Analysis of the SAITS alcoholism model on scale-free networks with demographic and nonlinear infectivity

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ABSTRACT
A more realistic alcoholism model on scale-free networks with demographic and nonlinear infectivity is introduced in this paper. The basic reproduction number $R_0$ is derived from the next-generation method. Global stability of the alcohol-free equilibrium is obtained. The persistence of our model is also derived. Furthermore, the SAITS model with nonlinear infectivity is also investigated. Stability of all the equilibria and persistence are also obtained. Some numerical simulations are also presented to verify and extend our theoretical results.

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1. Introduction
Alcohol consumption has been identified as an important risk factor for chronic diseases and injuries. Excessive drinking can cause many diseases, such as high blood pressure and heart attack, and also can lead to family conflicts. Alcohol consumption is a major contributor to the global burden of diseases [26,27]. Alcoholism can be viewed as a treatable contagious disease since social interaction is often considered to be the key factor in the spreading behaviour which is harmful to health. Mathematical models can mimic the process of drinking behaviour. Several different mathematical models for drinking have been formulated and studied recently. Huo and Zhang [12] investigated a novel alcoholism model which involves the impact of Twitter and studied the stability of all the equilibria. Furthermore, they also investigated the occurrence of backward, forward and Hopf bifurcation. They obtained that Twitter could serve as a good indicator of the alcoholism model and affected the spread of drinking. Xiang et al. [33] dealt with the global property of a drinking model with public health educational campaigns and concluded that the public health educational campaigns of drinking individuals could slow down the drinking dynamics. For other related alcoholism models or epidemic models, we refer to [1,3,6–8,11,13,14,16,21–23,28,31,39,40].

However, the above alcohol models usually assume homogeneous random mixing which implies that all individuals are equal to contact each other. It is rarely observed in the real world. Individuals often contact only a small, clustered, subpopulation in reality, so many people study epidemic or alcoholism models on complex networks recently.

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Pastor-Satorras and Vespiganai [24] studied a dynamical model for the spreading of infectious diseases on scale-free networks, and found the absence of an epidemic threshold and its associated critical behaviour. Zhu et al. [42] introduced a generalized epidemic model on complex networks. Their results explained why the heterogeneous connectivity patterns impacted the epidemic threshold. Yan et al. [34] developed a novel model for sexually transmitted diseases on a bipartite random contact network and found that the final size in either sex was heavily influenced by the degree distribution of the opposite sex. Huo et al. [15] introduced the novel Susceptible Light-problem Alcoholic Heavy-problem Alcoholic Treatment Susceptible [SAITS] alcoholism model on networks, and studied the dynamics of the model on unweighted and weighted networks.

Since many diseases can cause many deaths to influence the size of the population, it is necessary to take the birth and death rates into account. Jin et al. [17] proposed and studied epidemic models for complex networks with demographics and found that demographics had a great effect on the basic reproduction number. Liu et al. [20] introduced a modified epidemic model on scale-free networks with birth and death rates. Yang and Chen [35] mainly studied the Susceptible Infected Recovered [SIR] epidemic model with infection age on the complex network and proved the stability of the steady state.

On the other hand, the expression of infectivity is very important to the model and can affect the dynamical behaviour of the model largely. Chu et al. [4] introduced an SIR model on weighted scale-free networks with nonlinear infectivity. They found that the infectivity exponent had a stronger impact on the epidemic prevalence than the weight exponent. Various forms of infectivity were proposed to improve the actuality of models recently [30,32,36,37,41].

Comparing with the drinking models of [11,12,15,23,30,32,33,40], first, we introduce network structure and different compartments in our model. Second, we not only consider that susceptible individuals can become heavy problem alcoholics directly, but also take into account the recovery of light problem alcoholics and relapse of treatment individuals. Last, we not only take into account birth and death rates of individuals, but also introduce nonlinear infectivity in our model. Motivated by the above, we set up a new SAITS binge drinking model on scale-free networks with demographic and nonlinear infectivity, and study the dynamics of our model. Furthermore, we study the influence of nonlinear infectivity on the transmission of our drinking model on scale-free networks.

The structure of this paper is organized as follows. In Section 2, the alcoholism model with demographics is constructed and analysed. In Section 3, the basic reproduction number and existence of equilibria are studied. In Section 4, stability analysis of the equilibria is given. In Section 5, the SAITS alcoholism model with nonlinear infectivity is also introduced. Numerical simulations are illustrated in Section 6. In Section 7, we give some conclusions.

2. Model formulation

The total population \(N\) is divided into \(n\) groups according to the degree of a node, namely,

\[
N = N_1 + N_2 + \cdots + N_n.
\]  

One ‘standard’ drink contains roughly 14 g of pure alcohol by the research group of treatment and rehabilitation of the U.S. National Institute on Alcohol Abuse and Alcoholism
(NIAAA), in the United States. The daily alcohol consumption for men is no more than 4 standard drinks, and women are no more than 3 standard drinks. The ceiling of ‘low-risk’ alcohol consumption per week is 14 standard drinks for men and 7 for women [5]. If a person whose alcohol consumption is more than daily or weekly drinking ceiling, he/she most likely develop to ‘abuse alcohol’ or ‘addicted alcohol’. Furthermore, we divide every groups into four compartments, the susceptible compartment $S_k(t)$ refers to the people who do not drink or drink only moderately. The light problem alcoholics $A_k(t)$ refer to the drinkers who drink beyond the daily or weekly ceiling and drink 4 to 5 standard drinks per day. The heavy problem alcoholics $I_k(t)$ refer to the drinkers who drink more than daily and weekly limits and drink more than 5 standard drinks per day. The treatment compartment $T_k(t)$ refers to the drinkers who receive treatment.

On the network, each individual is represented by a node of the network and the edges are the connection among individuals. Individuals are spatially distributed on the network. Each site of the network is empty or occupied at most by one individual. Similar to that of [20], each site is given a number: 0, 1, 2, 3, 4. They denote the empty state, the susceptible individual, the light problem alcoholic individual, the heavy problem alcoholic individual and the individual who receives treatment, respectively. Each site can be transformed by a certain rate. An empty site can give birth to a susceptible individual at the rate $b$. A susceptible individual can become a light problem alcoholic at the rate $\alpha \rho(t)$ or become a heavy problem alcoholic at the rate $(1 - \alpha)\rho(t)$, where $\rho(t) = \rho_1 \theta_1(t) + \rho_2 \theta_2(t)$, $\theta_1(t)$ denotes the probability of a susceptible individual in contact with a light problem alcoholic, and $\theta_2(t)$ is the probability of a susceptible individual gets in touch with a heavy problem alcoholic. A susceptible individual can be infected by a light problem alcoholic at the rate $\rho_1$ or be infected by a heavy problem alcoholic at the rate $\rho_2$. A light problem alcoholic can become a susceptible individual at the rate $c\beta$ or become a heavy problem alcoholic individual at the rate $c(1 - \beta)$. $c$ is the departure rate of the light problem alcoholic compartment. The treatment rate of the heavy problem alcoholic compartment is $d$. A treatment individual can become a susceptible individual at the rate $a\gamma$ or become a heavy problem alcoholic at the rate $a(1 - \gamma)$. The success rate of treatment is $\gamma$. The departure rate of the treatment compartment is $a$. The death rate of all individuals is $\mu$. The structure of our model is shown in Figure 1.
Figure 1 leads to the following system of ordinary differential equations:

\[
\frac{dS_k(t)}{dt} = b(1 - S_k(t) - A_k(t) - I_k(t) - T_k(t)) - kS_k(t)\rho(t) + c\beta A_k(t) + a\gamma T_k(t) - \mu S_k(t),
\]

\[
\frac{dA_k(t)}{dt} = \alpha kS_k(t)\rho(t) - cA_k(t) - \mu A_k(t),
\]

\[
\frac{dI_k(t)}{dt} = (1 - \alpha)kS_k(t)\rho(t) + c(1 - \beta)A_k(t) + a(1 - \gamma)T_k(t) - dI_k(t) - \mu I_k(t),
\]

\[
\frac{dT_k(t)}{dt} = dI_k(t) - aT_k(t) - \mu T_k(t),
\]

for all mean degree value \(\langle k \rangle\) of the population with degree \(d\).

Adding the four equations of system (2), we can obtain

\[
\frac{dN_k(t)}{dt} = b - (b + \mu)N_k(t),
\]

and \(N_k(t) = (b/(b + \mu)) + N_k(0)e^{-(b+\mu)(t)}\), where \(N_k(0)\) is the initial density of the whole population with degree \(k\). By calculating, we know \(\lim_{t \to \infty} N_k(t) = b/(b + \mu)\), then for all \(t > 0\), \(N_k(t) \leq b/(b + \mu)\). For biological significance, the initial conditions for system (2) satisfy \(S_k(0) + A_k(0) + I_k(0) + T_k(0) \leq b/(b + \mu), \rho(0) > 0\). It is easy to know that the region

\[
\Omega = \{(S_k(t), A_k(t), I_k(t), T_k(t)) \in \mathbb{R}_+^{4n} | S_k(t) + A_k(t) + I_k(t) + T_k(t) \leq \frac{b}{b + \mu},
\]

\[k = 1, 2, \ldots, n,\]

is the positively invariant set for (2). Therefore, we consider the dynamics of system (2) in the set \(\Omega\) in this paper.

Since \(S_k(t) = (b/(b + \mu)) - A_k(t) - I_k(t) - T_k(t)\) at steady state, it is sufficient to study the limiting system

\[
\frac{dA_k(t)}{dt} = \alpha k \left(\frac{b}{b + \mu} - A_k(t) - I_k(t) - T_k(t)\right)\rho(t) - cA_k(t) - \mu A_k(t),
\]

\[
\frac{dI_k(t)}{dt} = (1 - \alpha)k \left(\frac{b}{b + \mu} - A_k(t) - I_k(t) - T_k(t)\right)\rho(t) + c(1 - \beta)A_k(t) + a(1 - \gamma)T_k(t) - dI_k(t) - \mu I_k(t),
\]

\[
\frac{dT_k(t)}{dt} = dI_k(t) - aT_k(t) - \mu T_k(t),
\]

(5)
in the subspace

\[
\Omega^* = \{(A_k(t), I_k(t), T_k(t)) \in \mathbb{R}_{+}^{3n} | A_k(t) + I_k(t) + T_k(t) \leq \frac{b}{b + \mu}, \ k = 1, \ldots, n\}.
\]

(6)

3. The basic reproduction number and existence of equilibria

Using the next-generation method in [29], it is easy to know that

\[
\mathcal{F}(\mathbf{x}) = \left(\begin{array}{c}
\alpha k \rho(t) \left(\frac{\beta}{b + \mu} - A_k - I_k - T_k\right) \\
0 \\
0
\end{array}\right)_{3n \times 1},
\]

\[
\mathcal{V}(\mathbf{x}) = \left(\begin{array}{c}
(d + \mu)I_k - (1-\alpha)k \rho(t) \left(\frac{\beta}{b + \mu} - A_k - I_k - T_k\right) - c(1-\beta)A_k - a(1-\gamma)T_k \\
(a + \mu)T_k - \beta I_k
\end{array}\right)_{3n \times 1}.
\]

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

\[
F = D\mathcal{F}(E^0) = \left(\begin{array}{ccc}
F_{11} & F_{12} & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{array}\right)_{3n \times 3n},
\]

\[
V = D\mathcal{V}(E^0) = \left(\begin{array}{ccc}
V_{11} & 0 & 0 \\
V_{21} & V_{22} & V_{23} \\
0 & V_{32} & V_{33}
\end{array}\right)_{3n \times 3n},
\]

\[
F_{11} = \frac{b}{b + \mu} \alpha \rho_1 \langle k \rangle \\
F_{12} = \frac{b}{b + \mu} \alpha \rho_2 \langle k \rangle
\]

\[
V_{11} = (c + \mu)I, \ \ V_{21} = [-c(1-\beta)]I - b/(b + \mu)(1-\alpha)\rho_1(\langle ijp(j)/\langle k \rangle \rangle), \ \ V_{22} = (d + \\
\mu)I - b/(b + \mu)(1-\alpha)\rho_2(\langle ijp(j)/\langle k \rangle \rangle), \ \ V_{23} = [-a(1-\gamma)]I, \ \ V_{32} = -dI, V_{33} = (a + \\
\mu)I.
\]

The basic reproduction number is denoted by

\[
R_0 = \rho(FV^{-1}) \langle k^2 \rangle \left[\frac{\rho_1 \alpha b}{(c + \mu)(b + \mu)}
+ \frac{\rho_2 bd[(\alpha \beta - 1) - \mu(1-\alpha)]}{(c + \mu)(b + \mu)[a(a + \mu)(1-\gamma) - d(d + \mu)]}\right].
\]

We have the following theorem.
Theorem 3.1: For system (5), there always exists an alcohol-free equilibrium \( E^0 = \{(0, 0, 0)\} \) and when \( R_0 > 1 \), then system (5) has a unique alcoholism equilibrium \( E^* = \{(A^*_k, I^*_k, T^*_k)\} \).

Proof: It is easy to know that system (5) always exists as an alcohol-free equilibrium \( E^0 = \{(0, 0, 0)\} \). Next, to get the positive equilibrium solution \( (A^*_k, I^*_k, T^*_k) \), let the right side of system (5) equals to zero. Then the equilibrium \( (A^*_k, I^*_k, T^*_k) \) must satisfy

\[
\begin{align*}
\alpha k ((b/(b + \mu)) - A_k(t) - I_k(t) - T_k(t)) \rho(t) - cA_k(t) - \mu A_k(t) &= 0, \\
(1 - \alpha) k ((b/(b + \mu)) - A_k(t) - I_k(t) - T_k(t)) \rho(t) + c(1 - \beta) A_k(t) &+ a(1 - \gamma) T_k(t) - dI_k(t) - \mu I_k(t) = 0, \\
dI_k(t) - aT_k(t) - \mu T_k(t) &= 0.
\end{align*}
\]

From (7), we have

\[
\begin{align*}
A^*_k &= \frac{\alpha k \rho b[a(a + \mu)(1 - \gamma) - d(d + \mu)]}{(a + \mu + d)(b + \mu)H_k + B_k[a(a + \mu)(1 - \gamma) - d(d + \mu)]}, \\
I^*_k &= \frac{bdH_k}{(a + \mu + d)(b + \mu)H_k + B_k[a(a + \mu)(1 - \gamma) - d(d + \mu)]}, \\
T^*_k &= \frac{b(a + \mu)H_k}{((a + \mu + d)(b + \mu)H_k + B_k[a(a + \mu)(1 - \gamma) - d(d + \mu)]},
\end{align*}
\]

where

\[
H_k = k\rho[c(\alpha\beta - 1) - \mu(1 - \alpha)], \quad B_k = (\alpha k\rho + c + \mu)(b + \mu),
\]

and \( \rho \leq (\rho_1 + \rho_2) \).

Substituting \( A^*_k \) and \( I^*_k \) into \( \rho \), an equation of the form \( \rho f(\rho) = 0 \) is obtained, where

\[
f(\rho) = 1 - A \left[ \frac{\rho_1 \alpha b[a(a + \mu)(1 - \gamma) - d(d + \mu)] + \rho_2 bd[c(\alpha\beta - 1) - \mu(1 - \alpha)]}{\langle k \rangle} \right],
\]

\[
A = \sum \frac{j^2 p(i)}{(a + \mu + d)(b + \mu)H_i + B_i[a(a + \mu)(1 - \gamma) - d(d + \mu)]}.
\]

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

\[
R_0 = \frac{\langle k^2 \rangle}{\langle k \rangle} \left[ \frac{\rho_1 \alpha b}{(c + \mu)(b + \mu)} + \frac{\rho_2 bd[c(\alpha\beta - 1) - \mu(1 - \alpha)]}{(c + \mu)(b + \mu)[a(a + \mu)(1 - \gamma) - d(d + \mu)]} \right] > 1,
\]

where \( \langle k^2 \rangle = \sum k^2 p(k) \). The proof is completed.
4. Stability analysis of all the equilibria

4.1. The stability of the alcohol-free equilibrium

In this subsection, we will prove the global asymptotic stability of the alcohol-free equilibrium.

**Theorem 4.1:** For system (5), the alcohol-free equilibrium \( E^0 = \{(0,0,0)\}_k \) is globally asymptotically stable if \( R_0 < 1 \).

**Proof:** Following Theorem 2 of [29], we know that the alcohol-free equilibrium \( E^0 = \{(0,0,0)\}_k \) is locally asymptotically stable if \( R_0 < 1 \). We suppose that \( A_1 = y_1, A_2 = y_2, \ldots, A_n = y_n, I_1 = y_{n+1}, I_2 = y_{n+2}, \ldots, I_n = y_{2n}, T_1 = y_{2n+1}, T_2 = y_{2n+2}, \ldots, T_n = y_{3n} \) and \( y = (y_1, \ldots, y_n, y_{n+1}, \ldots, y_{2n}, y_{2n+1}, \ldots, y_{3n})^T \). System (5) can be written as follows:

\[
\frac{dy}{dt} = Ay + N(y),
\]

where \( A = F - V \),

\[
N(y) = -\begin{pmatrix}
\alpha\rho(y_1 + y_{n+1} + y_{2n+1}) \\
2\alpha\rho(y_2 + y_{n+2} + y_{2n+2}) \\
\vdots \\
n\alpha\rho(y_n + y_{2n} + y_{3n}) \\
(1 - \alpha)\rho(y_1 + y_{n+1} + y_{2n+1}) \\
2(1 - \alpha)\rho(y_2 + y_{n+2} + y_{2n+2}) \\
\vdots \\
n(1 - \alpha)\rho(y_n + y_{2n} + y_{3n}) \\
0 \\
\vdots \\
0
\end{pmatrix}_{3n \times 1}.
\]

The matrices \( F, V \) are given by

\[
F(x) = \begin{pmatrix}
\alpha k \rho(t) \left( \frac{b}{b + \mu} - A_k - I_k - T_k \right) \\
0 \\
0
\end{pmatrix}_{3n \times 1},
\]

\[
V(x) = \begin{pmatrix}
(d + \mu)I_k - (1 - \alpha)k \rho(t) \left( \frac{b}{b + \mu} - A_k - I_k - T_k \right) - c(1 - \beta)A_k - a(1 - \gamma)T_k
\end{pmatrix}_{3n \times 1}.
\]

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

\[
F = D F(E^0) = \begin{pmatrix}
F_{11} & F_{12} & 0 \\
0 & 0 & 0 \\
0 & 0 & 0
\end{pmatrix}_{3n \times 3n}.
\]
V = D\, V\, (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} = \left( \frac{b}{b + \mu} \alpha_1 \langle k \rangle \right) \begin{pmatrix} P(1) & 2P(2) & \cdots & nP(n) \\ 2P(1) & 2^2P(2) & \cdots & 2nP(n) \\ \vdots & \vdots & \ddots & \vdots \\ nP(1) & 2nP(2) & \cdots & n^2P(n) \end{pmatrix}_{n \times n},

F_{12} = \left( \frac{b}{b + \mu} \alpha_2 \langle k \rangle \right) \begin{pmatrix} P(1) & 2P(2) & \cdots & nP(n) \\ 2P(1) & 2^2P(2) & \cdots & 2nP(n) \\ \vdots & \vdots & \ddots & \vdots \\ nP(1) & 2nP(2) & \cdots & n^2P(n) \end{pmatrix}_{n \times n},

V_{11} = (c + \mu)I, V_{21} = [-c(1 - \beta)]I - (b/(b + \mu))(1 - \alpha)\rho_1(ipp(j)/\langle k \rangle), V_{22} = (d + \mu)I - (b/(b + \mu))(1 - \alpha)\rho_2(ipp(j)/\langle k \rangle), V_{23} = [-a(1 - \gamma)]I, V_{32} = -dI, V_{33} = (a + \mu)I.

Hence

\frac{dy}{dt} \leq Ay. \quad (10)

If \( R_0 < 1 \), linear system (10) is stable. So, \( A_k \to 0, I_k \to 0, T_k \to 0, \) as \( t \to \infty \). Using the comparison theorem, nonlinear system (9) is stable, \( A_k \to 0, I_k \to 0, T_k \to 0, \) as \( t \to \infty \), for \( R_0 < 1 \). The alcohol-free equilibrium of system (5) is globally asymptotically stable. The proof is completed.

From the result of Theorem 4.1, we know that alcoholics will disappear if the basic reproduction number is less than 1, which means that alcoholism will disappear.

### 4.2. The persistence of the model

To show the persistence of our model, we introduce the following two lemmas.

**Lemma 4.1 ([19]):** Let \( A = a_{ij} \) be an irreducible \( n \times n \) matrix and assume \( a_{ij} \geq 0 \) whenever \( i \neq j \). Then there exists a positive eigenvector \( \omega \) of \( A \), and the corresponding eigenvalue is \( S(A) \).

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

**Lemma 4.2 ([19]):** Consider the system

\[
\frac{dy}{dt} = Ay + N(y),
\]

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 system (9), 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 \( (\omega \cdot y) \geq r \| y \| \) 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 (9) satisfies

\[
\liminf_{t \to \infty} \| \varphi(t, y_0) \| \geq e,
\]

\( e > 0 \) independent of \( y_0 \). Moreover, there exists a constant solution of (9), \( y = y^* \), \( y^* \in C - \{0\} \).

Here

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

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

\[
B_{11} = \begin{pmatrix} \frac{b}{b + \mu} \alpha \rho_1 G_1 - (c + \mu) & \cdots & \frac{b}{b + \mu} \alpha \rho_1 G_n \\ \vdots & \ddots & \vdots \\ \frac{b}{b + \mu} n\alpha \rho_1 G_1 & \cdots & \frac{b}{b + \mu} n\alpha \rho_1 G_n - (c + \mu) \end{pmatrix}_{n \times n},
\]

\[
B_{12} = \begin{pmatrix} \frac{b}{b + \mu} \alpha \rho_2 G_1 & \cdots & \frac{b}{b + \mu} \alpha \rho_2 G_n \\ \vdots & \ddots & \vdots \\ \frac{b}{b + \mu} n\alpha \rho_2 G_1 & \cdots & \frac{b}{b + \mu} n\alpha \rho_2 G_n \end{pmatrix}_{n \times n},
\]

\[
B_{21} = \begin{pmatrix} (1 - \beta)c + \frac{b}{b + \mu} (1 - \alpha) \rho_1 G_1 & \cdots & \frac{b}{b + \mu} (1 - \alpha) \rho_1 G_n \\ \vdots & \ddots & \vdots \\ \frac{b}{b + \mu} n(1 - \alpha) \rho_1 G_1 & \cdots & (1 - \beta)c + \frac{b}{b + \mu} (1 - \alpha) \rho_1 G_n \end{pmatrix}_{n \times n},
\]

\[
B_{22} = \begin{pmatrix} \frac{b}{b + \mu} (1 - \alpha) \rho_2 G_1 - (d + \mu) & \cdots & \frac{b}{b + \mu} (1 - \alpha) \rho_2 G_n \\ \vdots & \ddots & \vdots \\ \frac{b}{b + \mu} n(1 - \alpha) \rho_2 G_1 & \cdots & \frac{b}{b + \mu} n(1 - \alpha) \rho_2 G_n - (d + \mu) \end{pmatrix}_{n \times n},
\]

\[
A_{12} = \begin{pmatrix} 0 & \cdots & 0 \\ \vdots & \ddots & \vdots \\ c(1 - \gamma) & \cdots & 0 \end{pmatrix}_{2n \times n}.
\]
\[
A_{21} = \begin{pmatrix}
0 & \cdots & 0 & d & \cdots & 0 \\
\vdots & \ddots & \vdots & \vdots & \ddots & \vdots \\
0 & \cdots & 0 & 0 & \cdots & d
\end{pmatrix}_{n \times 2n},
\]
\[
A_{22} = -(c + \mu)I,
\]
and \( I \) is the identity matrix. Define \( G(j) = jp(j)/\langle k \rangle \).

**Theorem 4.2:** If \( R_0 > 1 \), then system (5) is permanent, that is, 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 (5), satisfying (6), and \( A_k(0) > 0, I_k(0) > 0, T_k(0) > 0 \).

**Proof:** We will prove it by using Lemma 4.2.

(i) We can see that the compact convex \( \Omega^* \) is a positive invariant set for system (5), and \( 0 \in \Omega^* \).

(ii) Using the limit rule and mean inequality, we know that \( \lim_{y \to 0} \| N(y) \| / \| y \| = 0 \).

(iii) Apparently \( A_{11}^T = (a_{ij})_{2n \times 2n} \) is irreducible and \( a_{ij} \geq 0 \), for all \( i \neq j \). Then from Lemma 4.1, there exists an eigenvector \( \omega = (\omega_1, \omega_2, \ldots, \omega_{2n}) \) of \( A_{11}^T \), and the associated eigenvalue is \( S(A_{11}^T) \). \( S(A_{11}^T) > 0 \), if \( R_0 > 1 \). Let \( \omega_{2n+1} = \cdots = \omega_{3n} = 0 \) and \( \omega = (\omega_1, \omega_2, \ldots, \omega_{3n}) \), then \( A^T \omega = S(A_{11}^T)\omega \). The vector \( \omega \) is the eigenvector of the matrix \( A_1^T \) that corresponds to the eigenvalue \( S(A_{11}^T) \). Assume \( r = \min_{1 \leq i \leq 2n} \{\omega_i\} > 0 \), for all \( y \in \Omega^* \), then we obtain \( (\omega \cdot y) \geq r \| y \| \).

(iv) As each item of \( N(y) \) is not positive and \( \omega \geq 0 \), so \( (\omega \cdot N(y)) \leq 0 \).

(v) For system (5), \( H = \{y \in \Omega^* | (\omega \cdot N(y)) = 0\} \). If \( y \in H \), we have \( (1/\langle k \rangle) \sum_{i=1}^n i\omega_i \alpha (\rho_1 \sum_{k=1}^n kp(k)y_i + \rho_2 \sum_{k=1}^n kp(k)y_{n+i})(y_i + y_{n+i} + y_{2n+i}) + (1/\langle k \rangle) \sum_{i=1}^n i\omega_i (1 - \alpha)(\rho_1 \sum_{k=1}^n kp(k)y_i + \rho_2 \sum_{k=1}^n kp(k)y_{n+i})(y_i + y_{n+i} + y_{2n+i}) = 0 \) for all \( i = 1, 2, \ldots, n \). Since \( \omega_1 > 0, i > 0 \), 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 system (5), 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 4.2 are satisfied. The proof is completed.

From the result of Theorem 4.2, we know that light and heavy alcoholics will maintain at a positive stationary value if the basic reproduction number is more than 1, which means that the alcoholism becomes an endemic ‘disease’.
5. The SAITS alcoholism model with nonlinear infectivity

In this section, we will investigate the following model with nonlinear infectivity:

\[
\frac{dS_k(t)}{dt} = b(1 - S_k(t) - A_k(t) - I_k(t) - T_k(t)) - kS_k(t)\rho(t) + c\beta A_k(t) + a\gamma T_k(t) - \mu S_k(t),
\]
\[
\frac{dA_k(t)}{dt} = \alpha kS_k(t)\rho(t) - cA_k(t) - \mu A_k(t),
\]
\[
\frac{dI_k(t)}{dt} = (1 - \alpha)kS_k(t)\rho(t) + c(1 - \beta)A_k(t) + a(1 - \gamma)T_k(t) - dI_k(t) - \mu I_k(t),
\]
\[
\frac{dT_k(t)}{dt} = dI_k(t) - aT_k(t) - \mu T_k(t),
\]

where \(\rho(t) = \rho_1 \theta_1(t) + \rho_2 \theta_2(t)\),

\[
\theta_1(t) = \sum_i p(i[k]) \frac{\varphi_1(i)}{i} A_i(t) = \langle k \rangle^{-1} \sum_k \varphi_1(k)p(k)A_k(t),
\]
\[
\theta_2(t) = \sum_i p(i[k]) \frac{\varphi_2(i)}{i} I_i(t) = \langle k \rangle^{-1} \sum_k \varphi_2(k)p(k)I_k(t),
\]

\(\varphi_1(k)\) is the nonlinear infectivity of the light problem alcoholic with degree \(k\) and \(\varphi_2(k)\) is the nonlinear infectivity of the heavy problem alcoholic with degree \(k\). Other parameters are the same as system (2). It is easy to know that the region

\[
\Omega = \{(S_k(t), A_k(t), I_k(t), T_k(t)) \in \mathbb{R}^{4n}_+ | S_k(t) + A_k(t) + I_k(t) + T_k(t) \leq \frac{b}{b + \mu}, k = 1, 2, \ldots, n\}
\]

is the positively invariant set for (12).

Since \(S_k(t) = 1 - A_k(t) - I_k(t) - T_k(t)\) at steady state, it is sufficient to study the limiting system

\[
\frac{dA_k(t)}{dt} = \alpha k(1 - A_k(t) - I_k(t) - T_k(t))\rho(t) - bA_k(t),
\]
\[
\frac{dI_k(t)}{dt} = b(1 - \beta)A_k(t) + (1 - \alpha)k(1 - A_k(t) - I_k(t) - T_k(t))\rho(t) + d(1 - \gamma)T_k(t) - cI_k(t),
\]
\[
\frac{dT_k(t)}{dt} = cI_k(t) - dT_k(t),
\]

in the subspace

\[
\Omega^* = \left\{(A_k(t), I_k(t), T_k(t)) \in \mathbb{R}^{3n}_+ | A_k(t) + I_k(t) + T_k(t) \leq \frac{b}{b + \mu}, k = 1, 2, \ldots, n\right\}.
\]
Theorem 5.1: Let

\[ R_1 = \frac{k\varphi_1(k)}{\langle k \rangle} \frac{\rho_1ab}{(c + \mu)(b + \mu)} + \frac{k\varphi_2(k)}{\langle k \rangle} \frac{\rho_2bd[c(\alpha\beta - 1) - \mu(1 - \alpha)]}{(c + \mu)(b + \mu)[a(a + \mu)(1 - \gamma) - d(d + \mu)]}. \]

1. When \( R_1 < 1 \), there exists an alcohol-free equilibrium \( E^1 = \{0, 0, 0\}_k \).
2. System (15) has a unique alcoholism equilibrium \( E^{**} = \{A^{**}_k, I^{**}_k, T^{**}_k\}_k \), when \( R_1 > 1 \).

Proof: To get the equilibrium solution \( (A^{**}_k, I^{**}_k, T^{**}_k) \), we need to make the right side of system (5) equal to zero. Then the equilibrium \( (A^{**}_k, I^{**}_k, T^{**}_k) \) should satisfy

\[ \alpha k \left( \frac{b}{b + \mu} - A_k(t) - I_k(t) - T_k(t) \right) \rho(t) - cA_k(t) - \mu A_k(t) = 0, \]
\[ (1 - \alpha)k \left( \frac{b}{b + \mu} - A_k(t) - I_k(t) - T_k(t) \right) \rho(t) + c(1 - \beta)A_k(t) \]
\[ + a(1 - \gamma)T_k(t) - dI_k(t) - \mu I_k(t) = 0, \]
\[ dI_k(t) - aT_k(t) - \mu T_k(t) = 0. \]

From (15), we have

\[ A^{**}_k = \frac{\alpha k\rho b[a(a + \mu)(1 - \gamma) - d(d + \mu)]}{(a + \mu + d)(b + \mu)H^*_k + B_k[a(a + \mu)(1 - \gamma) - d(d + \mu)]}, \]

\[ I^{**}_k = \frac{bdH^*_k}{(a + \mu + d)(b + \mu)H^*_k + B_k[a(a + \mu)(1 - \gamma) - d(d + \mu)]}, \]

\[ T^{**}_k = \frac{b(a + \mu)H^*_k}{((a + \mu + d)(b + \mu)H^*_k + B_k[a(a + \mu)(1 - \gamma) - d(d + \mu)]}, \]

where

\[ H^*_k = k\rho[c(\alpha\beta - 1) - \mu(1 - \alpha)], \quad B_k = (\alpha k\rho + c + \mu)(b + \mu) \]

and \( \rho \leq (\rho_1 + \rho_2) \). Obviously, when \( \rho = 0 \), \( A^{**}_k = 0, I^{**}_k = 0, T^{**}_k = 0 \), which is the alcohol-free equilibrium for (5).

Substituting \( A^{**}_k \) and \( I^{**}_k \) into \( \rho \), an equation of the form \( \rho f(\rho) = 0 \) is obtained, where

\[ f(\rho) = 1 - \left[ \frac{\sum_i i\varphi_1(i)p(i)}{\langle k \rangle} A + \frac{\sum_i i\varphi_2(i)p(i)}{\langle k \rangle} B \right], \]

\[ A = \frac{\rho_1ab[a(a + \mu)(1 - \gamma) - d(d + \mu)]}{(a + \mu + d)(b + \mu)H_i + B_i[a(a + \mu)(1 - \gamma) - d(d + \mu)]}, \]

\[ B = \frac{\rho_2bd[c(\alpha\beta - 1) - \mu(1 - \alpha)]}{(a + \mu + d)(b + \mu)H_i + B_i[a(a + \mu)(1 - \gamma) - d(d + \mu)]}. \]
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.

\[
R_1 = \frac{\langle k \varphi_1 (k) \rangle}{\langle k \rangle} \frac{\rho_1 ab}{(c + \mu)(b + \mu)} + \frac{\langle k \varphi_2 (k) \rangle}{\langle k \rangle} \frac{\rho_2 bd [c(\alpha \beta - 1) - \mu(1 - \alpha)]}{(c + \mu)(b + \mu)[a(a + \mu)(1 - \gamma) - d(d + \mu)]} > 1.
\]

The proof is completed.

Similar to the proofs of Theorems 4.1 and 4.2, we can have the following theorem of system (15).

**Theorem 5.2:** For system (15), there are two possibilities. Either \( R_1 < 1 \), the alcohol-free equilibrium \( E^1 \) is globally asymptotically stable in \( \Omega^* \), or \( R_1 > 1 \), there exists a constant solution \( E^{*} \in \Omega^* - \{0\} \), which is permanent, that is, there exists a constant \( \xi \) which satisfies \( \lim \inf_{t \to \infty} \{A_k(t), I_k(t), T_k(t)\}_{t=1}^n \geq \xi \), where \( (A_k(t), I_k(t), T_k(t)) \) is any solution of (15), satisfying (16) and \( A_k(0) > 0, I_k(0) > 0, T_k(0) > 0 \).

From the result of Theorem 5.2, we know that alcoholics will disappear if \( R_1 < 1 \), which means that alcoholism will disappear. Light and heavy alcoholics will maintain at a positive stationary value if \( R_1 > 1 \), which means that the alcoholism becomes an endemic ‘disease’.

6. Numerical simulations

In this section, we present some numerical simulations. Simulations are based on a scale-free network \([2,25]\) with \( P(k) = (r - 1)m^{(r-1)}k^{-r} \), where \( m \) represents the smallest degree on a scale-free network nodes; \( r \) is a variable of power-law exponent. Let \( m = 5, r = 5 \) and the number of nodes on a scale-free network is \( N = 100 \), and we add each new node with 5 new edges. We employ the iteration method and the difference method to calculate the density of \( S_k, A_k, I_k, T_k \) for different degrees of \( k \) as \( k_{\text{min}} = 5, k = 6, k = 7, k = 8, k = 9, k = 10, k = 11, k = 12, k = 13, k = 14, k = 15, k = 16, k = 17, k = 19, k = 20, k = 22, k = 23, k = 25, k = 26, k = 27, k = 29, k_{\text{max}} = 31. \) The dynamics of the nodes is implemented based on the BA network with 100 nodes (see Figure 2(a,b)).

Next, we verify and extend our theoretical results with numerical simulations using the above network. All the parameters are estimated. We let \( b = 0.4, \mu = 0.04 \).

Furthermore, we assume that \( c = 0.2, d = 0.4, a = 0.6, \alpha = 0.6, \beta = 0.8, \gamma = 0.8, \rho_1 = 0.01, \rho_2 = 0.01 \), then \( R_0 = 0.7480 < 1 \). From Figure 3(a), we know that alcoholics will disappear, which means that alcoholism will disappear. Let \( \rho_1 = 0.04, \rho_2 = 0.06 \), then \( R_0 = 3.6599 > 1 \). From Figure 3(b), we know that alcoholics population will maintain at a positive stationary level.

In Figure 4, we assume that \( c = 0.2, d = 0.4, a = 0.6, \alpha = 0.6, \beta = 0.8, \gamma = 0.8, \rho_1 = 0.01, \rho_2 = 0.01 \), then \( R_0 = 0.7480 < 1 \). Figure 4(a,b) shows the dynamics of light alcoholics and heavy alcoholics with different degrees on scale-free networks when \( R_0 < 1 \), respectively. One can see that light alcoholics and heavy alcoholics will tend to \( 0 \), which means that alcoholism will disappear. In Figure 5, we assume that \( c = 0.2, d = 0.4, a = 0.6, \alpha = 0.6, \beta = 0.8, \gamma = 0.8, \rho_1 = 0.01, \rho_2 = 0.01 \), then \( R_0 = 0.7480 < 1 \). Figure 5(a,b) shows the dynamics of light alcoholics and heavy alcoholics with different degrees on scale-free networks when \( R_0 < 1 \), respectively.
Figure 2. (a) Evolution of logarithm of node probability $P(k)$ versus logarithm of degree $k$ on a scale-free network with 100 nodes and $m = 5$; (b) A scale-free network with 100 nodes and $m = 5$.

Figure 3. Each compartment population changes over time on scale-free networks when $R_0 < 1$ and $R_0 > 1$.

$a = 0.6, \beta = 0.8, \gamma = 0.8, \rho_1 = 0.04, \rho_2 = 0.06$, then $R_0 = 3.6599 > 1$. Figure 5(a,b) shows the dynamics of light alcoholics and heavy alcoholics with different degrees on scale-free networks when $R_0 > 1$, respectively. One can see that light alcoholics and heavy alcoholics will maintain at a positive stationary value, which means that the alcoholism becomes an endemic ‘disease’. We also find that the larger degree leads to a larger value of the alcoholism level.

Figures 6 and 7 exhibit dynamical behaviour of light alcoholics and heavy alcoholics with different degrees on scale-free networks with nonlinear infectivity when $R_0 < 1$ and $R_0 > 1$, respectively. In Figure 6(a,b), we let $\varphi_1(k) = k^{r_1}, \varphi_2(k) = k^{r_2}, r_1 = 0.6, r_2 = 0.9, \rho_1 = 0.01, \rho_2 = 0.01, R_1 = 0.3254 < 1$. One can see that light alcoholics and heavy alcoholics will tend to 0. In Figure 7(a,b), we let $\rho_1 = 0.06, \rho_2 = 0.08, R_1 = 2.4022 > 1$. One can see that light alcoholics and heavy alcoholics will maintain at a positive stationary value. From Figures 5 and 7, we find that the positive steady value is lower than the case
Figure 4. Dynamical behaviour of light alcoholics and heavy alcoholics with different degrees on scale-free networks when $R_0 < 1$.

Figure 5. Dynamical behaviour of light alcoholics and heavy alcoholics with different degrees on scale-free networks when $R_0 > 1$.

Figure 6. Dynamical behaviour of light alcoholics and heavy alcoholics with different degrees on scale-free networks with nonlinear infectivity when $R_0 < 1$. 

without nonlinear infectivity, and we know that nonlinear infectivity plays an important role in the transmission of drinking.

7. Conclusions

The new SAITS binge drinking model on scale-free networks with demographic and nonlinear infectivity is introduced in this paper. For the model with demographic and linear infectivity, the basic reproduction number $R_0$ is obtained by using the next-generation method in [29]. The alcohol-free equilibrium is globally asymptotically stable if $R_0 < 1$, which means that alcoholism will disappear. When $R_0 > 1$, the model exists a unique alcoholism equilibrium and is permanent, which means that light and heavy alcoholics will maintain at a positive stationary value and the alcoholism becomes an endemic ‘disease’. Furthermore, the SAITS model with demographic and nonlinear infectivity is also studied. Existence and stability of all the equilibria are also obtained. Numerical simulations are also shown to verify and extended our theoretical results.

Our results show that the larger degree leads to a larger value of the alcoholism level. Demographic and nonlinear infectivity are very important factors which affect the spread of alcoholism.

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