The Spread Behavior Analysis of a SIQR Epidemic Model under the Small World Network Environment

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Abstract. In this paper, we focus on the behavior of virus spreading on complex networks. A SIQR epidemic model on small-world network is established. We find the spread of infectious diseases threshold, which can determine the dynamic behavior of the system in a positive invariant set. If \( R_0 > 1 \), there are two equilibriums in the system, the disease-free equilibrium and the endemic equilibrium. If \( R_0 < 1 \), there is only the disease-free equilibrium in the system. It is globally stable in the positive invariant set. Furthermore, we analyze the equilibrium and its stability. Finally, some numerical simulation studies are provided to illustrate the effectiveness of the results.

1. Introduction
Infectious diseases have always been an enemy to human health, history and reality tell us: human beings are facing the long-term and serious threat of infectious diseases. The research on the pathogenesis of infectious diseases, regularity of infectious diseases, prevention and control strategy is becoming important. It is a significant problem that needs to be urgently addressed in today's world.

The reemergence of tuberculosis (TB) from the 1980s to the early 1990s instigated extensive researches on the mechanisms behind the transmission dynamics of TB epidemics. The earliest mathematical models describing the TB dynamics appeared in the 1960s and focused on the prediction and control strategies using simulation approaches. Reference [1] provides a detailed review of the work on the dynamics and control of TB. In the late 20th century, complex network theory developed rapidly, and became a new trend to study infectious diseases in complex networks. Scientists focused on SIS epidemic model and SIR epidemic model. And it is generally assume that that total number of individual is constant. Reference [2] discusses the SIS epidemic model in local area networks. However, the birth and death of some diseases seriously affect the total number of individual, so it is necessary to consider the birth and death of individuals in such circumstances [3]. Jiang et al. investigated the self-affirmation effect on formation of public opinion in a directed small-world social network [4]. Christensen et al. developed a framework for simulating a realistic, evolving social network (a city) into which a disease is introduced. They compared the results to prevaccine era measles data for England and Wales, and finded that they captured the quantitative and qualitative features of epidemics in populations spanning two orders of magnitude [5]. Discrete epidemic models with periodic epidemiological parameters in [6] are formulated and studied to take into account seasonal variations of infectious diseases. In the process of transmission of infectious diseases, pre-exposure prophylaxis (re-exposure prophylaxis (PrEP) consists in the use of an antiretroviral medication to prevent the acquisition infection by uninfected
individuals. It can significantly reduce the spread of infectious diseases. In [7], Xiao and Ruan study an epidemic model with nonmonotonic incidence rate, which describes the psychological effect of certain serious diseases on the community when the number of infective is getting larger.

Small world networks have small perturbation degree, approximation for the average degree $<k>$ [8]. Assume that the total number of network nodes remains unchanged. And the state nodes are evenly distributed. It can simulate the spread of infectious diseases well. However, the above studies ignored the spread environment, just thinking about the epidemic model alone, or in building an infectious disease model without considering isolation and immunity. This is deviate from the spread of infectious diseases. Therefore, in the small world networks, the method of isolating the source of infection and enhancing immunity should be adopted. This makes the conclusion true and reliable. In this paper we analysis and research on the dynamics of SIQR infectious disease models in small world networks, we find the threshold $R_0=(\delta+\gamma)/\beta <k>$, which determines the dynamic behavior of the system in a positive invariant set $D$. If $R_0>1$, there are two equilibriums in the system, the disease-free equilibrium $E_0$ and the endemic equilibrium $E_*$. $E_0$ in the set $D$ is unstable. $E_*$ in the set $D$ is locally asymptotically stable. If the disease is present at the beginning, then the disease will be stable in the population, and it forms endemic disease. If $R_0<1$, there is only a disease-free equilibrium $E_0$ in the system. It is globally stable in the set $D$. The disease will eventually die out. Finally numerical simulations are provided to illustrate the effectiveness of the results.

2. Establishment of SIQR Infectious disease model
There are four types of nodes in the network: susceptible $S$, infectious $I$, quarantine $Q$, recovery $R$. Small world networks have small perturbation degree, approximation for the average degree $<k>$. Assume that the total number of network nodes remains unchanged. And the state nodes are evenly distributed. $S(t)$, $I(t)$, $Q(t)$, $R(t)$ represent the proportion of each state node at time $t$. At the same time, $\beta$ represents the infection rate of the infected state node, At time $t$, the probability of the node in the susceptible state infected with the infected node is $<k>\beta$. The transformation relationship between states is shown in Figure 1, in which $\delta$ represents the probability that the infected node is isolated, $\varepsilon$ indicates the probability that the isolation node is cured, $\gamma$ stands for the probability that the infected node is cured, and $\alpha$ is the probability that the immune node loses immunity and becomes susceptible.

![Figure 1. The transformation relationship between the states of SIQR models in a uniform network.](image)

In the model, if a node in a susceptible state is connected to a node in the state of infection, it is infected with the probability of $<k>\beta$. So at time $t$, the susceptible node with the degree of $k$ is infected with a disease node with the probability of $k\beta$, since the proportion of the susceptible nodes is $S(t)$. So the susceptible node with the degree of $k$ is infected with a disease node at a rate of $<k>\beta S(t)$. At the same time, the removal of the immune node has been redeveloped into a node of the susceptible state with the probability of $\alpha$. Therefore, the probability of the susceptible node in the total node is $<k>\beta S(t)+\alpha R(t)$ at the time $t$. Similarly, the probability of infected nodes, the quarantine nodes and the recovery nodes are respectively $<k>\beta S(t)-\gamma I(t)-\delta I(t)$, $\delta I(t)-\varepsilon Q(t)$, $\varepsilon Q(t)+\gamma I(t)-\alpha R(t)$. According to the mean-field theory, the propagation dynamics equation can be obtained as follows:
3. The equilibriums and stability analysis of SIQR epidemic Model

3.1. The existence of the equilibriums

Use the normalized condition \( S(t)+I(t)+Q(t)+R(t)=1 \), we can simplify (1) to the following model:

\[
\begin{align*}
\frac{dS(t)}{dt} &= -<k> \beta I(t) S(t) + aR(t), \\
\frac{dI(t)}{dt} &= <k> \beta I(t) S(t) - \gamma I(t) - \delta I(t), \\
\frac{dQ(t)}{dt} &= \delta I(t) - \varepsilon Q(t), \\
\frac{dR(t)}{dt} &= \varepsilon Q(t) + \gamma I(t) - aR(t).
\end{align*}
\]  

(1)

D=\{I(t), Q(t), R(t)\} \cap \{I(t), Q(t), R(t)>0, I(t)+Q(t)+R(t)\leq 1\} is the positive invariant set of system (2). Let the right-hand side of (2) be equal to zero, obviously the zero point is the equilibrium point. Then, the system (1) has disease-free equilibrium \( E(1,0,0,0) \).

Let the right-hand side of (2) be equal to zero, other variables are indicated by \( I, S(t) = (\delta + \gamma)/\beta < k >, Q(t) = \delta I(t)/\varepsilon, R(t) = (\delta + \gamma)I(t)/\alpha \), then we have (\delta + \gamma)/\beta < k > + I(t) + \delta I(t)/\varepsilon + (\delta + \gamma)I(t)/\alpha = 1 and \( I(t) = (1 - R_0)/[1 + \delta/\varepsilon + (\delta + \gamma)/\alpha] \), where \( R_0 = (\delta + \gamma)/\beta < k > \). If \( R_0 < 1 \), there is a unique positive equilibrium \( E^* = (S^*, 1^*, Q^*, R^*) \) of the system (1) in the set of \( D \), where \( S(t) = (\delta + \gamma)/\beta < k >, I(t) = (1 - R_0)/[1 + \delta/\varepsilon + (\delta + \gamma)/\alpha], Q(t) = \delta I(t)/\varepsilon \) and \( R(t) = (\delta + \gamma)I(t)/\alpha \).

**Theorem 3.1** The model (1) always has a disease-free equilibrium \( E \), while \( R_0 < 1 \), the model still has an endemic equilibrium \( E^* \).

**Proof.** The proof of this theorem can be found in [9].

3.2. The stability analysis of equilibrium

**Theorem 3.2** If \( R_0 \geq 1 \), \( E \) is local asymptotic stability. If \( R_0 < 1 \), \( E \) is unstable.

**Proof.** The Jacobian matrix of model (3.1) at \( E \) is:

\[
J_0 = \begin{pmatrix}
<k> \beta - \gamma - \delta & 0 & 0 \\
\delta & -\varepsilon & 0 \\
\gamma & \varepsilon & -a
\end{pmatrix}
\]

The characteristic equation of \( J_0 \) is \((\lambda - <k> \beta + \gamma + \delta)(\lambda + \varepsilon)(\lambda + \alpha) = 0\), and eigenvalues are \( \lambda_1 = <k> \beta - \gamma - \delta, \lambda_2 = \varepsilon, \lambda_3 = -\alpha \).

If \( R_0 \geq 1 \), we have \( \lambda_1 \leq 0, \lambda_2 < 0, \lambda_3 < 0 \), then \( E \) is local asymptotic stability. If \( R_0 < 1 \), we have \( \lambda_1 > 0 \), then \( E \) is unstable in the set of \( D \). In other words, if \( R_0 \geq 1 \), the system (1) converges to the disease-free equilibrium \( E(1,0,0,0) \).

**Theorem 3.3** If \( R_0 \geq 1 \), \( E^* \) is local asymptotic stability.

**Proof.** The Jacobian matrix of model (2) at \( E^* \) is...
\[
J_0 = \begin{pmatrix}
<k > \beta S(t) & -<k > \beta I(t) & -\gamma & -<k > \beta L(t) \\
\delta & -\varepsilon & 0 & 0 \\
\gamma & \varepsilon & -a & 0
\end{pmatrix}.
\]

The characteristic equation of \( J_0 \) is
\[
\lambda^3 + \left( <k > \beta I(t) + \varepsilon + \alpha \right) \lambda^2 + \left( <k > \beta I(t) \left( \varepsilon + a + \delta + \gamma \right) + a \varepsilon \right) \lambda + <k > \beta I(t) \left( \varepsilon \alpha + a \delta + \varepsilon \delta + \gamma \varepsilon \right) = 0.
\]

Let \( \rho_1 = <k > \beta I(t) + \varepsilon + \alpha \), \( \rho_2 = <k > \beta I(t) \left( \varepsilon + a + \delta + \gamma \right) + a \varepsilon \), \( \rho_3 = <k > \beta I(t) \left( \varepsilon \alpha + a \delta + \varepsilon \delta + \gamma \varepsilon \right) \).

According to Hurwitz
\[
H_1 = \rho_1 = <k > \beta I(t) + \varepsilon + \alpha = \beta (1 - R_0) / [1 + \delta / (\varepsilon + \delta + \gamma) / \alpha] + \varepsilon + \alpha > 0,
\]
\[
H_2 = \rho_2 - \rho_3 = [<k > \beta I(t) + \varepsilon + \alpha] \left[ <k > \beta I(t) \left( \varepsilon + a + \delta + \gamma \right) + a \varepsilon \right] - <k > \beta I(t) \left( \varepsilon \alpha + a \delta + \varepsilon \delta + \gamma \varepsilon \right),
\]
where \( <k > \beta I(t) \left( \varepsilon \alpha + a \delta + \varepsilon \delta + \gamma \varepsilon \right) > 0 \), we have
\[
H_3 = \rho_1 \rho_2 - \rho_3 \left[ <k > \beta I(t) + \varepsilon + \alpha \right] \left[ <k > \beta I(t) \left( \varepsilon + a + \delta + \gamma \right) + a \varepsilon \right] - <k > \beta I(t) \left( \varepsilon \alpha + a \delta + \varepsilon \delta + \gamma \varepsilon \right) > 0.
\]

Hence, all the characteristic roots of \( J_0 \) have a negative real part. If \( R_0 \geq 1 \), \( E^* \) is locally asymptotically stable.

**Theorem 3.4:** If \( R_0 \geq 1 \), \( E \) is globally stable.

**Proof:** Since \( S(t) + I(t) + Q(t) + R(t) = 1 \), then \( S(t) \leq 1 \), we have
\[
\frac{dl(t)}{dt} = \begin{cases}
<k > \beta I(t) S(t) - \gamma I(t) - \delta I(t) \\
\leq <k > \beta I(t) - \gamma I(t) - \delta I(t)
\end{cases}
\]
\[
= I(t) \left[ <k > \beta - \left( \gamma + \delta \right) \right]
\]
\[
= I(t) <k > \beta (1 - R_0).
\]

If \( R_0 \geq 1 \), we have \( \frac{dl(t)}{dt} \leq 0 \), \( \lim_{t \to +\infty} I(t) = 0 \). If \( t \to +\infty \), the extreme equations of model (1) is
\[
\begin{align*}
\frac{dS(t)}{dt} &= aR(t), \\
\frac{dQ(t)}{dt} &= -\varepsilon Q(t), \\
\frac{dR(t)}{dt} &= \varepsilon Q(t) - aR(t).
\end{align*}
\]

Then, we have \( Q(t) = C e^{at} \), \( \lim_{t \to +\infty} Q(t) = 0 \). It can be calculated that \( \lim_{t \to +\infty} R(t) = 0 \), \( \lim_{t \to +\infty} S(t) = 1 - \lim_{t \to +\infty} I(t) - \lim_{t \to +\infty} Q(t) - \lim_{t \to +\infty} R(t) = 1 \).

Therefore, the equilibrium of the extreme equations of model (1) is globally stable. By the extreme equation theory [10], we can obtain that any solution \( E \) of the model (1) satisfy \( \lim_{t \to +\infty} E = (1, 0, 0, 0) \) when \( R_0 \geq 1 \), the disease-free equilibrium of the model (1) is globally stable.

4. **Numerical simulations**

In the numerical simulations, the global stability of disease-free equilibrium and the local asymptotic stability of local equilibrium is verified. And better understand the development trend of diseases. Let parameter \( N = 50000 \), \( m = 3 \), \( <k> = 6 \) to construct a small world network in the system (1). In addition, let parameter \( \varepsilon = 0.8, \gamma = 0.2, \alpha = 0.4 \), \( \beta = 0.05 \), and calculate that \( R_0 < 1 \), the model (1) has a disease-free equilibrium \( E \), \( E \) is globally stable. Let the initial values are \( S(0) = 0.2, I(0) = 0.2, Q(0) = 0.3, R(0) = 0.1 \); \( S(0) = 0.1, I(0) = 0.2, Q(0) = 0.3, R(0) = 0.3 \); \( S(0) = 0.3, I(0) = 0.2, Q(0) = 0.1, R(0) = 0.2 \). Numerical simulation of system (1) by Matlab, we can get the following Figure 2.
Figure 2. Global stability of the disease-free equilibrium when $R_0>1$ and three different initial values.

To construct the same small world network in the system (1). Let parameter $\epsilon=\delta=0.8$, $\gamma=0.2$, $\alpha=0.4$, $\beta=0.5$. Calculating $R_0<1$, the model (1) has a endemic equilibrium $E^*$, and $E^*$ is local asymptotic stability. Let the initial values are $S(0)=0.2$, $I(0)=0.2$, $Q(0)=0.3$, $R(0)=0.1$; $S(0)=0.1$, $I(0)=0.2$, $Q(0)=0.2$, $R(0)=0.3$; $S(0)=0.3$, $I(0)=0.2$, $Q(0)=0.1$, $R(0)=0.2$. Numerical simulation of system (2.1) by Matlab, we can get Figure 3.

Figure 3. Local asymptotic stability of the endemic equilibrium when $R_0<1$ and different initial values.

5. Conclusion

We study the dynamics of SIQR infectious disease models in small world networks, and find the threshold $R_0=(\delta+\gamma)/\beta<k>$, which can determine the dynamic behavior of the system in a positive invariant set $D$. If $R_0>1$, there are two equilibriums in the system, the disease-free equilibrium $E_0$ and the endemic equilibrium $E^*$. $E_0$ in the set $D$ is unstable, $E^*$ in the set $D$ is locally asymptotically stable. If the disease is present at the beginning, then the disease will be stable in the population, and it forms endemic disease. If $R_0<1$, There is only $E_0$ in the system. It is globally stable in the set $D$. The disease will eventually die out.

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