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# Dynamics of an SAITS alcoholism model on unweighted and weighted networks<sup>\*</sup>

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#### HIGHLIGHTS

- A novel SAITS alcoholism model on unweighted and weighted networks is introduced.
- The basic reproduction number, existence and stability of equilibria are derived.
- Numerical simulations are performed.

#### ARTICLE INFO

Article history: Received 9 August 2017 Received in revised form 15 November 2017 Available online 4 January 2018

Keywords: Alcoholism Unweighted and weighted networks Equilibrium Stability

#### ABSTRACT

A novel SAITS alcoholism model on networks is introduced, in which alcoholics are divided into light problem alcoholics and heavy problem alcoholics. Susceptible individuals can enter into the compartment of heavy problem alcoholics directly by contacting with light problem alcoholics or heavy problem alcoholics and the heavy problem alcoholics who receive treatment can relapse into the compartment of heavy problem alcoholics are also considered. First, the dynamics of our model on unweighted networks, including the basic reproduction number, existence and stability of equilibria are studied. Second, the models with fixed weighted and adaptive weighted networks are introduced and investigated. At last, some simulations are presented to illustrate and extend our results. Our results show that it is very important to treat alcoholics to quit the drinking.

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#### 1. Introduction

Alcohol problem has become a very serious problem for young people with the development of society. Approximately 40% of US college students engage in drinking [1]. The proportion of students from ages 18–24 who are reported driving under the influence of alcohol also increase from 26.5% to 31.4% from 1998 to 2001 [2]. Drinking damages to the human body is huge and contributes to a number of human disease, such as cirrhosis, heart disease and so on [3–5].

Many scholars study alcohol problems or epidemic by constructing mathematical model [6–13]. Huo, Chen and Xiang [6] investigated a binge drinking model with time delay. Huo and Zhang [7] introduced a novel alcoholism model which involves impact of Twitter and studied the occurrence of backward, forward and Hopf bifurcations. Xiang, Song and Huo [8] addressed the global property of a drinking model with public health educational campaigns. Huo, Huang and Wang et al. [11] studied a new social epidemic model to depict alcoholism with media coverage. Xiang, Tang and Huo [12] introduced a classical of virus dynamics model with intracellular delay and humoral immunity.

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https://doi.org/10.1016/j.physa.2018.01.003 0378-4371/© 2018 Elsevier B.V. All rights reserved.







<sup>&</sup>lt;sup>†</sup> This work is supported by the NNSF of China (11461041), the NSF of Gansu Province of China (148RJZA024) and the Development Program for HongLiu Distinguished Young Scholars in Lanzhou University of Technology.

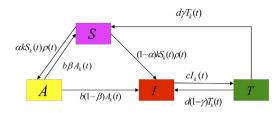


Fig. 1. Transfer diagram for alcoholism model.

Since the contact process of population cannot be uniform collision, many people begin to study the model on complex networks. The complex network is composed of a large number of nodes and a few edges connecting two nodes, in which each node represents an individual in the real system, and each edge between two nodes is a connection among individuals. If a node has *k* edges, we can say the individual has *k* neighborhoods, and define the node's degree is *k*. Many people have studied epidemic models on complex networks [14–32]. Pastor-Satorras and Vespiganai [16] introduced a dynamical model for the spreading of infectious on scale-free networks, and found the absence of an epidemic threshold and its associated critical behavior. Yang, Wang and Ren [17] proposed a modified SIR model, in which each node was assigned with an identical capability of active contacts, A, at each time step, and found that on scale-free networks, the density of the recovered individuals in the present model showed a threshold behavior. Liu and Zhang [23] investigated an SEIRS epidemic model on scale-free networks. Zhu, Fu and Chen [32] proposed a generalized epidemic model on complex networks. Their results explained why the heterogeneous connectivity patterns impacted the epidemic threshold.

Recently, mathematical models for alcoholism on complex networks have been investigated by many authors [33–35]. Motivated by the above works, we consider a new SAITS alcoholism model on scale-free networks. Comparing with the above models, we not only divide alcoholics into light problem alcoholics and heavy problem alcoholics but also take into account that susceptible individuals can enter into the compartment of heavy problem alcoholics directly by contacting with light problem alcoholics or heavy problem alcoholics. Furthermore, we consider the heavy problem alcoholics who receive treatment can relapse into the compartment of heavy problem alcoholics.

The rest of this paper is organized as follows: In the next section, a SAITS alcoholism model on scale-free networks is formulated and analyzed. In Section 3, the basic reproduction number and existence of equilibria are studied. In Section 4, stability analysis of the equilibria are given. In Section 5, the SAITS alcoholism model on weighted contact network is also introduced. Numerical simulations are illustrated in Section 6. In Section 7, we give the sensitivity analysis and the conclusion.

#### 2. Model formulation

The daily alcohol consumption for men is no more than 4 standard drinks, and for women is no more than 3 standard drinks. One "standard" drink contains roughly 14 grams of pure alcohol. The ceiling of "low-risk" alcohol consumption per week is 14 standard drinks for men and 7 standard drinks for women [36]. If a person whose alcohol consumption is more than daily or weekly drinking ceiling, he/she most likely develops into "abuse-alcohol" or "addicted-alcohol". In our model, each individual is represented by a node of the network and the edges are the connection between individuals. The whole population is divided into four compartments, namely: susceptibles S(t), refer to the people who do not drink or drink only moderately; the light problem alcoholics A(t), refer to the drinkers who drink beyond daily or weekly ceiling and drink 4 to 5 standard drinks per day; the heavy problem alcoholics I(t), refer to the drinkers who drink more than daily and weekly limits and drink more than 5 standard drinks per day; the treatments T(t), refer to the drinkers who receive treatment. We assume that each node in the k - th group has the same degree k, and share the same state. Correspondingly, they are compartmentalized into four groups with densities at time t, that is,  $S_k(t)$ ,  $A_k(t)$ ,  $I_k(t)$ , where the subscript k is the degree (k = 1, 2, ..., n). Transfer diagram for our model is described in Fig. 1.

Transfer diagram leads to the following system of ordinary differential equations:

$$\frac{dS_{k}(t)}{dt} = -kS_{k}(t)(\rho_{1}\theta_{1}(t) + \rho_{2}\theta_{2}(t)) + b\beta A_{k}(t) + d\gamma T_{k}(t), 
\frac{dA_{k}(t)}{dt} = \alpha kS_{k}(t)(\rho_{1}\theta_{1}(t) + \rho_{2}\theta_{2}(t)) - bA_{k}(t), 
\frac{dI_{k}(t)}{dt} = b(1 - \beta)A_{k}(t) + (1 - \alpha)kS_{k}(t)(\rho_{1}\theta_{1}(t) + \rho_{2}\theta_{2}(t)) 
+ d(1 - \gamma)T_{k}(t) - cI_{k}(t), 
\frac{dT_{k}(t)}{dt} = cI_{k}(t) - dT_{k}(t),$$
(2.1)

where *b*, *c*, *d* and  $\alpha$ ,  $\beta$ ,  $\gamma$  are nonnegative constants, and  $0 \le \alpha$ ,  $\beta$ ,  $\gamma \le 1$ .

In fact, we know that only the contacts between susceptible and the light or heavy problem alcoholics nodes have possible contributions in alcoholism transmission processes. A problem alcoholic node usually does not know whether its neighbors are "infected" in a real alcoholism transmission processes, so we assume that each problem alcoholic node will contact every neighbor once within one time step in our model.  $\theta_1(t)$  denotes the probability that a randomly selected neighbor of a given node is a light problem alcoholics. Then  $k\theta_1(t)$  is the expected number of light problem alcoholic neighbors of a susceptible node of degree k.  $\theta_2(t)$  denotes the probability that a randomly selected neighbor of a given node is a heavy problem alcoholics. Then  $k\theta_2(t)$  is the expected number of heavy problem alcoholic neighbors of a susceptible node of degree k. For uncorrelated networks [16,19], we have

$$\theta_1(t) = \sum_i p(i|k)A_i(t) = \langle k \rangle^{-1} \sum_i ip(i)A_i(t),$$
  

$$\theta_2(t) = \sum_i p(i|k)I_i(t) = \langle k \rangle^{-1} \sum_i ip(i)I_i(t).$$
(2.2)

Where,  $\langle k \rangle = \sum_i ip(i)$  denotes the mean degree. p(k) is the degree distribution. The conditional probability p(i|k) denotes the degree correlations that a node of degree k is connected to a node of degree i. In uncorrelated networks  $p(i|k) = \frac{ip(i)}{\langle k \rangle}$ .  $\theta_1(t)$  and  $\theta_2(t)$  are independent of k for uncorrelated networks. For simplicity, we set  $\rho(t) = \rho_1 \theta_1(t) + \rho_2 \theta_2(t)$ . The meanings of the parameters or variables in model (2.1) are as follows:

- (i)  $\rho(t)$  represents the loss rate of susceptible individuals due to contact with the light problem alcoholic and the heavy problem alcoholic, the portion  $\alpha \rho(t)$  turns into the light problem alcoholic compartment. The other turns into the heavy problem alcoholic compartment. Parameter  $\rho_1(\rho_2)$  is the infectious rate for a susceptible individual after a contact with a light problem alcoholic (a heavy problem alcoholic) one.
- (ii) *b* is the removal rate of the light problem alcoholic compartment, and  $\beta$  is the proportion of the light problem alcoholics that turn into susceptible. The other turns into the heavy problem alcoholic compartment.
- (iii) *c* is the removal rate of a heavy problem alcoholic compartment.
- (iv) *d* is the removal rate of the treatment compartment, and  $\gamma$  is the proportion of the treatment individuals that turn into susceptible. The other turns into the heavy problem alcoholic compartment.

Since  $\frac{d(S_k(t)+A_k(t)+I_k(t)+T_k(t))}{dt} = 0$ , the total individual number is constant. We assume that  $S_k(t)+A_k(t)+I_k(t)+T_k(t) = 1$ . The system (2.1) represents human population, it is reasonable to assume that all state variables and parameters are nonnegative for all  $t \ge 0$ . It is easy to know that the region

$$\Omega = \{ (S_k(t), A_k(t), I_k(t), T_k(t)) \in \mathbb{R}_+^{4n} | S_k(t) + A_k(t) + I_k(t) + T_k(t) \le 1, \\ k = 1, 2, \dots, n \},$$
(2.3)

is the positively invariant set for (2.1). Therefore, we consider the dynamics of system (2.1) in the set  $\Omega$  in this paper. Since  $S_k(t) = 1 - A_k(t) - I_k(t) - I_k(t)$ , it is sufficient to study the follow system:

$$\begin{cases} \frac{dA_{k}(t)}{dt} = \alpha k(1 - A_{k}(t) - I_{k}(t) - T_{k}(t))\rho(t) - bA_{k}(t), \\ \frac{dI_{k}(t)}{dt} = b(1 - \beta)A_{k}(t) + (1 - \alpha)k(1 - A_{k}(t) - I_{k}(t) - T_{k}(t))\rho(t) \\ + d(1 - \gamma)T_{k}(t) - cI_{k}(t), \end{cases}$$

$$(2.4)$$

$$\frac{dT_{k}(t)}{dt} = cI_{k}(t) - dT_{k}(t),$$

in the subspace

$$\Omega^* = \{ (A_k(t), I_k(t), T_k(t)) \in R^{3n}_+ | A_k(t) + I_k(t) + T_k(t) \le 1, k = 1, 2, \dots, n \}.$$

$$(2.5)$$

#### 3. The basic reproduction number and existence of equilibria

**Theorem 1.** Consider the system (2.4). Define

$$R_0 = \frac{\langle k^2 \rangle}{\langle k \rangle} \left[ \frac{\rho_1 \alpha}{b} + \frac{\rho_2 (1 - \alpha \beta)}{c \gamma} \right]$$

then the following statements hold:

(1) There always exists a alcohol-free equilibrium  $E^0 = \{(0, 0, 0)\}_k$ ;

(2) There is a unique alcoholism equilibrium  $E^* = \{(A_k^*, I_k^*, T_k^*)\}_k$  if  $R_0 > 1$ .

**Proof.** It is easy to know that the system (2.4) has a alcohol-free equilibrium  $E^0 = \{(0, 0, 0)\}_k$ .

To get the equilibrium solution  $(A_k^*, I_k^*, T_k^*)$ , we need to make the right side of system (2.4) equal to zero. Then the equilibrium  $(A_k^*, I_k^*, T_k^*)$  should satisfy

$$\begin{cases} \alpha k(1 - A_k(t) - I_k(t) - T_k(t))\rho(t) - bA_k(t) = 0, \\ b(1 - \beta)A_k(t) + (1 - \alpha)k(1 - A_k(t) - I_k(t) - T_k(t))\rho(t) \\ + d(1 - \gamma)T_k(t) - cI_k(t) = 0, \\ cI_k(t) - dT_k(t) = 0. \end{cases}$$
(3.1)

A direct calculation yields:

$$A_{k}^{*} = \frac{\alpha k \rho c d\gamma}{(c+d)H_{k} + c(\alpha k \rho + b)d\gamma},$$

$$I_{k}^{*} = \frac{dH_{k}}{(c+d)H_{k} + c(\alpha k \rho + b)d\gamma},$$

$$T_{k}^{*} = \frac{cH_{k}}{(c+d)H_{k} + c(\alpha k \rho + b)d\gamma},$$
(3.2)

where

$$H_k = k\rho[(\alpha k\rho + b)(1 - \alpha) + b(1 - \beta)\alpha - \alpha(1 - \alpha)k\rho],$$

and  $\rho \leq (\rho_1 + \rho_2)$ . Substituting  $A_k^*$  and  $I_k^*$  of (3.2) into  $\rho$ , we obtain an equation of the form  $\rho f(\rho) = 0$ , where

$$f(\rho) = 1 - \left[\frac{\rho_1 \alpha c d\gamma + \rho_2 db(1 - \alpha \beta)}{\langle k \rangle}\right] \sum \frac{i^2 p(i)}{(c + d)H_i + c(\alpha k\rho + b)d\gamma}$$

Since  $f'(\rho) > 0$  and  $f(\rho_1 + \rho_2) > 0$ , the equation  $f(\rho) = 0$  has a unique non-trivial solution if and only if f(0) < 0, i.e.

$$\frac{\langle k^2 \rangle}{\langle k \rangle} \left[ \frac{\rho_1 \alpha}{b} + \frac{\rho_2 (1 - \alpha \beta)}{c \gamma} \right] > 1,$$

where  $\langle k^2 \rangle = \sum i^2 p(i)$  is the diverging second moment. The proof is completed.

#### 4. Stability analysis of the equilibria

#### 4.1. The stability of the alcohol free equilibrium

**Theorem 2.** For system (2.4), the alcohol free equilibrium is globally asymptotically stable if  $R_0 < 1$ .

Proof. In order to use the comparison theorem to prove the global stability of the alcohol free equilibrium, the equations in (2.4) can be written in terms of

$$\begin{pmatrix} \frac{dA_k(t)}{dt} \\ \frac{dI_k(t)}{dt} \\ \frac{dT_k(t)}{dt} \end{pmatrix} = (F - V - U) \begin{pmatrix} A_k(t) \\ I_k(t) \\ T_k(t) \end{pmatrix},$$

where the matrices F, V, U are given by

$$\mathcal{F}(x) = \begin{pmatrix} \alpha k \rho(t)(1 - A_k - I_k - T_k) \\ 0 \end{pmatrix}_{3n \times 1}, \\ \mathcal{V}(x) = \begin{pmatrix} cI_k - (1 - \alpha)k\rho(t)(1 - A_k - I_k - T_k) - b(1 - \beta)A_k - d(1 - \gamma)T_k \\ dT_k - cI_k \end{pmatrix}_{3n \times 1}$$

The Jacobian matrices of  $\mathcal{F}(x)$  and  $\mathcal{V}(x)$  at the alcohol free equilibrium  $E^0$  are

$$F = D\mathcal{F}(E^{0}) = \begin{pmatrix} F_{11} & F_{12} & 0\\ 0 & 0 & 0\\ 0 & 0 & 0 \end{pmatrix}_{3n \times 3n}$$

$$V = DV (E^{0}) = \begin{pmatrix} V_{11} & 0 & 0 \\ V_{21} & V_{22} & V_{23} \\ 0 & V_{32} & V_{33} \end{pmatrix}_{3n \times 3n}^{,}$$

$$F_{11} = \frac{\alpha \rho_{1}}{\langle k \rangle} \begin{pmatrix} P(1) & 2P(2) & \cdots & nP(n) \\ 2P(1) & 2^{2}P(2) & \cdots & 2nP(n) \\ \vdots & \vdots & \ddots & \vdots \\ nP(1) & 2nP(2) & \cdots & n^{2}P(n) \end{pmatrix}_{n \times n}^{,}$$

$$F_{12} = \frac{\alpha \rho_{2}}{\langle k \rangle} \begin{pmatrix} P(1) & 2P(2) & \cdots & nP(n) \\ 2P(1) & 2^{2}P(2) & \cdots & 2nP(n) \\ \vdots & \vdots & \ddots & \vdots \\ nP(1) & 2nP(2) & \cdots & n^{2}P(n) \end{pmatrix}_{n \times n}^{,}$$

0 0 )

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 $\begin{aligned} V_{11} &= bI, V_{21} = [-b(1-\beta)]I - (1-\alpha)\rho_1 \frac{ijp(j)}{\langle k \rangle}, V_{22} = cI - (1-\alpha)\rho_2 \frac{ijp(j)}{\langle k \rangle}, \\ V_{23} &= [-d(1-\gamma)]I, V_{32} = -cI, V_{33} = dI, \end{aligned}$ 

$$U = \begin{pmatrix} \alpha k \rho(t) & \alpha k \rho(t) & \alpha k \rho(t) \\ (1 - \alpha) k \rho(t) & (1 - \alpha) k \rho(t) & (1 - \alpha) k \rho(t) \\ 0 & 0 & 0 \end{pmatrix}_{3n \times 3n},$$

where U is a nonnegative matrix, I is identity matrix and 0 is zero matrix. Thus

$$\begin{pmatrix} \frac{dA_k(t)}{dt} \\ \frac{dI_k(t)}{dt} \\ \frac{dT_k(t)}{dt} \end{pmatrix} \leq (F - V) \begin{pmatrix} A_k(t) \\ I_k(t) \\ T_k(t) \end{pmatrix}.$$

If  $R_0 < 1$ , which is equivalent to F - V having all its eigenvalues in the left half plane. It follows that the linearized differential inequality system is stable whenever  $R_0 < 1$ . So,  $A_k \rightarrow 0$ ,  $I_k \rightarrow 0$ ,  $T_k \rightarrow 0$ , as  $t \rightarrow \infty$ , for this linear ordinary differential equations system. Since it is a quasimonotone system, using a standard comparison theorem,  $A_k \rightarrow 0$ ,  $I_k \rightarrow 0$ ,  $T_k \rightarrow 0$ , for the nonlinear system for  $R_0 < 1$ , so that the alcohol free equilibrium is globally asymptotically stable in  $\Omega^*$  when  $R_0 < 1$ . The proof is completed.

#### 4.2. The persistence of system

In this subsection, at first, we introduce three lemmas to prove the persistence of the system (2.4).

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On the basis of Theorem 2, we suppose that  $A_1 = y_1, A_2 = y_2, ..., A_n = y_n, I_1 = y_{n+1}, I_2 = y_{n+2}, ..., I_n = y_{2n}, T_1 = y_{2n+1}, T_2 = y_{2n+2}, ..., T_n = y_{3n}$  and  $y = (y_1, ..., y_n, y_{n+1}, ..., y_{2n}, y_{2n+1}, ..., y_{3n})^T$ . System (2.4) can be written as follows

$$\frac{dy}{dt} = Ay + N(y),$$
  
where  $A = F - V,$   
$$N(y) = -\begin{pmatrix} \alpha \rho(y_1 + y_{n+1} + y_{2n+1}) \\ 2\alpha \rho(y_2 + y_{n+2} + y_{2n+2}) \\ \vdots \\ n\alpha \rho(y_n + y_{2n} + y_{3n}) \\ (1 - \alpha)\rho(y_1 + y_{n+1} + y_{2n+1}) \\ 2(1 - \alpha)\rho(y_2 + y_{n+2} + y_{2n+2}) \\ \vdots \\ n(1 - \alpha)\rho(y_n + y_{2n} + y_{3n}) \\ 0 \\ \vdots \\ 0 \end{pmatrix}_{3n \times 1}$$

$$A = \begin{pmatrix} A_{11} & A_{12} \\ A_{21} & A_{22} \end{pmatrix}_{3n \times 3n}$$

where

$$A_{11} = \begin{pmatrix} B_{11} & B_{12} \\ B_{21} & B_{22} \end{pmatrix}_{2n \times 2n},$$

where,

$$B_{11} = \begin{pmatrix} \alpha \rho_1 G_1 - b \cdots & \alpha \rho_1 G_n \\ \vdots & \vdots & \vdots \\ n \alpha \rho_1 G_1 & \cdots & n \alpha \rho_1 G_n - b \end{pmatrix}_{n \times n}, \\ B_{12} = \begin{pmatrix} \alpha \rho_2 G_1 & \cdots & \alpha \rho_2 G_n \\ \vdots & \vdots & \vdots \\ n \alpha \rho_2 G_1 & \cdots & n \alpha \rho_2 G_n \end{pmatrix}_{n \times n}, \\ B_{21} = \begin{pmatrix} (1 - \beta)b + (1 - \alpha)\rho_1 G_1 & \cdots & (1 - \alpha)\rho_1 G_n \\ \vdots & \vdots & \vdots \\ n \alpha \rho_1 G_1 & \cdots & (1 - \beta)b + (1 - \alpha)\rho_1 G_n \end{pmatrix}_{n \times n} \\ B_{22} = \begin{pmatrix} (1 - \alpha)\rho_2 G_1 - c & \cdots & (1 - \alpha)\rho_2 G_n \\ \vdots & \vdots & \vdots \\ n(1 - \alpha)\rho_2 G_1 & \cdots & n(1 - \alpha)\rho_2 G_n - c \end{pmatrix}_{n \times n}, \\ A_{12} = \begin{pmatrix} 0 & \cdots & 0 \\ \vdots & \vdots & \vdots \\ 0 & \cdots & 0 \\ d(1 - \gamma) & \cdots & 0 \\ \vdots & \vdots & \vdots \\ 0 & \cdots & d(1 - \gamma) \end{pmatrix}_{2n \times n} \\ A_{21} = \begin{pmatrix} 0 \cdots & 0 c \cdots & 0 \\ \vdots & \vdots & \vdots & \vdots \\ 0 & \cdots & 0 \end{pmatrix}_{n \times 2n}, \\ A_{22} = -dI, \end{cases}$$

and *I* is identity matrix. Define  $G(j) = \frac{jp(j)}{\langle k \rangle}$ .

**Lemma 1** ([37]). Let  $A = a_{ij}$  be an irreducible  $n \times n$  matrix and assume  $a_{ij} \ge 0$  whenever  $i \ne j$ . Then there exists an positive eigenvector  $\omega$  of A and the corresponding eigenvalue is S(A).

The stability modulus S(A) is defined by  $S(A) = max Re\lambda_i$ ,  $i = 1 \dots n$ , where  $\lambda_i$  are the eigenvalues of A.

**Lemma 2** ([37]). Consider the system

$$\frac{dy}{dt} = Ay + N(y),\tag{4.1}$$

where A is an  $n \times n$  matrix and N(y) is continuously differentiable in a region  $D \subset \mathbb{R}^n$ . Assume

- (i) the compact convex set  $C \subset D$  is positively invariant with respect to the system (4.1), and  $0 \in C$ ;
- (ii)  $\lim_{y\to 0} || N(y) || / || y || = 0;$
- (iii) there exist a constant r > 0 and a real eigenvector  $\omega$  of  $A^T$  such that  $(\omega \cdot y) \ge r \parallel y \parallel$  for all  $y \in C$ ;
- (iv)  $(\omega \cdot N(y)) \leq 0$  for all  $y \in C$ ;
- (v) y = 0 is the largest positively invariant set contained in  $H = \{y \in C | (\omega \cdot N(y)) = 0\}$ .

Then either y = 0 is globally asymptotically stable in C, or for any  $y_0 \in C - \{0\}$  the solution  $\varphi(t, y_0)$  of (4.1) satisfies

 $\liminf_{t\to\infty} \| \varphi(t,y_0) \| \ge e,$ 

e > 0 independent of  $y_0$ . Moreover, there exists a constant solution of (4.1),  $y = y^*$ ,  $y^* \in C - \{0\}$ .

**Theorem 3.** If  $R_0 > 1$ , the system (2.4) is permanent, and there exists a small constant  $\xi > 0$ , such that

 $\liminf_{t\to\infty} \{A_k(t), I_k(t), T_k(t)\}_{k=1}^n \geq \xi,$ 

where  $(A_k(t), I_k(t), T_k(t))$  is any solution of (2.4), satisfying (2.5), and  $A_k(0) > 0$ ,  $I_k(0) > 0$ ,  $T_k(0) > 0$ .

**Proof.** We will prove it by using the hypotheses of Lemma 2.

- (i) We can see the compact convex  $\Omega^*$  is a positively invariant set for system (2.4), and  $0 \in \Omega^*$ ;
- (ii) Using the limit rule and mean inequality, we can work out  $\lim_{y\to 0} || N(y) || / || y || = 0$ ;
- (iii) Apparently  $A_{11}^{T} = (a_{ij})_{2n \times 2n}$  is irreducible and  $a_{ij} \ge 0$ , for all  $i \ne j$ . Then from Lemma 1, there exists an eigenvector  $\omega = (\omega_1, \omega_2, \dots, \omega_{2n})$  of  $A_{11}^{T}$ , and the associated eigenvalue is  $S(A_{11}^{T})$ .  $S(A_{11}^{T}) > 0$ , if  $R_0 > 1$ . Let  $\omega_{2n+1} = \dots = \omega_{3n} = 0$ and  $\omega = (\omega_1, \omega_2, \dots, \omega_{3n})$ , then  $A^T \omega = S(A_{11}^{T})\omega$ . The vector  $\omega$  is the eigenvector of the matrix  $A^T$  that corresponds to the eigenvalue  $S(A_{11}^{T})$ . Assume  $r = \min_{1 \le i \le 2n} \{\omega_i\} > 0$ , for all  $y \in \Omega^*$ , then we obtain  $(\omega \cdot y) \ge r \parallel y \parallel$ ;
- (iv) As each item of N(y) is nonpositive and  $\omega \ge 0$ ,  $(\omega \cdot N(y)) \le 0$ ;
- (v) For system (2.4),  $H = \{y \in \Omega^* | (\omega \cdot N(y)) = 0\}$ . If  $y \in H$ , we can receive  $\frac{1}{\langle k \rangle} \sum_{i=1}^n i\omega_i (\rho_1 \sum_{k=1}^n kp(k)y_i + \rho_2 \sum_{k=1}^n kp(k)y_{n+i})(y_i + y_{n+i} + y_{2n+i}) = 0$  for all i = 1, 2, ..., n. Since  $\omega_1 > 0, k > 0$ , thus  $(\rho_1 \sum_{k=1}^n kp(k)y_i + \rho_2 \sum_{k=1}^n kp(k)y_{n+i})(y_i + y_{n+i} + y_{2n+i}) = 0$ . According to the system (2.4), we know y = 0 is the unique solution contained in H, so, y = 0 is the largest positively invariant set contained in  $H = \{y \in C | (\omega \cdot N(y)) = 0\}$ . All the hypotheses of Lemma 2 are satisfied. The proof is completed.

#### 5. The SAITS alcoholism model on weighted contact networks

In order to study the effect of the intimacy between individuals, we will introduce the modified SAITS models which are based on the model (2.1) with fixed weighted and adaptive weighted networks in this section.

#### 5.1. The SAITS model on fixed weighted networks

The modified SAITS model with fixed weighted networks can be described by the following:

$$\begin{cases} \frac{dS_k(t)}{dt} = -\frac{\lambda k S_k g(k)}{\langle kg(k) \rangle} (\theta_1(t) + \theta_2(t)) + b\beta A_k(t) + d\gamma T_k(t), \\ \frac{dA_k(t)}{dt} = \frac{\alpha \lambda k S_k g(k)}{\langle kg(k) \rangle} (\theta_1(t) + \theta_2(t)) - bA_k(t), \\ \frac{dI_k(t)}{dt} = b(1 - \beta) A_k(t) + \frac{(1 - \alpha) \lambda k S_k g(k)}{\langle kg(k) \rangle} (\theta_1(t) + \theta_2(t)) + d(1 - \gamma) T_k(t) \\ - cI_k(t), \\ \frac{dT_k(t)}{dt} = cI_k(t) - dT_k(t), \end{cases}$$
(5.1)

where  $S_k$ ,  $A_k$ ,  $I_k$ ,  $T_k$ , b, c, d,  $\alpha$ ,  $\beta$ ,  $\gamma$  have the same meaning with the standard SAITS model (2.1). We only change the contact transmission rate between susceptible people and light problem alcoholic or heavy problem alcoholic as  $\Theta_1$  or  $\Theta_2$ , where

$$\Theta_{1}(t) = \sum_{i} \lambda_{ik} \frac{\varphi_{1}(i)}{i} p(i|k) A_{i}(t) = \langle k \rangle^{-1} \sum_{i} \lambda_{ik} \varphi_{1}(i) p(i) A_{i}(t),$$
  

$$\Theta_{2}(t) = \sum_{i} \lambda_{ik} \frac{\varphi_{2}(i)}{i} p(i|k) I_{i}(t) = \langle k \rangle^{-1} \sum_{i} \lambda_{ik} \varphi_{2}(i) p(i) I_{i}(t).$$
(5.2)

The meanings of the parameters or variables in (5.2) are as follows:

- (i)  $\lambda_{ik}$  is the transmission rate from nodes with degree *i* to nodes with degree *k*.
- (ii)  $\varphi_1(i)(\varphi_2(i))$  is the infectivity of light problem alcoholic (heavy problem alcoholic) with degree *i*.
- (iii) The conditional probability p(i|k) denotes the degree correlations that a node of degree k is connected to a node of degree i. Considering the uncorrelated network,  $p(i|k) = \frac{ip(i)}{\langle k \rangle}$ , where  $\langle k \rangle = \sum_{i} ip(i)$  denotes the average degree of the network.

Making use of node's degrees to express the weights of edges is very common in many complex networks [33,38]. The weight between two nodes with degree *k* and *k'* may represent as a function of their degree. An example is  $\omega_{kk'} = \omega_0 (kk')^{\beta}$ , where  $\omega_0$  and  $\beta$  always change according to different network. Through the above analysis, we know  $\omega_{kk'}$  belongs to an

edge, thus, the strength of a node which also can be obtained by summing the weights of the links that connected to it  $(N_k = k \sum_{k'} p(k'|k) \omega_{kk'})$ . Now we use the function  $\omega(i, j) = g(i)g(j)$  to express the edge weights between two nodes. We assume that g(k) is an increasing function of k due to the nodes with more connections will be more powerful and gain more weights. So we can get the weight of a node with degree k, which is summing up the weights of links connected to it, hence,  $\phi(k) = k \sum_i p(i|k) \omega(i, k)$ . On uncorrelated networks,  $\phi(k) = kg(k) \langle kg(k) \rangle / \langle k \rangle$ . We assume that a total transmission rate  $\lambda i$  for each node with degree k, thus we can obtain the transmission by the link from the *i*-degree node to a *k*-degree node.

$$\lambda_{ik} = \lambda i \frac{\omega(i,k)}{\phi_i} = \frac{\lambda g(k) \langle k \rangle}{\langle kg(k) \rangle}.$$
(5.3)

Substituting (5.3) into (5.2), we obtain

$$\Theta_{1}'(t) = \frac{\lambda g(k)}{\langle kg(k) \rangle} \sum_{i} \varphi_{1}(i) p(i) A_{i}(t),$$

$$\Theta_{2}'(t) = \frac{\lambda g(k)}{\langle kg(k) \rangle} \sum_{i} \varphi_{2}(i) p(i) I_{i}(t).$$
(5.4)

Substituting (5.4) into (2.1), we obtain the fixed weight system (5.1). Where  $\theta_1(t) = \sum_i \varphi_1(i)p(i)A_i(t)$  and  $\theta_2(t) = \sum_i \varphi_2(i)p(i)I_i(t)$  in (5.1).

We denote that  $\theta(t) = \theta_1(t) + \theta_2(t)$ . Since  $S_k(t) = 1 - A_k(t) - I_k(t) - T_k(t)$ , it is sufficient to study the following system

$$\begin{cases} \frac{dA_k(t)}{dt} = \frac{\alpha\lambda k(1 - A_k(t) - I_k(t) - I_k(t))g(k)}{\langle kg(k) \rangle} \theta(t) - bA_k(t), \\ \frac{dI_k(t)}{dt} = \frac{(1 - \alpha)\lambda k(1 - A_k(t) - I_k(t) - T_k(t))g(k)}{\langle kg(k) \rangle} \theta(t) + b(1 - \beta)A_k(t) \\ + d(1 - \gamma)T_k(t) - cI_k(t), \end{cases}$$
(5.5)

in the subspace

$$\Omega^* = \{ (A_k(t), I_k(t), T_k(t)) \in R^{3n}_+ | A_k(t) + I_k(t) + T_k(t) \le 1, k = 1, 2, \dots, n \}.$$
(5.6)

Next, it is easy to have the following theorem.

Theorem 4. For system (5.5), define

$$R_{1} = \frac{\lambda}{\langle kg(k)\rangle} \left[ \frac{\alpha \langle k\varphi_{1}(k)g(k)\rangle}{b} + \frac{\langle k\varphi_{2}(k)g(k)\rangle(1-\alpha\beta)}{c\gamma} \right]$$

There always exists a alcohol-free equilibrium  $E^1 = \{(0, 0, 0)\}_k$ , when  $R_0 > 1$  has a unique alcoholism equilibrium  $E^{**} = \{(A_k^{**}, T_k^{**})\}_k$ .

**Proof.** To get the equilibrium solution  $(A_k^{**}, I_k^{**}, T_k^{**})$ , we need to make the right side of system (5.5) equal to zero. Then the equilibrium  $(A_k^{**}, I_k^{**}, T_k^{**})$  should satisfy

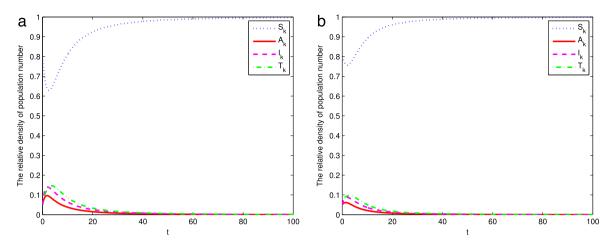
$$\frac{\alpha\lambda k(1 - A_{k}(t) - I_{k}(t)) - T_{k}(t)g(k)}{\langle kg(k) \rangle} \theta(t) - bA_{k}(t) = 0, 
\frac{(1 - \alpha)\lambda k(1 - A_{k}(t) - I_{k}(t)) - T_{k}(t)g(k)}{\langle kg(k) \rangle} \theta(t) + b(1 - \beta)A_{k}(t) 
+ d(1 - \gamma)T_{k}(t) - cI_{k}(t) = 0, 
cI_{k}(t) - dT_{k}(t) = 0.$$
(5.7)

A direct calculation yields:

$$A_{k}^{**} = \frac{\alpha\lambda kg(k)\theta cd\gamma \langle kg(k)\rangle}{[(c+d)\lambda kg(k)\theta H_{k}^{*} + c \langle kg(k)\rangle(\alpha\lambda kg(k)\theta + b \langle kg(k)\rangle)d\gamma]},$$

$$I_{k}^{**} = \frac{d\lambda kg(k)\theta H_{k}^{*}}{[(c+d)\lambda kg(k)\theta H_{k}^{*} + c \langle kg(k)\rangle(\alpha\lambda kg(k)\theta + b \langle kg(k)\rangle)d\gamma]},$$

$$T_{k}^{**} = \frac{c\lambda kg(k)\theta H_{k}^{*}}{[(c+d)\lambda kg(k)\theta H_{k}^{*} + c \langle kg(k)\rangle(\alpha\lambda kg(k)\theta + b \langle kg(k)\rangle)d\gamma]},$$
(5.8)



**Fig. 2.** Each compartment population changes over time on scale-free networks when  $R_0 < 1$ .

where

 $H_k^* = (\alpha \lambda kg(k)\theta + b\langle kg(k)\rangle)(1-\alpha) + b(1-\beta)\langle kg(k)\rangle\alpha - (1-\alpha)\lambda kg(k)\theta\alpha.$ 

Obviously,  $\theta = 0$  satisfies (5.8). Hence,  $A_k = I_k = T_k = 0$  is an equilibrium of (5.5), which is called the alcohol-free equilibrium.

Substituting  $A_k^{**}$  and  $I_k^{**}$  into  $\theta(t) = \theta_1(t) + \theta_2(t)$ , an equation of the form  $\theta F(\theta) = 0$  is obtained, where  $F(\theta) = 1 - (A + B)$ 

$$A = \sum_{k} \varphi_1(k) P(k) \frac{\alpha \lambda kg(k) c d\gamma \langle kg(k) \rangle}{(c+d)\lambda kg(k) \theta H_k^* + c \langle kg(k) \rangle (\alpha \lambda kg(k) \theta + b \langle kg(k) \rangle) d\gamma},$$

$$B = \sum_{k} \varphi_2(k) P(k) \frac{d\lambda kg(k) H_k^*}{(c+d)\lambda kg(k) \theta H_k^* + c \langle kg(k) \rangle (\alpha \lambda kg(k) \theta + b \langle kg(k) \rangle) d\gamma}.$$

Since  $F'(\theta) > 0$ , and  $F(\theta_1 + \theta_2) > 0$  the equation  $\theta F(\theta) = 0$  has a unique non-trivial solution if and only if F(0) < 0, i.e.

$$\frac{\lambda}{\langle kg(k)\rangle} \left[\frac{\alpha \langle k\varphi_1(k)g(k)\rangle}{b} + \frac{\langle k\varphi_2(k)g(k)\rangle(1-\alpha\beta)}{c\gamma}\right] > 1.$$

The proof is completed.

Similar to the proof of Theorems 2 and 3, we can also obtain follow theorem of the system (5.5).

**Theorem 5.** For system (5.5), there are two possibilities. Either  $R_1 < 1$ , the alcohol free equilibrium  $E^1$  is globally asymptotically stable in  $\Omega^*$ , or  $R_1 > 1$ , system (5.5) is permanent, that is there exists a constant  $\xi$  which satisfies  $\liminf_{t\to\infty} \{A_k(t), I_k(t), T_k(t)\} \ge \xi$ , where  $(A_k(t), I_k(t), T_k(t))$  is any solution of (5.5), satisfying (5.6), and  $A_k(0) > 0$ ,  $I_k(0) > 0$ ,  $T_k(0) > 0$ .

#### 5.2. The SAITS model on adaptive weighted networks

Some individuals tend to be more cautious in social contacts and make some reflects to reducing the intimacy when the alcoholism becomes severe. Such behaviors can change the strengths of nodes and the weights of links, which can be seen as an adaptive weighted networks. Next, we will study the following the modified SAITS model on adaptive weighted networks:

$$\frac{dS_{k}(t)}{dt} = -\frac{\lambda kS_{k}g(k)exp(-h(k)(A(t) + I(t)))}{\langle kg(k)exp(-h(k)(A(t) + I(t)))\rangle} (\theta_{1}(t) + \theta_{2}(t)) + b\beta A_{k}(t) + d\gamma T_{k}(t), 
\frac{dA_{k}(t)}{dt} = \frac{\alpha \lambda kS_{k}g(k)exp(-h(k)(A(t) + I(t)))}{\langle kg(k)exp(-h(k)(A(t) + I(t)))\rangle} (\theta_{1}(t) + \theta_{2}(t)) - bA_{k}(t), 
\frac{dI_{k}(t)}{dt} = b(1 - \beta)A_{k}(t) + \frac{(1 - \alpha)\lambda kS_{k}g(k)exp(-h(k)(A(t) + I(t)))}{\langle kg(k)exp(-h(k)(A(t) + I(t)))\rangle} (\theta_{1}(t) + \theta_{2}(t)) 
+ d(1 - \gamma)T_{k}(t) - cI_{k}(t),$$
(5.9)

We use the function

$$g'(k, t) = g(k)exp(-h(k)(A(t) + I(t))),$$

where h(k) is a increasing function of k. The corresponding  $\lambda_{ik}$  becomes

$$\lambda_{ik} = \lambda i \frac{\omega(i,k)}{\phi_i} = \frac{\lambda \langle k \rangle g(k) exp(-h(k)(A(t) + I(t)))}{\langle kg(k) exp(-h(k)(A(t) + I(t))) \rangle}.$$
(5.10)

Substituting (5.10) into (5.2), we obtain

$$\Theta_1''(t) = \frac{\lambda g(k)exp(-h(k)(A(t) + I(t)))}{\langle kg(k)exp(-h(k)(A(t) + I(t)))\rangle} \sum_i \varphi_1(i)P(i)A_i(t),$$

$$\Theta_2''(t) = \frac{\lambda g(k)exp(-h(k)(A(t) + I(t)))}{\langle kg(k)exp(-h(k)(A(t) + I(t)))\rangle} \sum_i \varphi_2(i)P(i)I_i(t).$$
(5.11)

Substituting (5.11) into (2.1), we obtain the adaptive weighted system (5.9). Where  $\theta_1(t) = \sum_i \varphi_1(i)p(i)A_i(t)$  and  $\theta_2(t) = \sum_i \varphi_2(i)p(i)I_i(t)$  in (5.9). Since  $S_k(t) = 1 - A_k(t) - I_k(t) - T_k(t)$ , it is sufficient to study the following system

$$\begin{cases} \frac{dA_{k}(t)}{dt} = \frac{\alpha\lambda k(1 - A_{k}(t) - I_{k}(t) - T_{k}(t))g(k)exp(-h(k)(A(t) + I(t)))}{\langle kg(k)exp(-h(k)(A(t) + I(t)))\rangle}\theta(t) - bA_{k}(t), \\ \frac{dI_{k}(t)}{dt} = \frac{(1 - \alpha)\lambda k(1 - A_{k}(t) - I_{k}(t) - T_{k}(t))g(k)exp(-h(k)(A(t) + I(t)))}{\langle kg(k)exp(-h(k)(A(t) + I(t)))\rangle}\theta(t) \\ + b(1 - \beta)A_{k}(t) + d(1 - \gamma)T_{k}(t) - cI_{k}(t), \\ \frac{dT_{k}(t)}{dt} = cI_{k}(t) - dT_{k}(t), \end{cases}$$
(5.12)

in the subspace

$$\Omega^* = \{(A_k(t), I_k(t), T_k(t)) \in R^{4n}_+ | A_k(t) + I_k(t) + T_k(t) \le 1, k = 1, 2, \dots, n\}.$$
(5.13)

The basic reproduction numbers of (5.12) is also  $R_1$ , which implies that the adaption of weights cannot change the basic reproduction number. Since the stability of the system (5.12) is difficult to prove, we will only give some simulations to discuss it in the next section.

#### 6. Numerical simulations

Numerical simulations are presented to illustrate the results of the system (2.1),(5.5) and (5.9). We take the scale-free networks with degree distribution is  $P(k) = ck^{-\gamma}$  (2 <  $\gamma \le 3$ ).

In Fig. 2, b = 0.6, c = 0.8, d = 0.7,  $\alpha = 0.4$ ,  $\beta = 0.8$ ,  $\rho_1 = 0.03$ ,  $\rho_2 = 0.03$ ,  $\gamma = 0.8$ , we know that the basic reproduction number  $R_0 = 0.9451$ . In Fig. 2(a) k = 100, in Fig. 2(b) k = 50. We can see that when  $R_0 < 1$ , alcoholics will disappear. Moreover, the smaller degree is, the faster the alcoholics disappear. This diagram is a better validation of Theorem 2, the alcohol free equilibrium is globally asymptotically stable when  $R_0 < 1$ .

Then we change the size of the infective rate in Fig. 3, let  $\rho_1 = 0.09$ ,  $\rho_2 = 0.10$  and the basic reproduction number  $R_0 = 3.0290$ . In Fig. 3(a) k = 100, in Fig. 3(b) k = 50. We can see that when  $R_0 > 1$ , alcoholics will converge to a positive stationary even for a small fraction of the light alcoholics and heavy alcoholics, which means the alcoholism state are stable. Moreover, the larger degree is, the faster the alcoholism equilibrium converge to a positive stationary. Fig. 3 is a better illustration of Theorem 3, alcoholism is permanent when  $R_0 > 1$ .

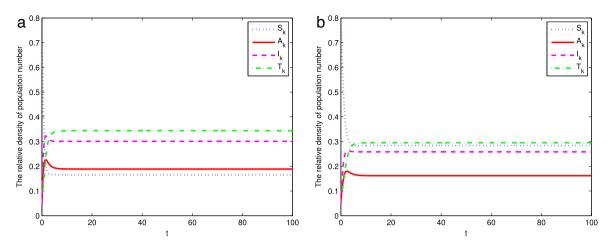
Figs. 4 and 5 show the dynamical behavior of light alcoholic and heavy alcoholic with different degree. We find the smaller degree is, the faster the alcoholics disappear; the larger degree is, the larger value of the alcoholics level.

Fig. 6 describes models with different degrees on fixed weight networks. Let n = 40,  $g(k) = k^{r0}$ ,  $\varphi_1(k) = k^{r1}$ ,  $\varphi_2(k) = k^{r2}$ , where  $r_0, r_1, r_2$  are positive constants.  $r_0 = 1$ ,  $r_1 = 0.6$ ,  $r_2 = 0.9$ ,  $\lambda = 0.02$ , b = 0.2, c = 0.3, d = 0.5,  $\alpha = 0.4$ ,  $\beta = 0.4$ ,  $\gamma = 0.8$ . Then  $R_0 = 0.9761$  and alcoholics decrease gradually even disappear.

In Fig. 7 n = 70,  $r_0 = 1.2$ ,  $r_1 = 0.5$ ,  $r_2 = 0.9$ ,  $\lambda = 0.013$ , b = 0.1, c = 0.1, d = 0.04,  $\alpha = 0.9$ ,  $\beta = 0.9$ ,  $\gamma = 0.9$ . Then  $R_0 = 1.5213$  and alcoholics densities reach their positive steady levels quickly and higher than the without weights.

Figs. 6 and 7 further validate the Theorem 5.

Figs. 8 and 9 illustrate the light alcoholic and heavy alcoholic with different degree on adaptive weight network when  $R_0 < 1$  and  $R_0 > 1$ . So, we choose the same parameters as Figs. 6 and 7, and  $h(k) = k^{r_3}$ ,  $r_3 = 1.1$  When h(k) = 0, the adaptive weight network is fixed weight network. Then we find that the light alcoholic and heavy alcoholic coverage to a smaller value.



**Fig. 3.** Each compartment population changes over time on scale-free networks when  $R_0 > 1$ .

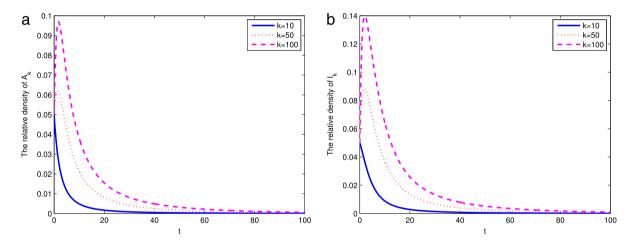


Fig. 4. Dynamical behavior of light alcoholic and heavy alcoholic with different degree on scale-free networks when  $R_0 < 1$ .

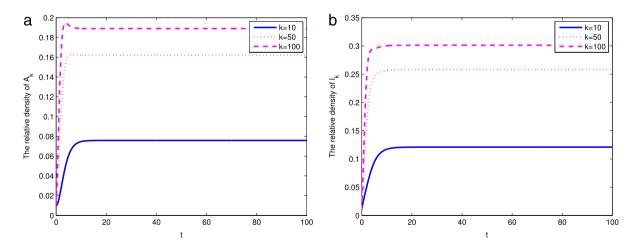


Fig. 5. Dynamical behavior of light alcoholic and heavy alcoholic with different degree on scale-free networks when  $R_0 > 1$ .

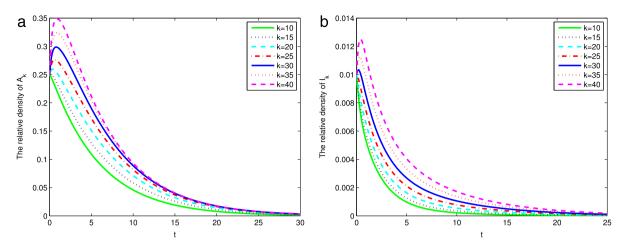


Fig. 6. Dynamical behavior of light alcoholic and heavy alcoholic with different degree on fix weight network when  $R_0 < 1$ .

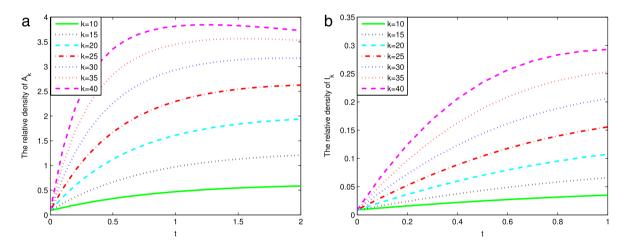


Fig. 7. Dynamical behavior of light alcoholic and heavy alcoholic with different degree on fix weight network when  $R_0 > 1$ .

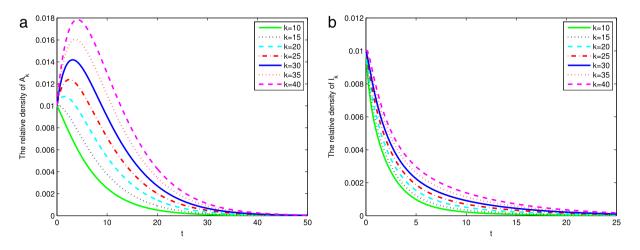


Fig. 8. Dynamical behavior of light alcoholic and heavy alcoholic with different degree on adaptive weight network when  $R_0 < 1$ .

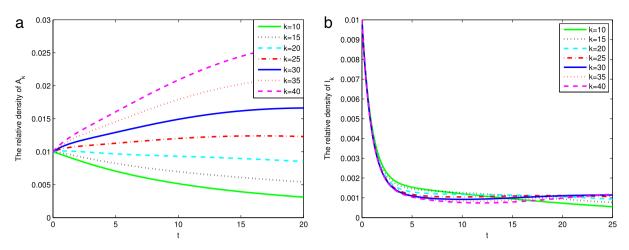


Fig. 9. Dynamical behavior of light alcoholic and heavy alcoholic with different degree on adaptive weight network when  $R_0 > 1$ .

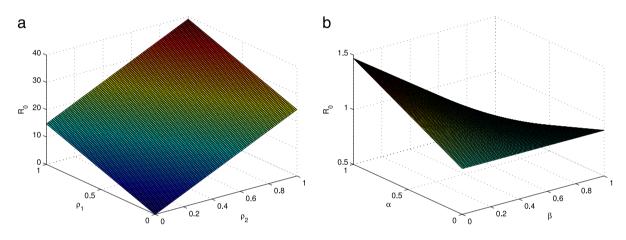


Fig. 10. The relationship between the basic reproduction number  $R_0$  and the parameters on scale-free networks.

#### 7. Sensitivity analysis and conclusions

The sensitivity analysis of the basic reproductive number  $R_0$  will be performed in terms of the parameters, obviously

$$\frac{\partial R_0}{\partial \rho_1} = \frac{\alpha \langle k^2 \rangle}{b \langle k \rangle}, \frac{\partial R_0}{\partial \rho_2} = \frac{(1 - \alpha \beta) \langle k^2 \rangle}{c \gamma \langle k \rangle},$$
$$\frac{\partial R_0}{\partial \alpha} = \frac{(\rho_1 c \gamma - b \beta \rho_2) \langle k^2 \rangle}{b c \gamma \langle k \rangle}, \frac{\partial R_0}{\partial \beta} = -\frac{\alpha \rho_2 \langle k^2 \rangle}{c \gamma \langle k \rangle}$$

By calculating, it is easy to know that  $\frac{\partial R_0}{\partial \rho_1} > 0$ ,  $\frac{\partial R_0}{\partial \rho_2} > 0$ ,  $\frac{\partial R_0}{\partial \beta} < 0$ . From Fig. 10(a), we can see that larger  $\rho_1$  or  $\rho_2$  can lead to larger  $R_0$ , that means long times contact with light problem alcoholics or heavy problem alcoholics can make the propagation of alcoholism easier. From Fig. 10(b),  $R_0$  increases as  $\alpha$  increases (when  $\rho_1 c\gamma > b\beta \rho_2$ ,  $\frac{\partial R_0}{\partial \alpha} > 0$ ).

In this paper, we have discussed a SAITS alcoholism model on scale-free networks, and subdivide the alcoholics into two groups (light problem alcoholics and heavy problem alcoholics). Through calculation, we find that the model exists two equilibria (alcohol-free equilibrium and alcoholism equilibrium). Using the existence of the alcoholism equilibrium, we obtained the basic reproduction  $R_0$ , which is closely related to the topology of the networks. As the basic reproduction number  $R_0$  is in direct proportion to the value  $\frac{\langle k^2 \rangle}{\langle k \rangle}$ , network heterogeneity may make the alcoholism more easily to spread. Using the comparison theorem, we analyze the stability of the alcohol-free equilibrium. If  $R_0 < 1$ , the alcoholics will disappear, but if not, the number of alcoholics will remain in a stable value. Furthermore, we also studied the modified SAITS model on fixed and adaptive weighted networks. By the same calculation, we know that they have the same basic reproduction  $R_1$ . Numerical simulations also confirm these theoretical results.

By the definition of  $R_0$ , we know that  $R_0 \propto \frac{\langle k^2 \rangle}{\langle k \rangle}$ . So  $R_0$  is proportional to the second moment of the degree, which diverges for increasing network sizes. we have that the alcoholism has a finite probability to generate a major outbreak in heterogeneous networks whatever the infection rate, and the heterogeneity of networks may make the alcoholism more easily to spread.

From Figs. 4 to 9, we find that the larger degree leads to larger value of the alcoholism level in our models with or without weighted.

From above sensitivity analysis, we can find the following control strategies: one is to reduce the infection rates  $\rho_1$  and  $\rho_2$  or to reduce the contact rates  $\theta_1$  and  $\theta_2$ . Due to the contact rates are affected by the degree *k*, by reducing the degree of alcoholics, we also can control the spread of the alcoholism.

It is easy to know that  $\frac{\partial R_0}{\partial b} = -\frac{\alpha \rho_1(k^2)}{b^2(k)} < 0$ ,  $\frac{\partial R_0}{\partial c} = -\frac{(1-\alpha\beta)\rho_2(k^2)}{c^2\gamma(k)} < 0$ . Increasing the remove rates *b*, *c* of the light alcoholic and heavy alcoholic compartment,  $R_0$  can also decrease. So, it is very important to treat alcoholics.

It is a very interesting to further study the effect of rewiring mechanism on alcoholic dynamics, and investigate the rich dynamics such as backward bifurcation, bistability and Hopf bifurcation, which is caused by rewiring mechanism on adaptive networks. We leave these work in the future.

#### References

- H. Wechsler, E. Lee, M. Kuo, M. Seibring, Trends in college binge dringking during a period of increased prevention efforts: Findings from 4 harvard school of public health college alcohol study surveys: 1993-2001, J. Am. Coll. Health 50 (2002) 203–217.
- [2] R. Hingson, T. Heeren, M. Winter, Magnitude of alcohol-related mortality and morbidity among US college students ages 18-24: Changes from 1998-2001, Annu. Rev. Public Health 26 (2005) 259–279.
- [3] J. Rehm, C. Mathers, S. Popova, et al., Global burden of disease and injury and economic cost attributable to alcohol use and alcohol-use disorders, Lancet 373 (2009) 2223–2233.
- [4] J. Rehm, The risks associated with alcohol use and alcoholism, Alcohol Res. Health 34 (2011) 135–143.
- [5] J. Baer, D. Kivlahan, G. Marlatt, High-risk drinking across the transition from high scholl to collage, Alcohol. Clin. Exp. Res. 19 (1995) 54–61.
- [6] H.F. Huo, Y.L. Chen, H. Xiang, Stability of a binge drinking model with delay, J. Biol. Dyn. 11 (2017) 210–226.
- [7] H.F. Huo, X.M. Zhang, Complex dynamics in an alcoholism model with the impact of twitter, Math. Biosci. 281 (2016) 24–35.
- [8] H. Xiang, N.N. Song, H.F. Huo, Modelling effects of public health educational campaigns on drinking dynamics, J. Biol. Dyn. 10 (2015) 164–178.
- [9] R. Bani, R. Hameed, S. Szymanowski, Influence of environmental factors on college alcohol drinking patterns, Commun. Nonlinear Sci. Numer. Simul. 10 (2013) 1281–1300.
- [10] G. Mulone, B. Straughan, Modeling binge drinking, Int. J. Biomath. 5 (2012) 1–14.
- [11] H.F. Huo, S.R. Huang, X.Y. Wang, H. Xiang, Optimal control of a social epidemic model with media coverage, J. Biol. Dyn. 11 (2017) 226–243.
- [12] H. Xiang, Y.L. Tang, H.F. Huo, A viral model with intracellular delay and humoral immunity, Bull. Malays. Math. Sci. Soc. 40 (2017) 1011–1023.
- [13] H.F. Huo, R. Chen, X.Y. Wang, Modelling and stability of HIV/AIDS epidemic model with treatment, Appl. Math. Model. 40 (2016) 6550–6559.
- [14] Y. Moreno, R. Pastor-Satorras, A. Vespignani, Epidemic outbreaks in complex heterogeneous networks, Eur. Phys. J. B 26 (2002) 521–529.
- [15] R. Olinky, L. Stone, Unexpected epidemic thresholds in heterogeneous networks: The role of disease transmission, Phys. Rev. E 70 (2004) 1-4.
- [16] R. Pastor-Satorras, A. Vespiganai, Epidemic spreading in scale-free networks, Phys. Rev. Lett. 86 (2001) 3200–3203.
- [17] R. Yang, B.H. Wang, J. Ren, Epidemic spreading on heterogeneous networks with identical infectivity, Phys. Lett. A 364 (2007) 189–193.
- [18] L. Wang, G.Z. Dai, Global stability of virus spreading in complex heteogeneous networks, Soc. Ind. Appl. Math. 68 (2008) 1459–1902.
- [19] R. Pastor-Satorras, A. Vespiganai, Epidemic dynamics and endemic states in complex networks, Phys. Rev. E 63 (2001) 066117.
- [20] R. Pastor-Satorras, A. Vespiganai, Epidemic dynamics in finte scale-free networks, Phys. Rev. E 65 (2002) 035108.
- [21] M. Boguna, R. Pastor-Satorras, A. Vespiganai, Absence of epidemic threshold in scale-free networks with connectivity correlations, Phys. Rev. Lett. 63 (2011) 1–9.
- [22] J. Joo, Behavior of susceptible-infected-susceptible epidemics on heterogeneous networks with saturation, Phys. Rev. E 69 (2004) 1–6.
- [23] J.L. Liu, T.L. Zhang, Epidemic spreading of an SEIRS model in scale-free networks, Commun. Nonlinear Sci. Numer. Simul. 16 (2011) 3375–3384.
- [24] T. Li, Y.M. Wang, Z.H. Guan, Spreading dynamics of a SIQRS epidemic model on scale-free networks, Commun. Nonlinear Sci. Numer. Simul. 19 (2014) 686–692.
- [25] C.H. Li, C.C. Tsai, S.Y. Yang, Analysis of epidemic spreading of an SIRS model in complex heterogeneous networks, Commun. Nonlinear Sci. Numer. Simul. 19 (2014) 1042–1054.
- [26] S.Y. Huang, F.D. Chen, L.G. Chen, Global dynamics of a network-based SIQRS epidemic model with demographics and vaccination, Commun. Nonlinear Sci. Numer. Simul. 43 (2017) 296–310.
- [27] S.Y. Huang, J.F. Jiang, Global stability of a network-based SIS epidemic model with a general nonlinear incidence rate, Math. Biosci. 13 (2016) 723–729.
- [28] G.H. Zhu, X.C. Fu, G.R. Chen, Global attractivity of a network-based epidemic SIS model with nonlinear infectivity, Commun. Nonlinear Sci. Numer. Simul. 17 (2012) 2588–2594.
- [29] H.F. Zhang, X.C. Fu, Spreading of epidemics on scale-free networks with nonlinear infectivity, Nonlinear Anal. 70 (2009) 3273–3278.
- [30] Z. Jin, G.O. Sun, H.P. Zhu, Epidemic models for complex networks with demographics, Math. Biosci, Eng. 11 (2014) 1295–1317.
- [31] J.P. Zhang, Z. Jin, The analysis of an epidemic model on networks, Appl. Math. Comput. 217 (2011) 7053-7064.
- [32] G.H. Zhu, X.C. Fu, G.R. Chen, Spreading dynamics and global stability of a generalized epidemic model on complex heterogeneous networks, Appl. Math. Model. 36 (2012) 5808-5817.
- [33] H. Xiang, Y.P. Liu, H.F. Huo, Stability of an SAIRS alcoholism model on scale-free networks, Physica A 473 (2017) 276-292.
- [34] H.F. Huo, Y.P. Liu, The analysis of the SIRS alcoholism models with relapse on weighted networks, Springer Plus 5 (2016) 1–19.
- [35] H.F. Huo, Y.Y. Wang, Impact of media coverage on the drinking dynamics in the scale-free networks, Springer Plus 5 (2016) 1–16.
- [36] Department of Health and Human Services, United States of America, National Institute on Alcohol Abuse and Alcoholism, p. 056108. (Last accessed March 11, 2009).
- [37] A. Lajmanovich, J.A. Yorke, A determinstic model for gonorrhea in a nonhomogenous population, Soc. Ind. Appl. Math. J. Math. Anal. 24 (1993) 407–435.
- [38] X.W. Chu, Z.Z. Zhang, J.H. Guan, S.G. Zhou, Epidemic spreading with nonlinear infectivity in weighted scale-free networks, Physica A 390 (2011) 471-481