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Pattern dynamics in the epidemic model with diffusion network

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Abstract – It is well known that the outbreak of infectious diseases is affected by the diffusion of the infected. However, the diffusion network is seldom considered in the network-organized SIR model. In this work, we investigate the effect of the maximum eigenvalue on Turing instability and show the role of network parameters (the network connection rate, the network's infection, etc.) on the outbreak of infectious diseases. Meanwhile, stability of network-organized SIR is given by using the maximum eigenvalue of the network matrix which is proportional to the network connection rate and the networks infection rate. The bridge between the two rates and Turing instability was also revealed which can explain the spread mechanism of infectious diseases. In the end, some measures to mitigate the spread of infectious diseases are proposed and the feasible strategies for prevention and control can be provided in our paper, the data from COVID-19 validated the above results.

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Introduction. – After COVID-19 was first reported in December 2019, some researchers investigated the early transmission dynamics and its assessment through datadriven models [1,2]. However, the available data is often incomplete or insufficient in such a short interval. Therefore, the mathematical model is an important tool for analyzing the spread and prevention of epidemic and pandemic diseases [3-8] through bifurcation [9,10]. Stolerman et al. tried to deal with the propagation of disease epidemics in highly populated cities through the SIR-network model [11]. Dottori and Fabricius investigated the quasistationary state of a SIR model on a dynamical network and found rescaling the rate of infective contacts of the model could absorb network effects partially [12]. And the propagation of SIR disease on random networks with spatial structure was studied, which provided a theoretical modeling framework for disease propagation [13]. Then Ball et al. investigated a stochastic SIR network epidemic model with the preventive dropping of edges to show the importance of the connection between nodes [14]. Although the diffusion network was treated as an important factor of infectious diseases [15,16], the diffusion network's role in the distribution of the infected was seldom investigated, especially in the SIR model with the random network [17,18]. Therefore, spatial considerations have been included in this study.

Since the Turing pattern was first proposed in 1952 [19], many researches about pattern formation [20–22] were carried out to explain some biological mechanisms. It was found that cellular networks also played a vital role in the Turing instability, and some extensive networks were proposed as well [23–25]. Recently, some theoretical research methods of the network-organized Turing pattern about the un-directed and directed network were provided, which laid the basis for the study of network-organized pattern dynamics [26–29]. The subsequent research showed key features (like stability, pattern formation) of Turing systems are determined purely by the network topology in the network-organized system [30–32]. However, the pattern dynamics of the SIR model with the diffusion network was never investigated.

It is well known that human relations in society can be treated as an extensive network, the epidemic can spread along with the network when infectious diseases occur. In general, the spreading along the network could induce the outbreak of infectious diseases, especially COVID-19. However, the diffusion (network) does not work in the

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stability of the reaction-diffusion SIR system. Namely, traditional diffusion no longer explains the spread of infectious diseases in the SIR model. Therefore, some dynamics mechanisms and biological mechanisms should be proposed to investigate the epidemic's diffusive behavior. To understand the dynamical and biological mechanism of the outbreak of infectious diseases and explore the practical measures, we will investigate the dynamic behavior of a modified SIR model and the effect of the random network on the distribution of infectious diseases. We first propose a modified SIR model based on the characteristic of infectious diseases, analyze why the outbreak of infectious diseases occurs, and show the importance of the strategies. Furthermore, we construct a network-organized SIR model, show the connection probability and the infection rate of network that play an essential role in the Turing instability. Also, we find the maximum of the network matrix's eigenvalues, which is proportional to the connection probability and the infection rate of the network, affects the Turing system's stability directly. The Turing instability is important due to spatial consideration, which is specifically important in the outbreak of infectious diseases. Finally, the evolution of the infected cases of the COVID-19 epidemic is analyzed.

Model of the modified SIR system. – It is well known that the death rate of infectious diseases for S(susceptible individuals), I (infectious individuals), R (removed or recovery individuals) is different [33]. Meanwhile, the infection rate of those diagnosed with infectious diseases is also different from the participant infection rate (*i.e.*, that could infect others), because most of the infected can be isolated. Nevertheless, the incubation of the infectious diseases is difficult to check, and it is infective when it is in the incubation stage, so the amount of the participant infection is different from the final diagnosis. Due to the characteristic of infectious diseases and the existing SIR model [5], a modified SIR model can be written as

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \alpha - \beta_1 S - \gamma_1 S I,$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \gamma_2 S I - \beta_2 I - \delta I,$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \delta I - \beta_3 R,$$
(1)

where $\beta_1, \beta_2, \beta_3$ is the death rate of S, I, R, and $\beta_3 \leq \beta_1 \leq \beta_2$ generally holds because the natural mortality rate is relatively low and the mortality rate of those infected is relatively high; γ_1 is the infection rate (including both isolated and un-isolated infected individuals), γ_2 is the infection rate of the participant infection (including the incubation and the un-isolated infected individuals) next, $(\gamma_1 - \gamma_2)SI \ (\gamma_1 \geq \gamma_2)$ infectious individuals may be isolated or hospitalized, who cannot infect the others next, namely only the infected who could make a difference to the outbreak of infectious diseases are considered; δ is the recovery rate.



Fig. 1: The stability and bifurcation of system (2). (a) The bifurcation about α . (b) The bifurcation about β_2 . (c) The bifurcation about γ_2 . (d) The bifurcation about δ .

There are two equilibrium points (S^*, I^*, R^*) : $E_1 = (\frac{\alpha}{\beta_1}, 0, 0)$ and $E_2 = (\frac{\beta_2 + \delta}{\gamma_2}, \frac{\alpha \gamma_2 - \beta_1 \delta - \beta_1 \beta_2}{\gamma_1 (\beta_2 + \delta)}, \frac{\delta(\alpha \gamma_2 - \beta_1 \delta - \beta_1 \beta_2)}{\gamma_1 \beta_3 (\delta + \beta_2)})$ making the right-hand side of system (2) equal to zero. In general, the equilibrium point E_1 means the infectious diseases disappears, and the equilibrium point E_2 means the outbreak of infectious diseases. Then, we analyze the stability of E_1, E_2 through linear stability analysis. The characteristic values are the solution of the characteristic equation, the system is stable when all the eigenvalues are negative and the system is unstable when a positive eigenvalue exists. The characteristic equation can be expressed as $|\lambda E - A| = 0$, where A is the Jacobian matrix and the linear part of system (2)

$$A = \begin{pmatrix} -\beta_1 - \gamma_1 I^* & -\gamma_1 S^* & 0\\ \gamma_2 I^* & \gamma_2 S^* - \beta_2 - \delta & 0\\ 0 & \delta & -\beta_3 \end{pmatrix}.$$

The characteristic equation for E_1 is $(\lambda + \beta_1)(\lambda + \beta_3)(\lambda - \beta_3)(\lambda -$ $\gamma_2 \frac{\alpha}{\beta_1} + \delta + \beta_2 = 0$ and the characteristic equation for E_2 is $(\lambda_1 + \beta_3)(\lambda^2 + (\beta_1 + \gamma_1 I^*)\lambda + \gamma_1 \gamma_2 S^* I^*) = 0$. There are two states for the epidemic in this paper, the outbreak of infectious diseases, and eventually, disappearance with time. According to the characteristic value of E_1, E_2 , E_1 is stable when $\alpha \gamma_2 \leq \beta_1 \delta + \beta_1 \beta_2$, E_2 is stable when $\alpha \gamma_2 \geq \beta_1 \delta + \beta_1 \beta_2$. It is well known that the infectious diseases can be effectively controlled when the equilibrium E_1 of system (2) is stable and the infectious diseases will spread far and wide when the equilibrium E_2 is stable. In this paper, the default parameters of our simulations are $\alpha = 0.1, \beta_1 = 0.01, \beta_2 = 0.1, \beta_3 = 0.01, \gamma_1 =$ $0.8, \gamma_2 = 0.1, \delta = 0.1$ and initial value (1, 1, 1). E_1 will tend to be stable (the infectious diseases disappear) for lower birth rate, and E_2 is stable (outbreak of infectious diseases) when α becomes larger (fig. 1(a)). We are more

concerned about the death rate β_2 of the infected people. Although the higher death rate can induce the disappearance of infectious diseases (fig. 1(b)), it is an unacceptable way to prevent contagious diseases. The theoretical results show that γ_1 does not affect the stability of the equilibrium E_1 . If we decrease the infection rate of the participant infection (*i.e.*, γ_2) from 0.1 to 0.01, the outbreak disappears (fig. 1(c)). Moreover, the outbreak of infectious diseases may occur when the incubation time is large because the incubation of infectious diseases could infect others. Therefore testing the incubation of people on a large scale and the isolation of the infected are important strategies for the prevention of the infection, especially in some places where the number of infected grows exponentially (fig. 1(c)). For the recovery rate δ of the infected, the lower recovery rate is not good for the prevention of infection (fig. 1(d)). The 10 fold increase of the recovery rate (δ) (*i.e.*, from 0.1 to 1.0) could make the infectious diseases disappear quickly (fig. 1(d)). We notice that the recovery rate is a vital factor to prevent the outbreak (fig. 1(d)). The higher recovery rate (including self-cure) could also lead to the generation of herd immunity, which enhances the immune system of the recovered against the infectious diseases. It is also explained that the vaccine and isolation strategies are an effective measure to take control of the spread of infectious diseases. Although high-efficacy drugs are challenging to obtain for this current epidemic, it is crucial to deal with future epidemic. From fig. 1, we know only the endemic disease or the disappearance of infectious diseases happens in the general SIR model. Still, we cannot explain the secondary outbreak of infectious diseases in the social network. Therefore, we investigate the network-organized SIR system in the following.

Model of the network-organized SIR system. - In fact, traditional diffusion no longer explains the spread of infectious diseases in the SIR model. Therefore, we try to transfer eq. (2) into the network scheme. And we combine the dynamics of the SIR model and the infection network to investigate the infection's dynamical behavior. For the random network [28], we can transfer it into a symmetric matrix A_1 . First, a null matrix A_1 is given; then if node i and j connect in a loop, $A_1(i,j) = 1, A_1(j,i) = 1$ are assigned. Finally, we repeat the above process till a symmetric matrix A_1 is obtained. Suppose r_i is the *i*-th row sum (degree) of A_1 , and $A_2 = A_1 - \text{diag}(r_1, \ldots, r_N)$ (the degree) is a Laplacian matrix (the elements of the main diagonal represent the spreading rate of the network through diffusion and the other non-zero element is the infection rate by the receipt of diffusion). We treat matrix A_2 as a diffusion matrix. Meanwhile, the number of the infected is invariable (all row sums are zero). Finally, assume $\Delta = A_2 + q \operatorname{diag}(r_1, \ldots, r_N)$ and q is the infection rate through the network (we know that the amount of susceptibility, infection, and recovery varies in the infectious process, we redefine the ratio of the spreading rate and the receipt in the diffusive matrix). Meanwhile, q can be treated as the perturbation of the social network. Here the social distance p is different from the isolation in this paper. Although the social distance may decrease the infection rate, it could infect some people, and it may not even work for a highly contagious disease q. The isolation here means there is no contact.

In general, the number of the infected will increase by diffusion. After this consideration, system (2) (suppose the infected are not isolated and move freely in the 1st place) on the network can be written as

$$\frac{\mathrm{d}S_i}{\mathrm{d}t} = \alpha - \beta_1 S_i - \gamma_1 S_i I_i + d_1 \sum_{j=1}^N \Delta_{ij} S_j,$$

$$\frac{\mathrm{d}I_i}{\mathrm{d}t} = \gamma_2 S_i I_1 - \beta_2 I_i - \delta I_i + d_2 \sum_{j=1}^N \Delta_{ij} I_j, \qquad (2)$$

$$\frac{\mathrm{d}R_i}{\mathrm{d}t} = \delta I_i - \beta_3 R_i,$$

where $i = 1, \ldots, N$, only the infected in the first city (or place) affect others and $\sum_{j=1}^{n} \Delta_{ij} v_j^k = \Lambda_k v_i^k$, $\Lambda_k, v^k = (v_1^k, \ldots, v_n^k)$ are the k-th eigenvalue of Δ and the corresponding eigenvector, respectively. Substituting $S_i = \sum_{k=1}^{n} c_k e^{\lambda_k t} v_i^k$, $I_i = \sum_{k=1}^{n} b_k e^{\lambda_k t} v_i^k$, $R_i = \sum_{k=1}^{n} a_k e^{\lambda_k t} v_i^k$ into the linear part of system (2) [26], we obtain the Jacobian matrix of system (2), the characteristic equation $(\lambda_k + \beta_1 - d_1 \Lambda_k)(\lambda_k + \beta_3)(\lambda_k - \gamma_2 \frac{\alpha}{\beta_1} + \delta + \beta_2 - d_2 \Lambda_k) = 0$ at E_1 ; and Turing instability occurs when $d_1 \Lambda_k > \beta_1$ or $d_2 \Lambda_k > \delta + \beta_2 - \gamma_2 \frac{\alpha}{\beta_1}$ holds. The characteristic equation for E_2 is $(\lambda^2 + a_k \lambda + b_k)(\lambda + \beta_3) = 0$, and $a_k = -d_1 \Lambda - d_2 \Lambda_k + \gamma_1 I^* + \beta_1$, $b_k = \Lambda_k^2 d_1 d_2 - \Lambda_k d_2 \gamma_1 I^* + \gamma_1 S^* \gamma_2 I^* - \Lambda_k \beta_1 d_2$. The corresponding characteristic values are $\lambda_{k1,2} = \frac{a_k \pm \sqrt{a_k^2 - 4b_k}}{2}$, $\lambda_{k3} = -\beta_3$. And the Turing instability occurs when $\lambda_{k1} = \frac{a_k + \sqrt{a_k^2 - 4b_k}}{2} > 0$ holds [26,34].

Here we assume $\Lambda_1 \leq \Lambda_2 \leq \ldots \leq \Lambda_n$. If A is a Laplacian matrix (q = 0), the corresponding eigenvalues are $\Lambda_1 \leq \Lambda_2 \leq \ldots \leq \Lambda_n = 0$. It is easy to know that system (2) is stable at E_1 because $\lambda_{k1} = -\beta_1 + d_1\Lambda_k < 0, \lambda_{k2} = -\beta_3 < 0, \lambda_{k3} = \gamma_2 \frac{\alpha}{\beta_1} - \delta - \beta_2 + d_2\Lambda_k < 0$ when system (2) is stable. For E_2 , $\lambda_{k1,2} = \frac{a_k \pm \sqrt{a_k^2 - 4b_k}}{2} < 0$ always holds because $a_k > 0, b_k > 0$ is true for $\Lambda_k \leq 0$. Namely, the diffusive network could not induce outbreak of infectious diseases, which is not consistent with the actual situation. Therefore, A is considered in this paper, which can be treated as the perturbation of the Laplacian matrix.

From the above analysis, we know that the eigenvalues of the network matrix play an important role in the stability of the network-organized system (2), especially the maximum eigenvalue. So the stability of system (2) depends on

$$\frac{\mathrm{d}S_i}{\mathrm{d}t} = \alpha - \beta_1 S_i - \gamma_1 S_i I_i + d_1 \Lambda_i S_i,$$

$$\frac{\mathrm{d}I_i}{\mathrm{d}t} = \gamma_2 S_i I_1 - \beta_2 I_i - \delta I_i + d_2 \Lambda_i I_i, \qquad (3)$$
$$\frac{\mathrm{d}R_i}{\mathrm{d}t} = \delta I_i - \beta_3 R_i.$$

Namely Turing instability occurs when $d_2\Lambda_{max} > \delta + \beta_2 - \gamma_2 \frac{\alpha}{\beta_1} > 0$ or $d_1\Lambda_k > \beta_1$ holds. And the stability of system (3) with i = 1 can be determined by the following system:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \alpha - \beta_1 S - \gamma_1 S I + d_1 \Lambda_{max} S,$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \gamma_2 S I - \beta_2 I - \delta I + d_2 \Lambda_{max} I,$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \delta I - \beta_3 R,$$
(4)

where Λ_{max} is the maximum of Λ_i , and it is the reduced system for a network-organized system.

Although the network matrix is generated randomly with p, the range of its eigenvalues can be estimated. Based on the Gershgorin circle theorem [35], the eigenvalue $qr_i - 2r_i \leq \Lambda_i \leq qr_i \approx Npq$, and $\Lambda_{max} \approx Npq$. So system (4) is approximate to the following system:

$$\frac{\mathrm{d}S}{\mathrm{d}t} = \alpha - \beta_1 S - \gamma_1 SI + d_1 g N p q S,$$

$$\frac{\mathrm{d}I}{\mathrm{d}t} = \gamma_2 SI - \beta_2 I - \delta I + d_2 g N p q I,$$

$$\frac{\mathrm{d}R}{\mathrm{d}t} = \delta I - \beta_3 R,$$
(5)

where g is uncertain and related to A. And the system is unstable when $dgNpq > \delta + \beta_2 - \gamma_2 \frac{\alpha}{\beta_1} > 0$, otherwise, it is stable.

According to the above analysis, we have the following. **Theorem 1**. In the network-organized system (2), the Turing instability occurs for the equilibrium E_1 when $d_2\Lambda_k > \delta + \beta_2 - \gamma_2 \frac{\alpha}{\beta_1}$ or $d_1\Lambda_k > \beta_1$ holds. The turing instability occurs for the equilibrium E_2 when $\lambda_k > 0$ holds. Meanwhile, the stability of system (2) is determined by system (5).

Proof. Please refer to the stability analysis of system (2) and system (5).

It is well known that the social network and transregional diffusion of infectious diseases can induce the increase of the infected, even global outbreak. In contrast, the infectious diseases will approach to zero when the social network and trans-regional diffusion is small (figs. 2–8). In our simulation, system (2) is stable when all I_i tend to zero, and system (2) is unstable (Turing instability) when I_i does not approach zero $[d_2\Lambda_k > \delta + \beta_2 - \gamma_2 \frac{\alpha}{\beta_1}]$. Here $d_1 = 0.01, d_2 = 1$ are set. For example, we can make the infected of infectious diseases gradually disappear by decreasing the susceptible rate ($\alpha = 0.01$) and the social contacts (p = 0.01) when the social contacts exist in the system (fig. 2(a) shows that the pattern formation is stable). The excessive contacts (p = 0.1) of the infected lead to more infected (fig. 2(b)) and show the occurrence of Turing instability. Although the higher



Fig. 2: The stability of system (2) about p when q = 0.02. (a) The system is stable when $p = 0.01, \alpha = 0.01$. (b) Turing instability occurs when $p = 0.1, \alpha = 0.01$. (c) The system is stable when $p = 0.1, \beta_2 = 1.5$. (d) Turing instability occurs when $p = 0.7, \beta_2 = 1.5$.



Fig. 3: The stability of system (2) about p when q = 0.02. (a) The system is stable when $p = 0.01, \beta_1 = 0.1$. (b) Turing instability occurs when $p = 0.1, \beta_1 = 0.1$. (c) The system is stable when $p = 0.01, \gamma_2 = 0.01$. (d) Turing instability occurs when $p = 0.1, \gamma_2 = 0.01$.

death rate of the infected could cut off the resource of the infection $(p = 0.1, \beta_2 = 1.5)$, and make the infected approach to zero (fig. 2(c)). Surprisingly, even if the death rate $(\beta_2 = 1.5)$ is high, but through human exposures (p = 0.7) the infectious diseases spread rapidly, which lead to more deaths eventually (fig. 2(d)). Meanwhile, so is the death rate of the susceptible (fig. 3(a), (b)). In ancient times, maybe increasing the death rate was a way to prevent the spread of infection diseases because of medical constraints and poor transportation. However,



Fig. 4: The stability of system (2) about p when q = 0.02. (a) The system is stable when $p = 0.1, \delta = 1$. (b) Turing instability occurs when $p = 0.5, \delta = 1$. (c) The bifurcation about p when $\gamma_2 = 0.01$. (d) The bifurcation about p when $\alpha = 0.01$.



Fig. 5: The bifurcation about p when $\delta = 1$ and q = 0.02.

human civilisation is now advanced with better technological innovations. Isolation can effectively prevent the spread of the disease (fig. 3(c)). However, just a few infected cause an infection in the whole community through social contact (fig. 3(d)). Therefore, isolation and the decrease of social contacts are necessary to stop the spread of infectious diseases. Moreover, increasing the recovery (self-cure) rate is the best way to prevent the spread of infectious diseases (fig. 4(a)). This is not easy to achieve due to the development of a vaccine. The other option is to live with infectious diseases. If no symptoms or mild symptoms for most of the infected exist, or most of the infected can recover quickly by the treatment, eventually almost all the people live with infectious diseases (fig. 4(b)). So the herd immunity also depends on a strong health care system. In addition, fig. 4(c), (d) and fig. 5 show the variation of the equilibrium about p. The equilibrium gradually becomes non-zero with the variation of p(fig. 4(c), (d)) when we stabilize system (2) through the isolation (γ_2) and the susceptible rate α . Figure 5 shows that the strategy of increasing the recovery has a better resistance for the spread of infectious diseases. By comparing (fig. 4(c), (d)) and (fig. 5), we know the strategy of increasing the recovery of the infected is better than the



Fig. 6: The stability of system (2) about q. (a) Turing instability occurs when $p = 0.01, q = 0.1, \alpha = 0.01$. (b) Turing instability occurs when $p = 0.1, q = 0.15, \beta_2 = 1.5$. (c) Turing instability occurs when $p = 0.01, q = 0.1, \beta_1 = 0.1$. (d) Turing instability occurs when $p = 0.01, q = 0.1, \gamma_2 = 0.01$.



Fig. 7: The stability of system (2) about q. (a) Turing instability occurs when $p = 0.1, q = 0.1, \delta = 1$. (b) The bifurcation about q when $p = 0.01, \gamma_2 = 0.01$. (c) The bifurcation about q when $p = 0.1, \delta = 1$. (d) The bifurcation about q when $p = 0.01, \alpha = 0.01$.

isolation and the decrease of the susceptible rate. Because the increase of p means the isolation is not enough, and the susceptible rate increases. Now we consider the infection rate q of the network under the stable circumstances about p. As we all know, the system is stable when the infection rate q = 0.02 of the network is small (fig. 2(a), (c), fig. 3(a), (c), fig. 4(a)). Figures 6 and 7(a) show the Turing instability induced by q, namely the Turing instability occurs when the infection rate of the network becomes larger (fig. 6 and fig. 7(a)). But the condition that the strategy of increasing the death rate stabilizes system (2) (fig. 6(b), (c)) is worse than other ways (fig. 6(a), (d), fig. 7(a)). Namely, the infection rate q of the network



Fig. 8: The relationship of Λ_{max} and p, q. (a) The relationship of Λ_{max} and q when p = 0.1. (b) The relationship of Λ_{max} and p when q = 0.1. (c) The distribution of Λ_{max} about p and q. (d) The distribution of Npq about p and q.

also plays an important role in system (2). Also, although the same initial conditions are set, the dynamical behavior (fig. 6(d)) is different because the topology varies. Then we obtain the bifurcation of I_i about p, q (fig. 7(b)–(d) show the distribution of I_i about q). From the numerical results, we know the isolation strategy (fig. 7(c)) is better at preventing the network diffusion of infection diseases. Although increasing the recovery rate (fig. 7(b)) and reducing the susceptible rate (fig. 7(d)) is efficient at the beginning, the infected will increase rapidly when the infection rate of the network becomes large (fig. 7(b), (d)). In addition, it is found that p, q are proportional to Λ_{max} (fig. 8). And it approaches the linearity relationship between Λ_{max} and p, q (fig. 8(a), (b)). In other words, the magnitude of p, q affects the stability of system (4). Meanwhile, the similar distribution (fig. 8(c), (d)) of Λ_{max} and Npq shows $\Lambda_{max} \approx Npq$ holds.

The distribution of Λ_{max} and Npq about p,q also verifies that system (4) and system (5) are approximately equal (fig. 8). Also, the stability of system (4) is consistent with the stability of system (2) (fig. 9). Such as fig. 3(c), (d)and 7(d) correspond to fig. 9(a), (b), (c), respectively, which have the same stability between system (4) and system (2). Meanwhile the bifurcation about Λ_{max} agrees with the condition of the Turing instability of system (4) (fig. 9(d)). In summary, although the infected of one city are not isolated, this also could induce a secondary outbreak through the network. Finally, the evolution of the infected cases of the COVID-19 epidemic is analyzed. Because of external factors, the system will not approach an equilibrium point when the infected (freely) exist, and even a secondary outbreak occurs (fig. 10), which is consistent with our theoretical results (fig. 2–7]. From fig. 10, the infected cases increase at the beginning, and decrease when some measures are taken (figs. 2(a), (c), 3(a), (c), 4(a)). However, a secondary outbreak occurs because of



Fig. 9: The stability of system (4). (a) E_1 is stable when $\gamma_2 = 0.01, p = 0.01, q = 0.02$ and $\Lambda_{max} = 0.0362$. (b) E_1 is unstable when $\gamma_2 = 0.01, p = 0.1, q = 0.02$ and $\Lambda_{max} = 0.2057$. (c) E_1 is unstable when $\gamma_2 = 0.01, p = 0.01, q = 0.1$ and $\Lambda_{max} = 0.2179$. (d) The bifurcation about Λ_{max} .



Fig. 10: The infected cases of the COVID-19 epidemic. (a) Switzerland from 2020.02.27 to 2020.8.20. (b) Austria from 2020.03.29 to 2020.8.20.

the diffusion network (like the imported cases, the effect of other cities) (fig. 2(b), (d), fig. 3(b), (d), 4(b), 7(a)). The above analysis means the theoretical results are in agreement with some experimental results in the qualitative analysis. Different implementation measures have produced different results; namely, the topology is different (fig. 6). Finally, a general SIR model could show the outbreak or the disappearance of COVID-19 [36], but it is difficult to express the interaction between nodes (cities or countries) and the secondary epidemic. Generally speaking, the control of the network diffusion plays a vital role in the prevention of COVID-19. Also, although the same initial conditions are set, the dynamical behavior is different because the topology varies. Meanwhile, the topology is determined by the protection strategy of the nodes. Therefore, we could prevent the spread of COVID-19 through the connection between the nodes.

Conclusion. – To explain the mechanism of the spread of infectious diseases, and to predict the effective strategy, a qualitative approach was applied to explain the outbreak of infectious diseases due to the characteristics of infectious diseases, and some measures were provided and turned out to be effective for the prevention of infectious diseases. In this paper, we first show the modified SIR model's stability under different

conditions and analyze the feasibility of some measures for infectious diseases in the actual situation. It is found that the isolation measure is an important approach to reduce the infection rate: reducing the birth rate of the vulnerable by isolation and improving the rate of recovery by good health care systems could make the system stable (infectious diseases disappears), which shows that these are effective strategies. Then the effect of the random network on the distribution of infectious diseases is shown by the connection probability p and the network infection rate q, which overcomes the disadvantage that the network does not affect the dynamical behavior of infectious diseases in the network-organized SIR model. Meanwhile, social distancing, along with higher recovery rates and higher isolation, are effective measures because of the higher connection probability, which means lower social distancing, leading to the Turing instability. Also, the maximum of the network matrix's eigenvalues, which is proportional to the connection probability and the infection rate of the network, affects the stability of the Turing system directly. Above all, some measures and methods proposed may play a vital role in the outbreak of infectious diseases and Turing instability based on the theoretical analysis. In summary, although the infection of one city is not isolated, it could also induce a secondary outbreak through the network. Finally, the reduced system method for a network-organized system is proposed on the basis of Theorem 1, which is a novel approach to investigate the complex system.

It will be better to carry out the analysis based on the literature or estimates from accurate data. However, the SIR model is considered on the network, making the estimation and collection of accurate data more difficult. We only show the effect of the diffusion network on the epidemic by evolving the infected cases of the COVID-19 epidemic in Austria and Switzerland. And the network-organized system we proposed could describe the dynamical behavior of infectious diseases more accurately. Because the system's theoretical analysis holds for any parameters, the modified SIR model is valid for COVID-19. The validity of the model used in this paper and another data-based model is predicated on having accurate data. Therefore we may do some research about actual data, directed network, multilayer network, etc., in a subsequent study.

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