m^2d^2\alpha^2(\beta+\gamma)\gamma\Theta^2}{\beta^2(\gamma+\delta)^2}\ln\left[1 + \dfrac{\beta(\gamma+\delta)}{m\alpha d(\beta+\gamma)\Theta}\right]}{1 + \dfrac{2\mu md\alpha\Theta}{\beta} - \dfrac{2\mu m^2d^2\alpha^2(\beta+\gamma)\Theta^2}{\beta^2(\gamma+\delta)}\ln\left[1 + \dfrac{\beta(\gamma+\delta)}{m\alpha d(\beta+\gamma)\Theta}\right]}. \tag{6}$$

Integrating Eq. (4) and taking the limit $N \to \infty$ yields

$$\Theta = (\gamma - \gamma\mu\rho_\infty - \delta\mu\rho_\infty)\frac{md\alpha\Theta}{\beta(\gamma+\delta)} \ln\left[1 + \frac{\beta(\gamma+\delta)}{md\alpha(\beta+\gamma)\Theta}\right]. \tag{7}$$

Equation (7) can be simplified as

$$\Theta = \frac{\beta(\gamma+\delta)}{md\alpha(\beta+\gamma)}\left\{\exp\left[\frac{\beta(\gamma+\delta)}{md\alpha(\gamma-\gamma\mu\rho_\infty-\delta\mu\rho_\infty)}\right]-1\right\}^{-1}. \tag{8}$$

Substituting Eq. (8) into Eq. (6), we find that ρ_∞ satisfies the relation

$$\begin{aligned}\rho_\infty = {} & \frac{2\left[\gamma-(\gamma+\delta)\mu\rho_\infty\right]}{\beta+\gamma}\left\{\exp\left[\frac{\beta(\gamma+\delta)}{md\alpha(\gamma-\gamma\mu\rho_\infty-\delta\mu\rho_\infty)}\right]-1\right\}^{-1} \\ & - \frac{2\beta(\gamma+\delta)}{md\alpha(\beta+\gamma)}\left\{\exp\left[\frac{\beta(\gamma+\delta)}{md\alpha(\gamma-\gamma\mu\rho_\infty-\delta\mu\rho_\infty)}\right]-1\right\}^{-2}.\end{aligned} \tag{9}$$

Equation (9) is a transcendental equation. Although we can not obtain the analytic expression for ρ_∞, the relations between ρ_∞ and μ, and between ρ_∞ and d can be obtained by setting relational parameters, which are plotted in Figs. 1, 2 and 3, respectively.

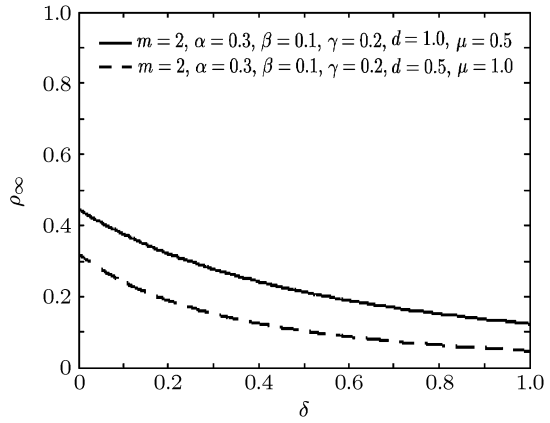


Fig. 1. Relation between ρ_∞ and δ in the SIRS model.

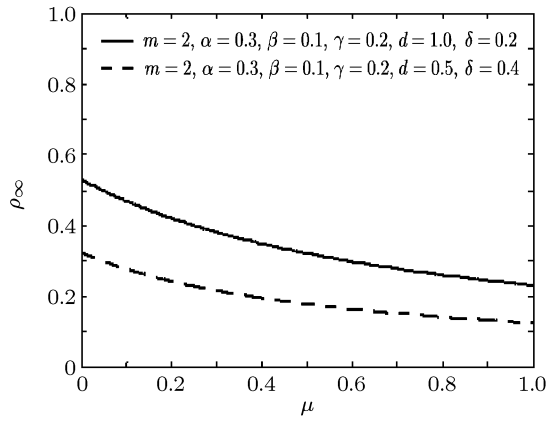


Fig. 2. Relation between ρ_∞ and μ in SIRS model.

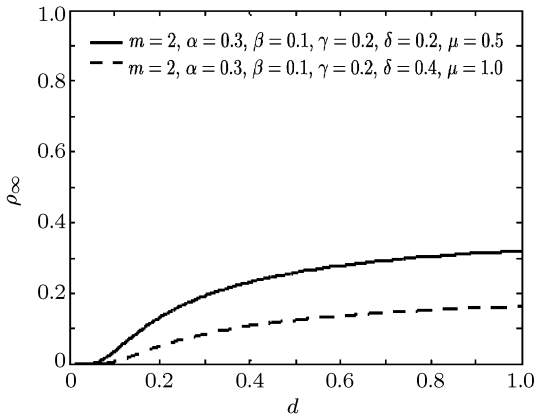


Fig. 3. Relation between ρ_∞ and d in SIRS model.

From Fig. 1, we can see that ρ_∞ decreases with the increase of δ. From Fig. 2, we can see that ρ_∞ decreases with the increase of μ. From Fig. 3, we can see that ρ_∞ increases with the increase of d.

If the size of the network is finite (N is finite),[32] the maximum connectivity $k_c \approx mN^{1/2}$, then

$$\begin{aligned}\lambda_c &= \frac{\langle k\rangle}{\langle k^2\rangle}\left(\frac{1}{d}+\frac{\delta}{d\gamma}\right) \\ &= \frac{\int_m^{k_c} k(2m^2k^{-3})\mathrm{d}k}{\int_m^{k_c} k^2(2m^2k^{-3})\mathrm{d}k}\left(\frac{1}{d}+\frac{\delta}{d\gamma}\right) \\ &\approx \frac{2}{m\ln N}\left(\frac{1}{d}+\frac{\delta}{d\gamma}\right).\end{aligned} \tag{10}$$

From the above expression, we can see that the epidemic threshold λ_c of the system is not zero. Moreover, it is related to the artificial immunization rate δ and crowd density d, but is unrelated to the feedback parameter μ.

For a scale-free network with degree correlations, such as the Internet, $P(k'|k)$ denotes the conditional

probability that a node of connectivity degree k is connected to a node of connectivity degree k', then

$$\Theta(t) = \sum_{k'} P(k'|k)\rho_{k'}. \tag{11}$$

When the system is in the steady state, relation (11) yields

$$\Theta = \sum_{k'} P(k'|k) \frac{\alpha(k'd)\Theta(\gamma - \gamma\mu\rho_\infty - \delta\mu\rho_\infty)}{\alpha(k'd)(\beta+\gamma)\Theta + \beta(\beta+\delta)}. \tag{12}$$

From the condition that Eq. (12) has a nonzero solution, we can obtain

$$\lambda_c = \frac{\alpha}{\beta} = \frac{1}{\sum\limits_{k'}(k'd)P(k'|k)} \left(\frac{1}{d} + \frac{\delta}{\gamma d}\right). \tag{13}$$

Obviously, if conditional probability $P(k\prime|k)$ is given, we can obtain the epidemic threshold λ_c from expression (13).

From the above analysis, when $d = 1.0$, $\delta = 0$, $\mu = 0$, the model changes into a classical SIRS model.[33]

3. Computer simulation

In order to test the results above, simulation experiments are carried out in a BA scale-free network. The size N of the network is 5000. The initial infective ratio is 0.01.

Experiment 1 In order to investigate how the crowd density, artificial immunity and the feedback mechanism affect the disease spreading, the simulation parameters are set as $\alpha = 0.3$, $\beta = 0.1$, $\gamma = 0.2$. Moreover, we set $d = 1.0$, $\delta = 0.0$, $\mu = 0.0$; $d = 0.6$, $\delta = 0.0$, $\mu = 0.0$; $d = 0.6$, $\delta = 0.1$, $\mu = 0.0$ and $d = 0.6$, $\delta = 0.1$, $\mu = 0.5$, respectively. The simulation experiments are carried out under the conditions that the crowd is static or is moving, respectively. Each experiment is repeated 50 times and then the averages are taken. The results of the simulations are shown in Fig. 4.

From Fig. 4, we can see that the results of the experiments agree with the conclusions of the theoretical analysis. In addition, we find that the individual movement can affect the stable infection ratio of the system.

Experiment 2 In order to investigate the evolution tendency of the stable infection ratio with the spreading rate, the simulation parameters are set as follows: α is uncertain, $\beta = 0.1$, $\gamma = 0.2$. Moreover, we set $d = 1.0$, $\delta = 0.0$, $\mu = 0.0$ and $d = 0.6$, $\delta = 0.0$, $\mu = 0.0$, respectively. The value of λ is determined by infected probability α. The simulation experiments are carried out under the conditions that the crowd is static or is moving, respectively. Each experiment is repeated 50 times and then the averages are taken. The results of the simulations are shown in Fig. 5.

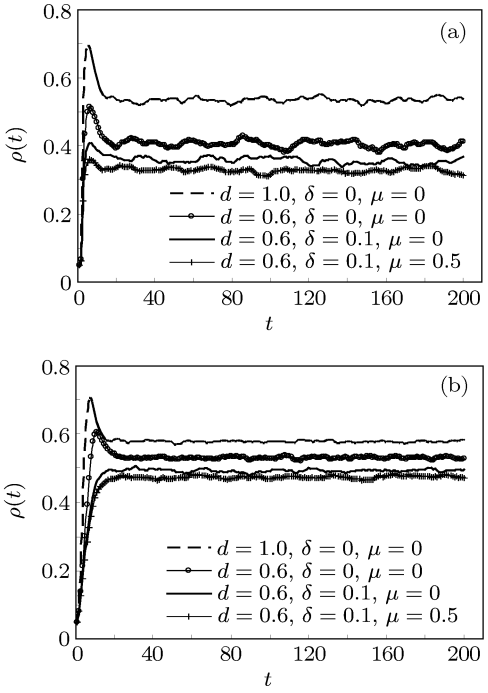


Fig. 4. Evolution tendencies of a stable ratio with time ($\lambda = \alpha/\beta = 3$) for (a) a static crowd and (b) a moving crowd.

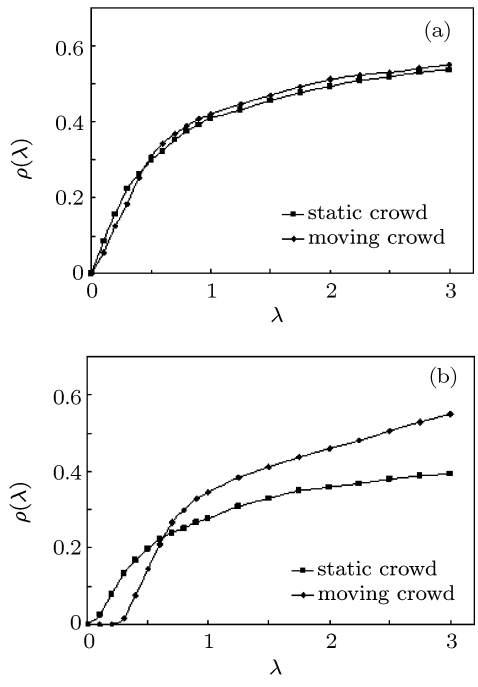


Fig. 5. Effect of crowd density and crowd moving on the spreading threshold of the SIRS model for (a) $d = 1.0$ and (b) $d = 0.6$.

From Fig. 5, we can see that the spreading threshold tends to zero because of the power law of the node degree distribution in the scale-free network. In addition, if the crowd density is low, the individual movement can enhance the stable infection ratio of the system only under the condition that the spreading rate is high. However, the individual movement can reduce the stable infection ratio of the system under the condition that the spreading rate is low.

Experiment 3 In order to investigate the effect of artificial immunity on the spreading threshold of the system, the simulation parameters are set as follows: α is uncertain, $\beta = 0.1$, $\gamma = 0.2$. Moreover, we set $d = 1.0$, $\delta = 0.0$, $\mu = 0.0$; $d = 1.0$, $\delta = 0.2$, $\mu = 0.0$ and $d = 1.0$, $\delta = 0.4$, $\mu = 0.0$, respectively. The value of λ is determined by infection probability α. The simulation experiments are carried out under the conditions that the crowd is static or is moving, respectively. Each experiment is repeated 50 times and the averages are taken. The results of the simulations are shown in Fig. 6.

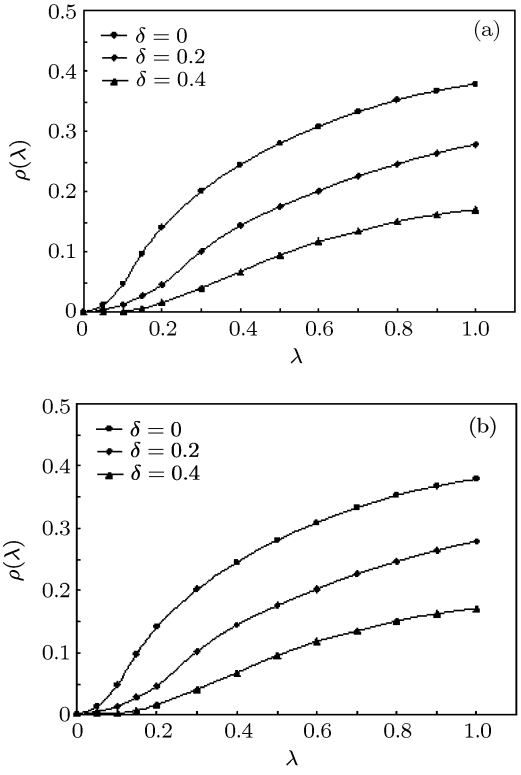


Fig. 6. Effect of artificial immunity on the spreading threshold of the SIRS model for (a) a static crowd and (b) a moving crowd.

From Fig. 6, we can see that artificial immunity can decrease the stable ratio and increase the spreading threshold of the system. However, when the network is scale-free, the spreading threshold of the system disappears when the network is infinite.

Experiment 4 In order to investigate the effect of the feedback mechanism on the spreading threshold of the system, the simulation parameters are set as follows: α is uncertain, $\beta = 0.1$, $\gamma = 0.2$. Moreover, we set $d = 1.0$, $\delta = 0.0$, $\mu = 0.0$; $d = 1.0$, $\delta = 0.0$, $\mu = 0.5$ and $d = 1.0$, $\delta = 0.0$, $\mu = 1.0$, respectively. The value of λ is determined by the infection probability α. The simulation experiments are carried out under the conditions that the crowd is static or is moving, respectively. Each experiment is repeated 50 times and then the averages are taken. The results of the simulations are shown in Fig. 7.

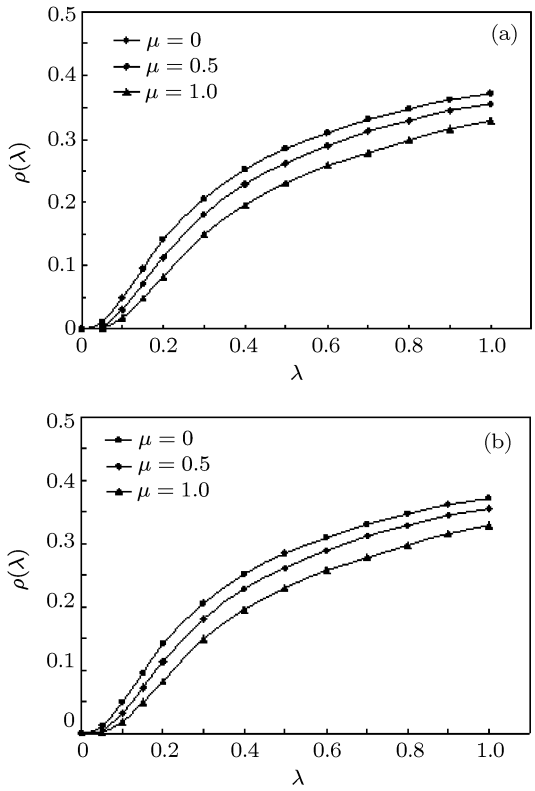


Fig. 7. Effect of feedback mechanisms on the spreading threshold of the SIRS model for (a) a static crowd and (b) a moving crowd.

From Fig. 7, we can see that the feedback mechanism can reduce the stable ratio of the system, but can not affect the spreading threshold of the system. Moreover, the spreading threshold of the system tends to zero when the network is infinite.

4. Conclusion

In this paper, based on an integrated SIRS model, the effects of crowd density, artificial immunity, the

feedback mechanism and individual movement on the spread of disease in a scale-free network are studied. Theoretical analysis and computer simulation show that artificial immunity can decrease the stable ratio and increase the spreading threshold of the systems; the feedback can reduce the stable ratio of the system, but cannot affect the spreading threshold of the system. With the decrease of crowd density, the stable infective ratio of the system declines, meanwhile, the epidemic threshold of the system extends. However, due to the scale-free property of the network, the spreading threshold of the system disappears when the network is infinite. The simulation also shows that individual movement can enhance the stable infection ratio of the system only under the condition that the spreading rate is high, however, individual movement can reduce the stable infection ratio of the system. These conclusions provide theoretical support for disease prevention and control measures, which include artificial immunity, the epidemic reporting and announcement policy, and the restriction of massive crowd gathering activities and crowd movement.

The work is based on the unweighted networks. In realistic systems, the connections between individuals are different, so the disease spreading process on weighted networks will be studied in the near future.

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