

GLOBAL DYNAMICAL ANALYSIS OF A HEROIN EPIDEMIC MODEL ON COMPLEX NETWORKS*

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Abstract In this paper, a heroin epidemic model on complex networks is proposed. By the next generation matrix, the basic reproduction number R_0 is obtained. If $R_0 < 1$, then the drug-free equilibrium is globally asymptotically stable. If $R_0 > 1$, there is an unique endemic equilibrium and it is also globally asymptotically stable. Our results show that if the degree of the network is large enough, the drug transmission always spreads. Sensitivity analysis of the basic reproduction number with the various parameters in the model are carried out to verify the important effects for control the drug transmission. Some simulations illustrate our theoretical results.

Keywords Heroin model, complex networks, global stability.

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1. Introduction

It has been reported that whether injected, snorted or smoked, heroin will begin to affect the body's central nervous system almost immediately after it is used since it is more soluble in the fat cells [9]. Heroin users are more prone to catch the human immunodeficiency virus (HIV) and the other blood-borne viruses diseases. One of the dangerous effects for heroin use is the highly addictive nature of the drug [16]. All heroin users, even those who only snort or smoke the drug, can become addicted with repeated use [1]. Heroin epidemics, as well as communicable diseases, are characterized by three main factors: a state of susceptibility in the given region, introduction of a provocative stimulus, and some degree of sensory contact between those primarily and those secondarily affected. Compartment modeling is one of the main method for studying the epidemiology. It is an interesting project to use the similar method to investigate the dynamics of heroin epidemic transmission.

Some very interesting models [4, 7, 8, 15, 24] have been recently proposed. White and Comiskey [24] propose an ODE model for studying the opiate addiction, based on the principles of mathematical epidemiology. Mulone and Straughan [8] restudied the model presented by White and Comiskey, and investigate the global stability

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of the equilibrium using the Poincaré-Bendixson theory. Liu and Zhang [7] investigated a heroin epidemic model with distributed time delays, they obtained the transmission threshold R_0 which determines the extinct and persistence of a heroin spread. Huang and Liu [4] got the the global stability of the model proposed in [7] by employing the suitable Lyapunov function. Wang et al [18] considered the mass action incidence rate and proved that the drug use-free equilibrium and the unique endemic equilibrium are globally asymptotically stable under some conditions by using the second compound matrix. Fang et al [2] used a DDE model with two distribute delays to study the global stability of a heroin epidemic model. Samantha [13] built a nonautonomous heroin epidemic model with distributed time delay and discuss the global stability of the model with the method of Lyapunov functional. For the ODE model, they generally assume that the individuals are uniform mixtures with homogenous contacts. These assumptions are not suitable for the large scale social network with obvious heterogeneities.

Recently complex network is a useful tool to describe the behaviors of the transmission disease. A new kind of complex networks: scale-free networks (BA) was introduce by Barabási in 1999. He assumed that the distribution probability of any node with degree k linking to other nodes is according to a power law $p(k) \sim k^{-\gamma}$. Many realistic systems have been verified to belongs to a BA complex network. Many communities diseases also exhibit characteristics consistent with a BA scale free complex network. The spread mechanism of disease on network have been reported and investigated in some papers and some text books by many authors. Some classical SIS and SIR models [3, 10–12, 19–23, 25, 26] have been widely investigated on large scale free networks. Wang and Dai [19, 20] gave the strict mathematical proof for the Pastor and Vespignani [10, 11] established SIS epidemic model in heterogeneous network. Wang and Jin [23] proposed an SIS epidemic model on scale-free network with multiple transmission routes to study the global dynamics of the equilibria. Zhang et al [25] also built an SIS epidemic model to describe the sexual transmission disease, they also got the global dynamics of the system using matrix theory and dynamics of the differential equation. Many other works about global analysis of the epidemic model on network are referenced in [3, 21, 22, 26].

Our model is aim to study the heroin transmission on a BA scale free complex network. This paper is organized as follows: Sec.2 introduces a a modified heroin model on complex networks. In Sec.3, the threshold and global stability of the drug-free equilibrium are studied. In Sec.4, we obtain the existence and global stability of the endemic equilibrium. In Sec.5, we give sensitive analysis of the basic reproduction number with the model parameters to compare the importance for control the drug transmission. Some simulations illustrate the theoretical results.

2. The model formulation

The nodes of the network links the possible individuals. The nodes have three different states, one is susceptible, and the drug-taking and the drug detoxification, denoted by $S_k(t)$, $U_{1k}(t)$ and $U_{2k}(t)$, respectively. The three states are described by "SU₁U₂U₁S" model. A drug-taking individual links with a susceptible individual and then may make the susceptible individual become a new drug-taking. The drug-taking and the drug detoxification return to an susceptible individual since he(he) is treated or educated. The drug detoxification individual is prone to repeat use

drug. This model is describe as follows.

$$\begin{cases} \frac{dS_k}{dt} = -k\sigma S_k \Theta(U_{1k}) + \gamma_1 U_{1k} + \gamma_2 U_{2k}, \\ \frac{dU_{1k}}{dt} = k\sigma S_k \Theta(U_{1k}) - (\gamma_1 + \delta_1) U_{1k} + \delta_2 U_{2k}, \\ \frac{dU_{2k}}{dt} = \delta_1 U_{1k} - (\delta_2 + \gamma_2) U_{2k}, \end{cases} \quad (2.1)$$

here σ is the infection transmission rate which each susceptible individual acquires the infection from an infected neighbor during one time step. δ_1 is abandon rate at which a drug-taking individual transfer to a drug detoxification individual, so that $1/\delta_1$ is the average abandon drug habit period. γ_1 is the cure rate at which the drug-taking can be cured. γ_2 is the cure rate at which the drug detoxification can be cured. δ_2 is repeated rate at which a drug detoxification individual repeats use

drug. $\Theta(U_{1k}) = \frac{\sum_{k=1}^n p(k'|k)U_{1k'}}{\sum_{k=1}^n kN_k}$, which denotes the expectation that any given edge

points to an individual infected by drug-taking at time t . $N_k = S_k + U_{1k} + U_{2k}$ is the total number of individuals with degree k on the network. Assume that the complex network is an uncorrelated networks, thus the initial node is independent with the conditional probability, i.e $p(k'|k) = k'p(k')/\langle k \rangle$, $\langle k \rangle = \sum_{k=1}^n kp(k)$. Thus,

$$\Theta(U_{1k}) = \langle k \rangle^{-1} \frac{\sum_{k=1}^n kp(k)U_{1k}(t)}{\sum_{k=1}^n kN_k}.$$

According to (2.1), the total population with degree k is a constant. Dividing system (2.1) by N_k , and denoting $s_k = \frac{S_k}{N_k}$, $u_{1k} = \frac{U_{1k}}{N_k}$, $u_{2k} = \frac{U_{2k}}{N_k}$, we have

$$\begin{cases} \frac{ds_k}{dt} = -k\sigma s_k \Theta(u_{1k}) + \gamma_1 u_{1k} + \gamma_2 u_{2k}, \\ \frac{du_{1k}}{dt} = k\sigma s_k \Theta(u_{1k}) - (\gamma_1 + \delta_1) u_{1k} + \delta_2 u_{2k}, \\ \frac{du_{2k}}{dt} = \delta_1 u_{1k} - (\delta_2 + \gamma_2) u_{2k}, \end{cases} \quad (2.2)$$

where $\Theta(u_{1k}) = \frac{\sum_{k=1}^n kp(k)u_{1k}}{\langle k \rangle}$. Since $s_k + u_{1k} + u_{2k} = 1$, then (2.2) can be changed into

$$\begin{cases} \frac{du_{1k}}{dt} = (1 - u_{1k} - u_{2k})k\sigma \Theta(u_{1k}) - (\gamma_1 + \delta_1) u_{1k} + \delta_2 u_{2k}, \\ \frac{du_{2k}}{dt} = \delta_1 u_{1k} - (\delta_2 + \gamma_2) u_{2k}, \end{cases} \quad (2.3)$$

with the initial condition

$$u_{1k}(0) = u_{1k0} \geq 0, u_{2k}(0) = u_{2k0} \geq 0.$$

Due to [14], (2.3) is well-posed.

Theorem 2.1. *The solution of (2.3) is nonnegative and bounded, i.e. $0 \leq s_k \leq 1, 0 \leq u_{1k} \leq 1, 0 \leq u_{2k} \leq 1$. If $\Theta(0) > 0$, then $0 < s_k < 1, 0 < u_{1k} < 1, 0 < u_{2k} < 1$ for all $t > 0$.*

Proof. First, we prove $\Theta(t) > 0$ for all $t > 0$ if $\Theta(0) > 0$. From the second equation of (2.3), one can obtain

$$\begin{aligned} \frac{d\Theta}{dt} &= \langle k \rangle^{-1} \sigma \Theta \sum_{k=1}^n k^2 p(k) s_k - (\gamma_1 + \delta_1) \Theta + \delta_2 \langle k \rangle^{-1} \sum_{k=1}^n k p(k) u_{2k} \\ &\geq [\langle k \rangle^{-1} \sigma \sum_{k=1}^n k^2 p(k) s_k - (\gamma_1 + \delta_1)] \Theta. \end{aligned} \tag{2.4}$$

Solving (2.4), we have $\Theta(t) \geq \Theta(0) e^{[\langle k \rangle^{-1} \sigma \sum_{k=1}^n k^2 p(k) s_k - (\gamma_1 + \delta_1)] t} > 0$ if $\Theta(0) > 0$.

Next, we will show there exist a τ such that for all $0 < t \leq \tau, 0 < s_k < 1$. Note that $s_k(0) \geq 0$, there exist a τ such that $s_k(t) > 0$ for all $t \in (0, \tau)$. Now we prove $\tau = +\infty$. If it is not true, then there exists a $t_0 > \tau$, such that $s_k(t_0) = 0$. From the second equation of (2.1),

$$\begin{aligned} u'_{1k}(t) &= k \sigma s_k(t) \Theta(u_{1k}(t)) - (\gamma_1 + \delta_1) u_{1k}(t) + \delta_2 u_{2k}(t) \\ &\geq -(\gamma_1 + \delta_1) u_{1k}(t), \end{aligned}$$

then $u_{1k}(t) \geq u_{1k}(0) e^{(\gamma_1 + \delta_1)t} \geq 0$. Similarly, $u_{2k}(t) \geq 0$ for $t \in (0, t_0)$. Again using the first equation of (2.1), one obtains

$$s'_k(t_0) = \gamma_1 u_{1k}(t_0) + \gamma_2 u_{2k}(t_0) \geq 0.$$

From the definition of $t_0, s'_k(t_0) \leq 0$. If $s'_k(t_0) < 0$, then this directly leads to a contradiction. If $s'_k(t_0) = 0$, then $\gamma_1 u_{1k}(t_0) + \gamma_2 u_{2k}(t_0) = 0$. From the nonnegativity of u_{1k} and u_{2k} this implies $u_{1k}(t_0) = 0$ and $u_{2k}(t_0) = 0$, this also leads to a contradiction with $s_k(t_0) + u_{1k}(t_0) + u_{2k}(t_0) = 1$. Hence for all $t > 0$, then $s_k(t) > 0$. From the second equation of (2.1), we have $u_{1k} > 0$. Finally from the third equation of (2.1), we get $u_{2k}(t) > 0$ for all $t > 0$.

Summing up all the equation of (2.1), we have $n'_k = 0$, that is $n_k = s_k + u_{1k} + u_{2k} = 1$. Combining the positivity of s_k, u_{1k} and u_{2k} , we have $s_k < 1, u_{1k} < 1, u_{2k} < 1$ for all $t > 0$. \square

For the convenience, we denote

$$\begin{aligned} \Omega &= \{(s_1, s_2, \dots, s_n, u_{11}, u_{12}, \dots, u_{1n}, u_{21}, u_{22}, \dots, u_{2n})\}, \\ 0 &\leq s_k, u_{1k}, u_{2k} \leq 1, \sum_{k=1}^n (s_k + u_{1k} + u_{2k}) = 1\}, \\ \overset{\circ}{\Omega} &= \{(s_1, s_2, \dots, s_n, u_{11}, u_{12}, \dots, u_{1n}, u_{21}, u_{22}, \dots, u_{2n}) \mid \sum_{k=1}^n k p(k) u_{1k} > 0\}, \\ \partial\Omega &= \Omega / \overset{\circ}{\Omega}. \end{aligned}$$

From Theorem 2.1, $\Omega, \overset{\circ}{\Omega}$ and $\partial\Omega$ are the positive invariant omega sets of (2.1).

3. Threshold and global stability of the drug free equilibrium

In this section, the basic reproduction number R_0 is obtained by the next-generation matrix. Using the method introduced by Van den Driessche and Watmough [17], let F and V be the $2n \times 2n$ matrix defined by

$$F = \left[\frac{\partial \mathcal{F}}{\partial x_i} \right]_{2n \times 2n}, \quad V = \left[\frac{\partial \mathcal{V}}{\partial x_i} \right]_{2n \times 2n},$$

where $x = (u_{11}, u_{12}, \dots, u_{1n}, u_{21}, u_{22}, \dots, u_{2n})$. Then F is non-negative and V is a non-singular M-matrix. Hence the reproduction number $R_0 = \rho(FV^{-1})$, where

$$F = \begin{pmatrix} A_{11} & A_{12} \\ A_{21} & A_{22} \end{pmatrix}, \quad V = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}, \tag{3.1}$$

here

$$A_{12} = \begin{pmatrix} \sigma \frac{p(1)}{\langle k \rangle} & \sigma \frac{2p(2)}{\langle k \rangle} & \dots & \sigma \frac{np(n)}{\langle k \rangle} \\ 2\sigma \frac{p(1)}{\langle k \rangle} & 2\sigma \frac{2p(2)}{\langle k \rangle} & \dots & 2\sigma \frac{np(n)}{\langle k \rangle} \\ & & \dots & \dots \\ n\sigma \frac{p(1)}{\langle k \rangle} & n\sigma \frac{2p(2)}{\langle k \rangle} & \dots & n\sigma \frac{np(n)}{\langle k \rangle} \end{pmatrix}$$

and

$$B_{11} = (\gamma_1 + \delta_1)\text{diag}(1, 1, \dots, 1), \quad B_{21} = B_{43} = -\delta_2\text{diag}(1, 1, \dots, 1),$$

$$B_{22} = -\delta_1\text{diag}(1, 1, \dots, 1), \quad B_{33} = (\delta_2 + \gamma_2)\text{diag}(1, 1, \dots, 1).$$

Other matrixes which are not special listed are zero matrixes with according dimensions. Denote the basic reproduction number is

$$R_0 = \frac{\langle k^2 \rangle}{\langle k \rangle} \frac{\sigma(\delta_2 + \gamma_2)}{(\delta_2 + \gamma_2)(\delta_1 + \gamma_1) - \delta_2\delta_1}.$$

Then it is easy to compute that $R_0 = \rho(FV^{-1})$, ρ is the radius spectrum of FV^{-1} . Due to [17], one gets the following theorem.

Theorem 3.1. *If $R_0 < 1$, then the drug-free equilibrium E_0 of (2.3) is locally asymptotically stable.*

Theorem 3.2. *When $R_0 < 1$, the drug-free equilibrium E_0 is globally asymptotically stable.*

Proof. Note that $s_k \leq 1$, it follows from (2.3), one can obtain

$$\begin{cases} \frac{du_{1k}}{dt} \leq k\sigma\Theta(u_{1k}) - (\gamma_1 + \delta_1)u_{1k} + \delta_2u_{2k}, \\ \frac{du_{2k}}{dt} = \delta_1u_{1k} - (\delta_2 + \gamma_2)u_{2k}. \end{cases} \tag{3.2}$$

Consider the following auxiliary system

$$\begin{cases} \frac{du_{1k}}{dt} = k\sigma\Theta(u_{1k}) - (\gamma_1 + \delta_1)u_{1k} + \delta_2u_{2k}, \\ \frac{du_{2k}}{dt} = \delta_1u_{1k} - (\delta_2 + \gamma_2)u_{2k}, \end{cases} \quad (3.3)$$

(3.3) can be written as

$$\frac{dx}{dt} = (F - V)x, \quad (3.4)$$

where F and V are defined as (3.1), $x = (u_{1k}(t), u_{2k}(t))^T$. It follows from [14] $\lim_{t \rightarrow \infty} x(t) = 0$ when $R_0 < 1$. Because of the comparison principle, thus

$$\lim_{t \rightarrow \infty} u_{1k}(t) = \lim_{t \rightarrow \infty} u_{2k}(t) = 0.$$

Hence, the drug free equilibrium E_0 is globally asymptotically stable. \square

Remark 3.1. From the expression of R_0 , then

$$\lim_{k \rightarrow \infty} R_0 = \lim_{k \rightarrow \infty} \frac{\langle k^2 \rangle}{\langle k \rangle} \frac{\sigma(\delta_2 + \gamma_2)}{(\delta_2 + \gamma_2)(\delta_1 + \gamma_1) - \delta_2\delta_1} = +\infty.$$

Hence, if the network is large enough, the basic reproduce number $R_0 \gg 1$, the drug transmission always spreads in some region.

4. Existence and stability of the endemic equilibrium

In this section we discuss the existence and the stability of the endemic equilibrium E^* . To get the endemic equilibrium $E^* = (u_{1k}^*, u_{2k}^*)$, then u_{1k}^*, u_{2k}^* satisfy

$$\begin{cases} 0 = k\sigma(1 - u_{1k}^* - u_{2k}^*)\Theta^* - (\gamma_1 + \delta_1)u_{1k}^* + \delta_2u_{2k}^*, \\ 0 = \delta_1u_{1k}^* - (\delta_2 + \gamma_2)u_{2k}^*, \end{cases} \quad (4.1)$$

where $\Theta^* = \frac{\sum_{k=1}^n kp(k)u_{1k}^*}{\langle k \rangle}$. From the second equation of (4.1), then $u_{2k}^* = \frac{\delta_1}{\delta_2 + \gamma_2}u_{1k}^*$. Substitute it into the first equation, it leads to

$$u_{1k}^* = \frac{k\sigma\Theta^*}{k\sigma(1 + \frac{\delta_1}{\delta_2 + \gamma_2})\Theta^* + \delta_1 + \gamma_1 - \frac{\delta_1\delta_2}{\delta_2 + \gamma_2}}. \quad (4.2)$$

Multiplying $\frac{kp(k)}{\langle k \rangle}$ on both side of (4.2) and summing up, one obtains

$$\Theta^* = \frac{1}{\langle k \rangle} \sum_{k=1}^n \frac{k^2\sigma\Theta^*}{k\sigma(1 + \frac{\delta_1}{\delta_2 + \gamma_2})\Theta^* + \delta_1 + \gamma_1 - \frac{\delta_1\delta_2}{\delta_2 + \gamma_2}} \triangleq f(\Theta(u_{1k}^*)). \quad (4.3)$$

(4.3) is a consistency equation. A non-zero stationary prevalence can be obtained when the right-hand side and the left-hand side of (4.3), expressed as functions of

$\Theta(u_{1k}^*)$, cross in the interval $0 < \Theta(u_{1k}^*) \leq 1$, allowing a nontrivial solution. It is easy to know that

$$\begin{aligned} \frac{df(\Theta(u_{1k}^*))}{d\Theta(u_{1k}^*)} \Big|_{\Theta(u_{1k}^*)=0} &= R_0 > 1, \\ \frac{d^2f(\Theta(u_{1k}^*))}{d\Theta(u_{1k}^*)^2} &= -\frac{2\sigma^2(\delta_1 + \gamma_1 - \frac{\delta_1\delta_2}{\delta_2+\gamma_2})(1 + \frac{\delta_1}{\delta_2+\gamma_2})}{\langle k \rangle}, \\ \sum_{k=1}^n \frac{k^3 p(k)}{[k\sigma(1 + \frac{\delta_1}{\delta_2+\gamma_2})\Theta^* + \gamma_1 + \delta_1 - \frac{\delta_1\delta_2}{\delta_2+\gamma_2}]^3} &< 0. \end{aligned}$$

There exists an uniqueness solution of (4.3), and thus (4.3) has an uniqueness solution $0 < u_{1k}^* < 1$.

Theorem 4.1. *If $R_0 > 1$, (4.1) has the endemic equilibrium E^* and $0 < s_k < 1, 0 < u_{1k}^* < 1, 0 < u_{2k} < 1$.*

Theorem 4.2. *If $R_0 > 1$, (2.3) is persistent with respect $(\partial\Omega, \overset{\circ}{\Omega})$.*

Proof. Under the assumption of Theorem 3.2, E_0 is a covering of Ω , which is isolate and is acyclic. Then we just need to prove $E^s(E_0) \cap \overset{\circ}{\Omega} = \emptyset$, where $E^s(E_0)$ is the stable manifold of E_0 . Since the global stability of E_0 , there exists a T_1 , for all $t > T_1$, one obtains

$$1 - \varepsilon \leq s_k \leq 1 + \varepsilon, 0 \leq u_{1k} \leq \varepsilon, 0 \leq u_{2k} \leq \varepsilon.$$

Then (2.3) leads to the follows inequalities

$$\begin{cases} \frac{du_{1k}(t)}{dt} \geq k(1 - \varepsilon)\sigma\Theta(u_{1k}) - (\gamma_1 + \delta_1)u_{1k} + \delta_2u_{2k}, \\ \frac{du_{2k}(t)}{dt} = \delta_1u_{1k} - (\gamma_2 + \delta_2)u_{2k}. \end{cases} \tag{4.4}$$

Consider the following auxiliary system

$$\begin{cases} \frac{du_{1k}(t)}{dt} = k(1 - \varepsilon)\sigma\Theta(u_{1k}) - (\gamma_1 + \delta_1)u_{1k} + \delta_2u_{2k}, \\ \frac{du_{2k}(t)}{dt} = \delta_1u_{1k} - (\gamma_2 + \delta_2)u_{2k}. \end{cases} \tag{4.5}$$

Let $x = (u_{1k}, u_{2k})$, (4.5) can be written as

$$\frac{dx}{dt} = (F(\varepsilon) - V)x, \tag{4.6}$$

where $F(\varepsilon)$ is the function substituting 1 with $1 - \varepsilon$ in F , and V is defined by (3.1). Since $R_0 > 1$ and the arbitrary of ε , then the spectral radius of $F(\varepsilon) - V$ is larger than zero. Then $x \rightarrow +\infty$ as $t \rightarrow +\infty$. With the comparison of principle, $u_{1k} \rightarrow +\infty$ and $u_{2k} \rightarrow +\infty$ as $t \rightarrow +\infty$. This is a contradiction with the boundness of u_{1k}, u_{2k} . \square

Theorem 4.3. *If $R_0 > 1$, and there exists a k such that $k \leq \frac{\gamma_2\Theta^*}{\delta_1\delta_2s_k^*}$, then the endemic equilibrium E^* is globally asymptotically stable.*

Proof. From the third equation of (2.3), one can obtain

$$\begin{aligned} \frac{d\Theta}{dt} &= \frac{\sum_{k=1}^n kp(k)u'_{1k}}{\langle k \rangle} \\ &= \langle k \rangle^{-1} \sum_{k=1}^n kp(k)[k\sigma s_k\Theta - (\mu + \delta)u_{1k} + \delta_2 u_{2k}] \\ &= \langle k \rangle^{-1} \sigma\Theta \sum_{k=1}^n k^2 p(k)s_k - (\mu + \delta)\Theta + \delta_2 \langle k \rangle^{-1} \sum_{k=1}^n kp(k)u_{2k}. \end{aligned} \quad (4.7)$$

Define a Lyapunov function

$$V = \frac{1}{2} \frac{1}{\langle k \rangle} \sum_{k=1}^n \frac{kp(k)}{s_k^*} [(s_k - s_k^*)^2 + \frac{\gamma_2}{\delta_1} (u_{2k} - u_{2k}^*)^2] + \Theta - \Theta^* \ln \Theta.$$

Then the derivative of V along the solution of (2.1) is

$$\begin{aligned} V' &= \frac{1}{\langle k \rangle} \sum_{k=1}^n \frac{kp(k)}{s_k^*} [(s_k - s_k^*)s'_k + \frac{\gamma_2}{\delta_1} (u_{2k} - u_{2k}^*)u'_{2k}] + (1 - \frac{\Theta^*}{\Theta})\Theta' \\ &= \frac{1}{\langle k \rangle} \sum_{k=1}^n \frac{kp(k)}{s_k^*} [(s_k - s_k^*)(-\sigma k\Theta + \gamma_1 u_{1k} + \gamma_2 u_{2k}) + \frac{\gamma_2}{\delta_1} (u_{2k} - u_{2k}^*)(\delta_1 u_{1k} \\ &\quad - (\delta_2 + \gamma_2)u_{2k})] + (1 - \frac{\Theta^*}{\Theta})[\langle k \rangle^{-1} \sigma\Theta \sum_{k=1}^n k^2 p(k)s_k \\ &\quad - (\mu + \delta_1)\Theta + \delta_2 \langle k \rangle^{-1} \sum_{k=1}^n kp(k)u_{2k}] \\ &= -\frac{1}{\langle k \rangle} \sum_{k=1}^n k^2 p(k)/s_k^* (s_k - s_k^*)^2 - \frac{\gamma_2}{\delta_1 \langle k \rangle} \sum_{k=1}^n kp(k)/s_k^* (u_{2k} - u_{2k}^*)^2 \\ &\quad + \frac{\delta_2}{\Theta^* \langle k \rangle} \sum_{k=1}^n kp(k)(\Theta - \Theta^*)(u_{2k} - u_{2k}^*) \\ &\quad - \frac{\gamma_2}{\delta_1 \langle k \rangle} \sum_{k=1}^n kp(k)/s_k^* (u_{1k} - u_{1k}^*)(u_{2k} - u_{2k}^*) \\ &\quad - \frac{\gamma_2}{\langle k \rangle} \sum_{k=1}^n kp(k) \frac{u_{2k}}{\Theta} (\Theta - \Theta^*)^2 \\ &\quad - (\delta_1 + \delta_2 + \gamma_2) \frac{\gamma_2}{\langle k \rangle} \sum_{k=1}^n kp(k)/s_k^* (u_{2k} - u_{2k}^*)^2 \\ &= -\frac{1}{\langle k \rangle} \sum_{k=1}^n k^2 p(k)/s_k^* (s_k - s_k^*)^2 - \frac{\gamma_2}{\delta_1 \langle k \rangle} \sum_{k=1}^n kp(k)/s_k^* (u_{2k} - u_{2k}^*)^2 \\ &\quad + \frac{1}{\langle k \rangle} \sum_{k=1}^n kp(k) (\frac{k\delta_2}{\Theta^*} - \frac{\gamma_2}{\delta_1 s_k^*}) (u_{1k} - u_{1k}^*) (u_{2k} - u_{2k}^*) \\ &\quad - \frac{\gamma_2}{\langle k \rangle} \sum_{k=1}^n kp(k) \frac{u_{2k}}{\Theta} (\Theta - \Theta^*)^2 \end{aligned}$$

$$-(\delta_1 + \delta_2 + \gamma_2) \frac{\gamma_2}{\langle k \rangle} \sum_{k=1}^n kp(k)/s_k^*(u_{2k} - u_{2k}^*)^2. \quad (4.8)$$

If there exists a k such that $k \leq \frac{\gamma_2 \Theta^*}{\delta_1 \delta_2 s_k^*}$, then (4.8) is less than 0. Since $V' = 0$ holds if and only if $s_k = s_k^*, u_{1k} = u_{1k}^*, u_{2k} = u_{2k}^*$ for $k = 1, 2, \dots, n$. Hence, the largest invariant set in Ω for $V' = 0$ is the singleton $\{E^*\}$. Due to [6], the endemic equilibrium E^* is globally asymptotically stable. \square

Remark 4.1. For the condition of Theorem 4.3, if $k \leq \frac{\gamma_2 \Theta^*}{\delta_1 \delta_2 s_k^*}$, that is $\frac{k\delta_2}{\Theta^*} - \frac{\gamma_2}{\delta_1 s_k^*} \leq 0$, then the derivative of V is

$$\begin{aligned} V' &\leq -\frac{1}{\langle k \rangle} \sum_{k=1}^n k^2 p(k)/s_k^*(s_k - s_k^*)^2 - \frac{\gamma_2}{\langle k \rangle} \sum_{k=1}^n kp(k) \frac{u_{2k}}{\Theta} (\Theta - \Theta^*)^2 \\ &\quad - \left(\frac{k\delta_2}{2\Theta^*} + \frac{\gamma_2}{2\delta_1 s_k^*} + \delta_1 + \delta_2 + \gamma_2 \right) \frac{1}{\langle k \rangle} \sum_{k=1}^n kp(k)(u_{2k} - u_{2k}^*)^2 \\ &\leq 0. \end{aligned}$$

If $k \geq \frac{\gamma_2 \Theta^*}{\delta_1 \delta_2 s_k^*}$, that is $\frac{k\delta_2}{\Theta^*} - \frac{\gamma_2}{\delta_1 s_k^*} \geq 0$, then the derivative of V is

$$\begin{aligned} V' &\leq -\frac{1}{\langle k \rangle} \sum_{k=1}^n k^2 p(k)/s_k^*(s_k - s_k^*)^2 - \frac{\gamma_2}{\langle k \rangle} \sum_{k=1}^n kp(k) \frac{u_{2k}}{\Theta} (\Theta - \Theta^*)^2 \\ &\quad + \left(\frac{k\delta_2}{2\Theta^*} - \frac{\gamma_2}{\delta_1 s_k^*} (3/2 + \delta_1 + \delta_2 + \gamma_2) \right) \frac{1}{\langle k \rangle} \sum_{k=1}^n kp(k)(u_{2k} - u_{2k}^*)^2. \end{aligned}$$

Therefore, if $\frac{k\delta_2}{\Theta^*} \leq \frac{2\gamma_2}{\delta_1 s_k^*} (3/2 + \delta_1 + \delta_2 + \gamma_2)$, then $V' \leq 0$.

Hence, if $k \leq \frac{2\gamma_2 \Theta^*}{\delta_1 \delta_2 s_k^*} (3/2 + \delta_1 + \delta_2 + \gamma_2)$, then the endemic equilibrium E^* is globally asymptotically stable.

5. Sensitivity Analysis and Simulation

In this section, from Theorem 3.2 and Theorem 4.2, the reproduction number R_0 is a key threshold for the control of the disease. It is necessary for R_0 to the terms of transmission parameters to do sensitive analysis. Thus, the reproduction number is changed according with the single parameter defined as follows:

$$\begin{aligned} \frac{\partial R_0}{\partial \sigma} &= \frac{\delta_2 + \gamma_2}{\gamma_1(\delta_1 + \delta_2) + \gamma_2 \delta_1} \frac{\langle k^2 \rangle}{\langle k \rangle} > 0, \\ \frac{\partial R_0}{\partial \gamma_1} &= -\sigma \frac{(\delta_1 + \delta_2)(\delta_2 + \gamma_2)}{[\gamma_1(\delta_1 + \delta_2) + \gamma_2 \delta_1]^2} \frac{\langle k^2 \rangle}{\langle k \rangle} > 0, \\ \frac{\partial R_0}{\partial \gamma_2} &= \sigma \frac{\gamma_1(\delta_1 + \delta_2) - \delta_1 \delta_2}{[\gamma_1(\delta_1 + \delta_2) + \gamma_2 \delta_1]^2} \frac{\langle k^2 \rangle}{\langle k \rangle}, \\ \frac{\partial R_0}{\partial \delta_1} &= -\sigma \frac{(\delta_1 + \delta_2)(\gamma_1 + \gamma_2)}{[\gamma_1(\delta_1 + \delta_2) + \gamma_2 \delta_1]^2} \frac{\langle k^2 \rangle}{\langle k \rangle} < 0, \\ \frac{\partial R_0}{\partial \delta_2} &= \sigma \frac{\gamma_1(\delta_1 - \gamma_2) + \gamma_2 \delta_1}{[\gamma_1(\delta_1 + \delta_2) + \gamma_2 \delta_1]^2} \frac{\langle k^2 \rangle}{\langle k \rangle}. \end{aligned} \quad (5.1)$$

From the computation results, decreasing transmission rate, improving the cure rate for the drug-taking and detoxification rate are helpful to protect the outbreak of the drug transmission. From the medical knowledge, the forcing a drug-taking to give up the drug behavior is more prone than curing a drug-taking back to a susceptible person, i.e. $\delta_1 > \gamma_1$. So cutting the way for the drug detoxification to contact with the drug-taking is efficient way to control the drug transmission. For the cure level for the drug detoxification, this is a complex problem for control of the drug behavior. If the cure rate for the drug-taking is large enough, improving the treatment for the drug detoxification is harmful for control the disease. While the cure rate for the drug-taking is small enough and the detoxification is large enough, improving the treatment for the drug detoxification is helpful for control the drug transmission. For the importance of R_0 between the two parameters, elasticity is a powerful tool to help us to realize it. According to the definition of elasticity, the elasticity of R_0 to the parameters, $\sigma, \gamma_1, \gamma_2, \delta_1$ and δ_2 are

$$\begin{aligned}
 E_{R_0}^\sigma &= \frac{\sigma}{R_0} \frac{\partial R_0}{\partial \sigma} = 1, \\
 E_{R_0}^{\gamma_1} &= \frac{\gamma_1}{R_0} \frac{\partial R_0}{\partial \gamma_1} = -\frac{\gamma_1(\delta_1 + \delta_2)}{\gamma_1(\delta_1 + \delta_2) + \gamma_2\delta_1}, \\
 E_{R_0}^{\gamma_2} &= \frac{\gamma_2}{R_0} \frac{\partial R_0}{\partial \gamma_2} = \frac{\gamma_2[\gamma_1(\delta_1 + \delta_2) - \delta_1\delta_2]}{(\gamma_1(\delta_1 + \delta_2) + \gamma_2\delta_1)(\delta_2 + \gamma_2)}, \\
 E_{R_0}^{\delta_1} &= \frac{\delta_1}{R_0} \frac{\partial R_0}{\partial \delta_1} = -\frac{\delta_1(\delta_1 + \delta_2)(\gamma_1 + \gamma_2)}{(\gamma_1(\delta_1 + \delta_2) + \gamma_2\delta_1)(\delta_2 + \gamma_2)}, \\
 E_{R_0}^{\delta_2} &= \frac{\delta_2}{R_0} \frac{\partial R_0}{\partial \delta_2} = \frac{\delta_2[\gamma_1(\delta_1 - \gamma_2) + \gamma_2\delta_1]}{(\gamma_1(\delta_1 + \delta_2) + \gamma_2\delta_1)(\delta_2 + \gamma_2)}.
 \end{aligned}
 \tag{5.2}$$

From these expressions, it follows that changing $\sigma, \gamma_1, \gamma_2, \delta_1$ and δ_2 have the some effect on the basic reproduction number R_0 . For the convenience, we rewrite $E^i = |E_{R_0}^i|, i = \sigma, \gamma_1, \gamma_2, \delta_1, \delta_2$. What's more, it is easy to see that $E^\sigma > E^{\gamma_1}$ and $E^{\delta_1} > E^{\delta_2}$. If $\gamma_2[\gamma_1(\delta_1 + \delta_2) - \delta_1\delta_2] > \delta_1(\delta_1 + \delta_2)(\gamma_1 + \gamma_2)$, then $E^\sigma > E^{\gamma_1} > E^{\gamma_2} > E^{\delta_1} > E^{\delta_2}$. Cutting the way that the susceptible individuals contact the group of the drug-taking is most effective method for control the drug transmission. While the effect for cutting the way that the drug detoxification again contact the group of the drug-taking is weaker than the way for the susceptible. If $\gamma_1(\delta_1 + \delta_2) < \delta_1\delta_2$, then $E^{\delta_2} > E^{\gamma_2}$. This may lead to $E^{\delta_1} > E^{\delta_2} > E^{\gamma_2} > E^\sigma > E^{\gamma_1}$ which means that improving the detoxification rate is the best control method for the drug transmission. In order

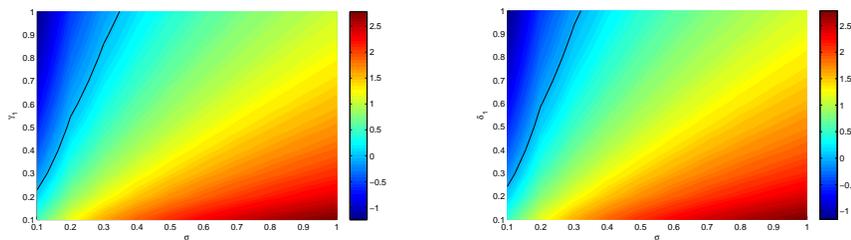


Figure 1. The logarithm of the basic reproduction number of system (2.1). $\log R_0$ is shown (a) when γ_1 is increased from 0 to 1 and σ is varied from 0 to 1 when $\delta_1 = 0.1$; (b) when δ_1 is increased from 0 to 1 and σ is varied from 0 to 1 when $\gamma_1 = 0.1$.

to illustrate these results, we set $m = 3, n = 100, \gamma_2 = 2, \delta_2 = 0.2, p(k) = 2m^2k^{-3}$. Figure 4 (a) gives R_0 relations with the transmission rate σ and cure rate γ_1 for the drug-taking. It says that the basic reproduction number R_0 decreases as increase as the cure rate γ_1 and decreasing the transmission rate σ . Visually the transmission rate σ has the greater effect than the cure rate γ_1 . Fig 5.1 (b) shows the relations of the reproduction number R_0 with the transmission rate σ and the detoxification rate δ_1 . It shows that the transmission rate has the greater effect than the detoxification rate.

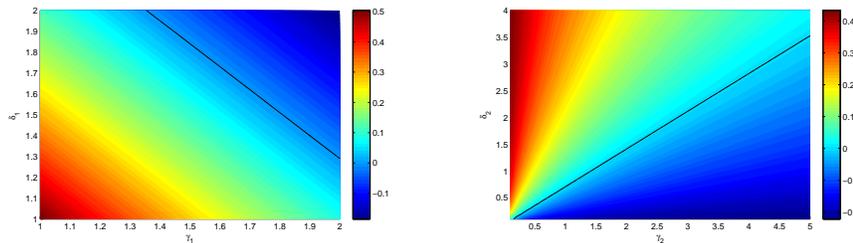


Figure 2. The logarithm of the basic reproduction number of system (2.1). $\log R_0$ is shown (a) when γ_1 is increased from 1 to 2 and δ_1 is varied from 1 to 2 if $\gamma_2 = 2, \delta_2 = 0.2$; (b) when γ_2 is increased from 0 to 5 and δ_2 is varied from 0 to 4 if $\gamma_1 = 0.1, \delta_1 = 0.1$.

Figure 2 (a) shows the relations of the reproduction number R_0 with the cure rate γ_1 and detoxification rate δ_1 . It shows that the cure rate almost has same effect than the detoxification rate. (b) shows that the basic reproduction number R_0 is larger than 1 even if the cure rate γ_2 for the drug detoxification is large while the temptation rate δ_2 is large enough. The effective method for control the drug transmission is improving the treatment rate γ_2 for the drug detoxification and decreasing the temptation rate δ_2 . Temptation rate has the greater effect than the treatment rate γ_2 .

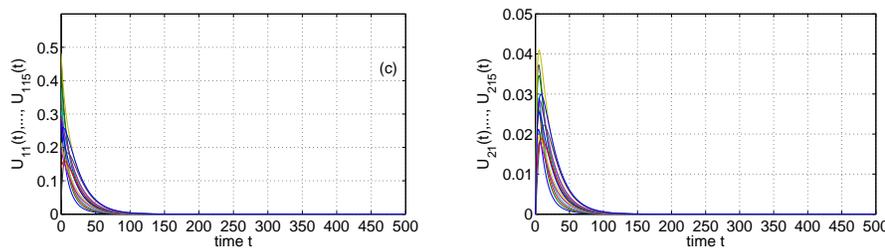


Figure 3. Simulated results of system (2.3). (a) The time evolution of the drug-taking node for an initial condition. (b) The time evolution of the drug detoxification node for an initial condition.

In order to illustrate the theoretical results, set $\sigma = 0.01, \gamma_1 = 0.1, \gamma_2 = 0.2, \delta_1 = 0.05, \delta_2 = 0.2$. We choose BA scale-free network with $m = 3, N = 100$. Then $R_0 = 0.5944 < 1$. According to Theorem 3.2, disease-free equilibrium is globally stable. (See Figure 3).

If we enlarge the transmission rate $\sigma = 0.02$. Then $R_0 = 1.1888 > 1$. According to Theorem 4.3, then the endemic equilibrium E^* is globally asymptotically stable. (See Figure 4).

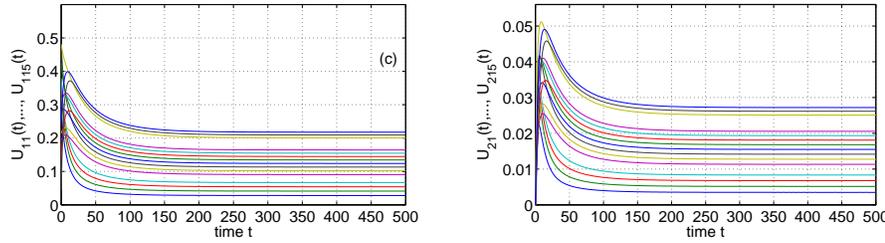


Figure 4. Simulated results of system (2.3). (a) The time evolution of the drug-taking node for an initial condition. (b) The time evolution of the drug detoxification node for an initial condition.

6. Discussion

In this paper, a heroin epidemic model on complex network is investigated. The threshold for extinction and persistence of the disease is obtained. The global stability of the equilibria is also presented. Sensitivity of the basic reproduction number to the parameters in system (2.3) is described. Some simulations illustrate our results. Decreasing the transmission rate and improving the detoxification rate are the effective methods for control the drug transmission spread. This means enhancing the propaganda for the drug transmission and increasing the compulsory treatment measures for the drug-taking individuals are the effective measures for control the drug transmission spread. From the above discussion, if we want to cut the spread of the drug transmission, one must decrease the transmission rate below the critical value $\sigma^{critical} = \frac{\langle k \rangle}{\langle k^2 \rangle} [\delta_1 + \gamma_1 - \frac{\delta_2 \delta_1}{\delta_2 + \gamma_2}]$.

Combining the knowledge of disease transmissibility, complex network structures with a good strategy is useful to mitigate the effects of a heroin epidemic outbreak. Because of the complexities of the model, we just discuss a heroin epidemic model on a static complex network. The population maintains some level, it is a constant. In fact, the population should be varying due to the disease death rate and the input of the population. This leads that one node of the network is lost or added, then the structure of the network will be change. In addition, the complex dynamics such as oscillations and chaotic attractors of these models inducing by complex network are also very interesting. Heroin mathematical epidemic models are useful to design public health policies. Without a doubt estimating model parameters is realistic. We will refer some heroin epidemic database and then apply it to the model. These may be our future work.

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