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ANALYSIS OF THE BINGE DRINKING MODELS WITH DEMOGRAPHICS AND NONLINEAR INFECTIVITY ON NETWORKS

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Abstract Two new binge drinking models incorporating demographics on different weighted networks are investigated. First, the dynamics of the drinking model with the linear infectivity $\varphi(k) = k$ on the unweighted network is investigated. The basic reproduction number R_0 and the uniqueness and stability of all the equilibria are derived. Second, the model with the nonlinear infectivity $\varphi(k) = k^a (0 < a < 1)$ and two kinds of weights is introduced, and stability of all the equilibria is studied. At last, some simulations are presented to illustrate our analytic results. Our results show that the spread of drinking behaviors on the fixed weighted network is the most easily to break out, and the infectivity exponent also has a greater effect on the spread of drinking behaviors than that of the weight exponent.

Keywords Binge drinking, demographics, stability, nonlinear infectivity, weight networks.

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1. Introduction

Drinking is an increasingly serious problem, especially among the group of college students [1]. Injuries and deaths caused by alcohol increased by 6% per 100,000 college students from 1998 to 2001 [11]. There are about 3.8% of global deaths and 4.6% of global DALY (disability adjusted life years) attributable to alcohol [29]. Excessive drinking not only harms personal health, but also induces serious consequences for the family and society.

Nowadays many researchers investigate the drinking behaviors by constructing mathematical models [2, 28, 33, 34, 38]. Mubayi et al. [28] introduced a simple framework where drinking was modelled as a socially contagious process in low and high-risk connected environments. Bhunu [2] presented a deterministic model for the spread of alcoholism. Thomas and Lungu [33] constructed a two-sex model to analyze the influence of heavy drinking on HIV/AIDS among men and women, they found that binge drinking was a major driving force for HIV/AIDS. Wang et al. [34] constructed an alcohol quitting model with distributed time delay, and derived the optimal control strategies with the help of proposing an objective functional and using classic Pontryagin's Maximum Principle. Xiang [38] proposed a drinking model with public health educational campaigns, and concluded that

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the public health educational campaigns of drinking individuals can slow down the drinking dynamics.

Media coverage plays a crucial role in modern life. It will transmit a lot of health information when some diseases appear. So many scholars take into account media coverage in their models to study its impact on the spread of epidemic. Huo and Wang [15] considered the impact of media coverage on the drinking spread, they divided the health drinkers into two classes: $X(t)$, who will avoid contacting with binge drinkers for the reason of media coverage; $S(t)$, who will not be influenced by media coverage. Ma et al. [24] studied a dynamic alcohol consumption model with awareness programs and time delay, they found that awareness programs were effective measures in controlling the alcohol problems and the bifurcation might appear by increasing the value of time delay. Other related epidemic or population models, we refer to see [4, 5, 8, 10, 13, 17, 26, 27, 39, 44]

Above alcohol models are all based on the assumption of homogeneous mixing. A few drinking models are studied on complex networks [3, 16], but the epidemic spread models on complex networks have been studied extensively in recent years [7, 12, 18, 31, 35, 37, 41] and references cited therein. They constructed models on the networks, in which each node represents an individual in the real system, and each edge between two nodes denotes the relationship between individuals. The degree distribution of the complex networks is defined as $p(k) = \frac{N_k}{N}$, which means the probability that a node randomly chosen has k links. Most researches on epidemic behaviors are all investigated on the scale-free network, since it considers the growth and connection tendency of the real world. And the degree distribution on the scale-free network follows a power law $p(k) \sim ck^{-\gamma}$, with $2 < \gamma \leq 3$, where c satisfies the equality of $\sum_{k=1}^{n} p(k) = 1$ [23]. Huo and Wang [35] analyzed the impact of media coverage on the binge drinking model on the heterogeneous network, they assumed that the social network was a closed group which means that the total number of individuals in the process of the alcohol spread remained unchanged. But some behaviors will continue for a long time even until the end of individual life. Thus, it is necessary to consider the influence of individual birth and death. We will improve the original drinking model by introducing the empty nodes to maintain the static stability of the complex networks, this method has been used in many literature [20–22, 43] and references cited therein.

The weight of complex networks is often used to represent the intimacy of nodes, the greater the weight is, the more the intimacy is. Zhu et al. [45] introduced a modified SIS model on the adaptive weighted network, they employed a function $\omega(i, j) = g(i)g(j)$ of nodes' degrees to express the weights of links, where $g(i)$ was an increasing function of degree i. Chu et al. [6] proposed an SIR model in weighted scale free networks with the nonlinear infectivity. They found that the infectivity exponent had a stronger impact on the epidemic prevalence than the weight exponent. Thus, the expression of infectivity is the key to the model, it affects the dynamic behavior largely. Various forms of infectivity were proposed to improve the actuality of models [14, 30, 36, 40, 42].

Motivated by the above, we set up binge drinking models with demographics and nonlinear infectivity on weighted networks, and study dynamics of binge drinking models. Our results show that the drinking behaviors spread on the fixed weighted network is the most easily to break out and the infectivity exponent has a greater effect on the drinking threshold than the weight exponent.

The organization of this paper is as follows: In Section 2, we present a binge

drinking model with demographics and linear infectivity $\varphi(k) = k$ on unweighted scale-free network, and analyze the dynamic properties of the model. In Section 3, we modify the model by introducing different weights of networks and nonlinear infectivity $\varphi(k) = k^a (0 < a < 1)$. In Section 4, we perform some numerical simulations. Some conclusions are also given in last section.

2. The model with demographics and linear infectivity on unweighted networks

2.1. System description

In our model, we divide the total population N into n (n is the maximum degree) groups according to the degree of nodes. Each group is divided into three subgroups according to the alcohol consumption: $S_k(t)$ represents the density of nondrinkers or moderate drinkers, $X_k(t)$ represents the aware population who avoid contacting with heavy drinkers due to media coverage, $I_k(t)$ represents the heavy drinkers, respectively. Then $N_k(t) = S_k(t) + X_k(t) + I_k(t), k = 1, 2, ..., n$. $M(t)$ represents the cumulative density of awareness programs driven by media, it is proportional to the total density of binge drinkers. Then there are three kinds of nodes' states in our model.

Similar to that of [21] , we take a number set $\{0, 1, 2, 3\}$ to express the subgroup in our model, where 0 represents the empty state, 1 represents the nondrinkers or moderate drinkers state, 2 represents the aware population state, and 3 represents the heavy drinkers state. Different states can transform from each other. Taking the empty node as the example, it can change the state to the health drinkers at rate b, this progress means that there are some new individuals birth. If some people die, they will turn into the empty state, let the death rate is d. Similarly, nondrinkers or moderate drinkers turn to heavy drinkers at rate β. Heavy drinkers recover at rate μ. Awareness disseminates among nondrinkers or moderate drinkers at rate α. When the awareness of avoiding contacting with alcoholics is gradually fading, X_k will no longer consciously cut off the contact with alcoholics. Then they will return to S_k at rate σ. ω represents the growth rate of media coverage. γ represents the depletion rate of media coverage resulted by ineffective measures. All the parameters can be found in Table 1, and are positive constants.

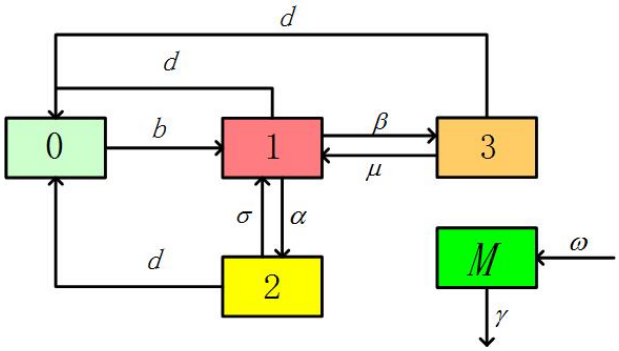


Figure 1. Transfer diagram of model (2.1).

The model's structure is shown in Figure 1. The transfer diagram leads to the following system of $3n+1$ ordinary differential equations

$$\begin{aligned}
\frac{dS_k(t)}{dt} &= b(1-S_k-X_k-I_k)-\beta k S_k\theta(t)-\alpha S_k M+\sigma X_k+\mu I_k-dS_k,\\
\frac{dX_k(t)}{dt} &= \alpha S_k M-\sigma X_k-dX_k,\\
\frac{dI_k(t)}{dt} &= \beta k S_k\theta(t)-\mu I_k-dI_k, k=1,2,...,n,\\
\frac{dM(t)}{dt} &= \omega\sum_{k=1}^{n}p(k)I_k-\gamma M.
\end{aligned} \tag{2.1}$$

Table 1. The parameters description of model (2.1).

Parameter	Description
b	The birth rate of population.
d	The death rate of population.
β	The transmission coefficient for nondrinkers or moderate drinkers turning to heavy drinkers.
μ	The recovery rate of heavy drinkers.
α	The dissemination rate of awareness among nondrinkers or moderate drinkers.
σ	The transformation rate from aware individuals to nondrinkers or moderate drinkers.
ω	The growth rate of media coverage.
γ	The depletion rate of media resulted by ineffective measures.

For the node with degree k, which means that the number of links connected to the node is k. Let the proportion be $\frac{\varphi(k)}{k}$, where $\varphi(k)$ denotes the infectivity of a k-degree node and it is less than or equal to k. Then $\theta(t)=\sum_{k=1}^{n}p(k|i)\frac{\varphi(k)}{k}I_k(t)$ represents the probability that an edge of nondrinkers or moderate drinkers links to binge drinkers, and it is between 0 and $\frac{b}{b+d}$. For simplicity, we consider the uncorrelated networks where the conditional probability satisfies $p(k|i)=kp(k)/\langle k\rangle$ [22], where $\langle k\rangle=\sum_{k=1}^{n}kp(k)$, then $\theta(t)=\frac{1}{\langle k\rangle}\sum_{k=1}^{n}\varphi(k)p(k)I_k$. We first study the dynamics of the drinking model with linear infectivity $\varphi(k)=k$ on unweighted network, i.e., $\theta(t)=\frac{1}{\langle k\rangle}\sum_{k=1}^{n}kp(k)I_k$.

2.2. Positivity and boundedness of solutions

Lemma 2.1. *Let $(S_1(t),\ X_1(t),\ I_1(t),\ \ldots,\ S_n(t),\ X_n(t),\ I_n(t),\ M(t))$ be the solution of system (2.1), if $S_k(0)>0,\ X_k(0)>0,\ I_k(0)>0,\ M(0)>0$ and $\theta(0)>0$, then for $k=1,2,...,n$, we have $S_k(t)>0, X_k(t)>0, I_k(t)>0,\ M(t)>0$ and $\theta(t)>0$ for all $t>0$.*

Proof. Substituting the third equation of system (2.1) into the formula of $\theta(t)$, we get

$$\theta'(t)=\frac{1}{\langle k\rangle}\sum_{k=1}^{n}kp(k)I'_k(t)=\theta(t)[\frac{1}{\langle k\rangle}\sum_{k=1}^{n}kp(k)\beta kS_k(t)-(\mu+d)],$$

it implies that

$$\theta(t) = \theta(0)exp[-(\mu + d)t + \frac{1}{\langle k \rangle} \int_0^t \sum_{k=1}^{n} kp(k)\beta k S_k(\tau)d\tau].$$

Since $\theta(0) > 0$, we obtain $\theta(t) > 0$ for all $t > 0$. Using the continuity of $S_k(t)$, since $S_k(0) > 0$, we can find a small $\delta > 0$, such that $S_k(t) > 0$ for $0 < t < \delta$. Now we prove that $S_k(t) > 0$ for all $t > 0$. If not, we assume a contradiction that there exists $t_1 \geq \delta > 0$, such that $S_k(t_1) = 0$ and $S_k(t) > 0$ for all $0 < t < t_1$. From the third equation of system (2.1), we have

$$I_k'(t) + (\mu + d)I_k(t) = \beta k S_k(t)\theta(t) > 0, 0 < t < t_1,$$

then

$$I_k(t) > I_k(0)e^{-(\mu+d)t} > 0, 0 < t < t_1.$$

From the last equation of system (2.1), we have

$$M'(t) > 0 - \gamma M(t), 0 < t < t_1,$$

it follows that

$$M(t) > M(0)e^{-\gamma t} > 0, 0 < t < t_1.$$

Similarly, from the second equation of system (2.1), we have

$$X_k'(t) + (\sigma + d)X_k(t) = \alpha S_k(t)M(t) > 0, 0 < t < t_1,$$

then

$$X_k(t) > X_k(0)e^{-(\sigma+d)t} > 0, 0 < t < t_1.$$

Using the continuity of $X_k(t)$ and $I_k(t)$, we have $X_k(t_1) \geq 0$ and $I_k(t_1) \geq 0$. Adding the first three equations of system (2.1), for all $t \geq 0$, we have

$$\frac{dN_k(t)}{dt} = b - (b + d)N_k(t),$$

since

$$N_k(0) = S_k(0) + X_k(0) + I_k(0) > 0,$$

then

$$N_k(t) = \frac{b}{b+d} + N_k(0)e^{-(b+d)t} \leq \frac{b}{b+d},$$

and

$$S_k(t_1) + X_k(t_1) + I_k(t_1) \leq \frac{b}{b+d} < 1,$$

so

$$S_k'(t_1) = b[1 - (S_k(t_1) + X_k(t_1) + I_k(t_1))] + \sigma X_k(t_1) + \mu I_k(t_1) > 0.$$

That is to say, $S_k(t) < 0$ for $0 < t < t_1$, which is contradictory. Thus $S_k(t) > 0$ for all $t > 0$. Similarly, we can prove that $X_k(t) > 0$, $I_k(t) > 0$ and $M(t) > 0$ for all $t > 0$. Hence the proof is completed. □

Lemma 2.2. *All feasible solutions of system (2.1) are in the following bounded region*

$$\Omega = \Big\{(S_1(t), X_1(t), I_1(t), \ldots, S_n(t), X_n(t), I_n(t), M(t)) \in R_+^{3n+1} \,|0 \le S_k(t), X_k(t), I_k(t) \le \frac{b}{b+d}, S_k(t) + X_k(t) + I_k(t) \le \frac{b}{b+d}, 1 \le k \le n, 0 \le M(t) \le \frac{\omega}{\gamma}\Big\}. \tag{2.2}$$

Proof. $S_k(t) + X_k(t) + I_k(t) \le \frac{b}{b+d}$ and the positivity of solutions have been proved in lemma 2.1, it implies that $0 \le S_k(t), X_k(t), I_k(t) \le \frac{b}{b+d}$. From the last equation of system (2.1), we have

$$0 - \gamma M \le M'(t) \le \omega - \gamma M,$$

it follows that

$$0 \le M(0)e^{-\gamma t} \le M(t) \le \frac{\omega}{\gamma} + M(0)e^{-\gamma t},$$

thus

$$\lim_{t\to\infty} \sup M(t) \le \frac{\omega}{\gamma}.$$

So the region Ω is a positively invariant set of system (2.1). This completes the proof of Lemma 2.2. □

2.3. The basic reproduction number

The asymptotic theory of autonomous systems in [32] shows that dynamics of the original and the limiting system are consistent asymptotically. Then we study the dynamics of the limiting system of system (2.1), the limiting system can be written as

$$\begin{aligned} \frac{dX_k(t)}{dt} &= \alpha(\frac{b}{b+d} - X_k - I_k)M - (\sigma + d)X_k, \\ \frac{dI_k(t)}{dt} &= \beta k(\frac{b}{b+d} - X_k - I_k)\theta(t) - (\mu + d)I_k, k = 1, 2, ..., n, \\ \frac{dM(t)}{dt} &= \omega \sum_{k=1}^{n} p(k)I_k - \gamma M. \end{aligned} \tag{2.3}$$

Let

$$\Gamma = \Big\{(X_1(t), I_1(t), \cdots, X_n(t), I_n(t), M(t)) \in R_+^{2n+1} \Big| 0 \le X_k(t), I_k(t) \le \frac{b}{b+d}, 0 \le X_k(t) + I_k(t) \le \frac{b}{b+d}, 1 \le k \le n, 0 \le M(t) \le \frac{\omega}{\gamma}\Big\}. \tag{2.4}$$

It can be verified that region Γ is a positively invariant set of system (2.3). System (2.3) has a unique alcohol free equilibrium $E_0 = (0,\ 0,\ \ldots,\ 0,\ 0, \ldots,\ 0,\ 0,\ 0)$. Using the next generation matrix method [9], we calculate the basic reproduction

number $R_0 = \rho(FV^{-1})$. In our case, the production of new binge drinkers $\mathscr{F}$ and the rate of transfer of individuals $\mathscr{V}$ are given by

$$\mathscr{F} = \begin{pmatrix} \beta(\frac{b}{b+d} - X_1 - I_1)\theta \\ \beta 2(\frac{b}{b+d} - X_2 - I_2)\theta \\ \vdots \\ \beta n(\frac{b}{b+d} - X_n - I_n)\theta \\ 0 \\ 0 \\ \vdots \\ 0 \\ 0 \end{pmatrix}_{2n+1}, \mathscr{V} = \begin{pmatrix} (\mu+d)I_1 \\ (\mu+d)I_2 \\ \vdots \\ (\mu+d)I_n \\ (\sigma+d)X_1 - \alpha(\frac{b}{b+d} - X_1 - I_1)M \\ (\sigma+d)X_2 - \alpha(\frac{b}{b+d} - X_2 - I_2)M \\ \vdots \\ (\sigma+d)X_n - \alpha(\frac{b}{b+d} - X_n - I_n)M \\ \gamma M - \omega \sum_{k=1}^{n} p(k)I_k \end{pmatrix}_{2n+1}.$$

Calculating the Jacobian matrices of $\mathscr{F}$ and $\mathscr{V}$ at E_0 as follows

$$F = D\mathscr{F}(E_0) = \begin{pmatrix} F_{11} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}_{(2n+1)\times(2n+1)},$$

$$V = D\mathscr{V}(E_0) = \begin{pmatrix} V_{11} & 0 & 0 \\ 0 & V_{22} & V_{23} \\ V_{31} & 0 & V_{33} \end{pmatrix}_{(2n+1)\times(2n+1)},$$

where

$$F_{11} = \frac{\beta b}{(b+d)\langle k\rangle} \begin{pmatrix} p(1) & 2p(2) & \cdots & np(n) \\ 2p(1) & 2^2p(2) & \cdots & 2np(n) \\ \vdots & \vdots & \ddots & \vdots \\ np(1) & n2p(2) & \cdots & n^2p(n) \end{pmatrix}_{n\times n},$$

$$V_{23} = (\, -\tfrac{\alpha b}{b+d} \; -\tfrac{\alpha b}{b+d} \; \cdots \; -\tfrac{\alpha b}{b+d} \,)_n^T,$$

$$V_{31} = \Big(-\omega p(1) \; -\omega p(2) \; \cdots \; -\omega p(n) \Big)_n,$$

$V_{11} = (\mu+d)E, V_{22} = (\sigma+d)E, V_{33} = \gamma$, E represents a unit matrix and 0 represents a zero matrix. We get the basic reproduction number R_0 as follows

$$R_0 = \frac{\beta b\langle k^2\rangle}{(b+d)(\mu+d)\langle k\rangle}.$$

2.4. The stability of the alcohol free equilibrium

Using the Theorem 2 of [9], we have the following result on the local stability of E_0.

Theorem 2.1. *The alcohol free equilibrium E_0 of system (2.3) is locally asymptotically stable when $R_0 < 1$, but unstable when $R_0 > 1$.*

We will prove the globally asymptotically stability of E_0 of system (2.3) in the following theorem.

Theorem 2.2. *The alcohol free equilibrium E_0 of system (2.3) is globally asymptotically stable when $R_0 < 1$.*

Proof. Let $I_1 = y_1, I_2 = y_2, \ldots, I_n = y_n, X_1 = y_{n+1}, X_2 = y_{n+2}, \ldots, X_n = y_{2n}, M = y_{2n+1}, y = (y_1, y_2, \ldots, y_{2n+1})^T$, $g(j) = \frac{jp(j)}{\langle k \rangle}$, then the vectorial form of system (2.3) can be written as follows

$$\frac{dy}{dt} = Ay + N(y), \tag{2.5}$$

where

$$A = \begin{pmatrix} A_{11} & 0 & 0 \\ 0 & A_{22} & A_{23} \\ A_{31} & 0 & A_{33} \end{pmatrix},$$

$$A_{11} = \begin{pmatrix} \frac{\beta b}{b+d}g(1) - (\mu+d) & \frac{\beta b}{b+d}g(2) & \cdots & \frac{\beta b}{b+d}g(n) \\ \frac{\beta b}{b+d}2g(1) & \frac{\beta b}{b+d}2g(2) - (\mu+d) & \cdots & \frac{\beta b}{b+d}2g(n) \\ \vdots & \vdots & \ddots & \vdots \\ \frac{\beta b}{b+d}ng(1) & \frac{\beta b}{b+d}ng(2) & \cdots & \frac{\beta b}{b+d}ng(n) - (\mu+d) \end{pmatrix}_{n\times n},$$

$$A_{23} = \left(\frac{\alpha b}{b+d} \ \frac{\alpha b}{b+d} \cdots \frac{\alpha b}{b+d} \right)_n^{\mathrm{T}},$$

$$A_{31} = \left(\omega p(1) \ \omega p(2) \cdots \omega p(n) \right)_n,$$

$$A_{22} = -(\sigma+d)E, A_{33} = -\gamma,$$

and

$$N(y) = -\begin{pmatrix} \beta\theta(y_1 + y_{n+1}) \\ 2\beta\theta(y_2 + y_{n+2}) \\ \vdots \\ n\beta\theta(y_n + y_{2n}) \\ \alpha y_{2n+1}(y_1 + y_{n+1}) \\ \alpha y_{2n+1}(y_2 + y_{n+2}) \\ \vdots \\ \alpha y_{2n+1}(y_n + y_{2n}) \\ 0 \end{pmatrix}_{2n+1}.$$

Thus

$$\frac{dy}{dt} \leq Ay. \tag{2.6}$$

Considering the following linear system

$$\frac{dy}{dt} = Ay. \tag{2.7}$$

If $R_0 < 1$, all eigenvalues of A have negative real parts [9]. It means that system (2.7) is stable whenever $R_0 < 1$. Then $I_k(t) \to 0, X_k(t) \to 0, M(t) \to 0$, when $t \to \infty$, for this linear system. Since (2.7) is a quasi monotone system, by citing the comparison theorem [19], we can get the result is that the nonlinear system (2.3) satisfies that $I_k(t) \to 0, X_k(t) \to 0, M(t) \to 0$, as $t \to \infty$, when $R_0 < 1$. So the alcohol free equilibrium E_0 of system (2.3) is globally asymptotically stable. The proof is complete. □

2.5. The uniqueness of the alcohol present equilibrium

Theorem 2.3. *If $R_0 > 1$, system (2.3) has a unique alcohol present equilibrium*

$$E^*(X_1^*, I_1^*, ..., X_n^*, I_n^*, M^*).$$

Proof. Let the right side of system (2.3) be 0, and $I^* = \sum_{k=1}^{n} p(k)I_k^* > 0$. We obtain the following system

$$\begin{aligned} \alpha(\frac{b}{b+d} - X_k^* - I_k^*)M^* - (\sigma + d)X_k^* = 0, \\ \beta k(\frac{b}{b+d} - X_k^* - I_k^*)\theta - (\mu + d)I_k^* = 0, \\ \omega \sum_{k=1}^{n} p(k)I_k^* - \gamma M^* = 0, \end{aligned} \tag{2.8}$$

From the third equation of system (2.8), we have

$$M^* = \frac{\omega I^*}{\gamma}. \tag{2.9}$$

Taking (2.9) into the first equation of system (2.8), we have

$$X_k^* = \frac{\alpha\omega b I^* - \alpha\omega(b+d)I^* I_k^*}{\alpha\omega(b+d)I^* + \gamma(\sigma+d)(b+d)}. \tag{2.10}$$

Substituting (2.10) into the second equation of system (2.8), we obtain

$$\alpha\omega(\mu+d)(b+d)I^* I_k^* + \beta k\theta\gamma(\sigma+d)(b+d)I_k^* + \gamma(\sigma+d)(\mu+d)(b+d)I_k^* - \beta k\theta b\gamma(\sigma+d) = 0. \tag{2.11}$$

Multiplying equation (2.11) by $p(k)$ and summing over k, we get

$$\alpha\omega(\mu+d)(b+d)(I^*)^2 + \gamma(\sigma+d)(\mu+d)(b+d)I^* - \beta\gamma(\sigma+d)\langle k\rangle\theta[b-(b+d)\theta] = 0. \tag{2.12}$$

According to the definition of I^*, we have

$$I^* = \frac{-\gamma(\mu+d)(b+d)(\sigma+d)+\sqrt{\Delta}}{2\alpha\omega(\mu+d)(b+d)}, \tag{2.13}$$

where

$$\Delta = [\gamma(\mu+d)(\sigma+d)(b+d)]^2 + 4\alpha\omega\beta\gamma\langle k\rangle(\mu+d)(\sigma+d)(b+d)\theta[b-(b+d)\theta] > 0,$$

since $\theta \in [0, \frac{b}{b+d}]$. On the other hand, we get the following equation from (2.11) is that

$$I_k^* = \frac{\beta k\theta b\gamma(\sigma+d)}{\alpha\omega(\mu+d)(b+d)I^* + \gamma(\sigma+d)(b+d)(\beta k\theta+\mu+d))}. \tag{2.14}$$

Substituting (2.13) into (2.14), we have

$$I_k^* = \frac{2\beta k\theta b\gamma(\sigma+d)}{\gamma(\sigma+d)(b+d)(2\beta k\theta+\mu+d)+\sqrt{\Delta}}. \tag{2.15}$$

Substituting (2.15) into the expression of $\theta(t) = \frac{1}{\langle k\rangle}\sum\limits_{k=1}^{n} kp(k)I_k^*$, then we obtain a self-consistency equation as follows

$$\theta(t) = \frac{1}{\langle k\rangle}\sum_{k=1}^{n} kp(k)\frac{2\beta k\theta b\gamma(\sigma+d)}{\gamma(\sigma+d)(b+d)(2\beta k\theta+\mu+d)+\sqrt{\Delta}}. \tag{2.16}$$

If we let

$$f(\theta) = 1 - \frac{2}{\langle k\rangle}\sum_{k=1}^{n} kp(k)\frac{\beta kb\gamma(\sigma+d)}{\gamma(\sigma+d)(b+d)(2\beta k\theta+\mu+d)+\sqrt{\Delta}}, \tag{2.17}$$

then equation (2.16) is equivalent to the following equation

$$\theta f(\theta) = 0. \tag{2.18}$$

Obviously, equation (2.18) has a trivial solution $\theta = 0$. For

$$f'(\theta) = \frac{2}{\langle k\rangle}\sum_{k=1}^{n} kp(k)\frac{A}{B^2}, \tag{2.19}$$

where

$$A = 2\gamma^2(\sigma+d)^2\beta^2(b+d)bk[k+\alpha\omega(\mu+d)\langle k\rangle[b-2(b+d)\theta]/\sqrt{\Delta}],$$
$$B = \gamma(\sigma+d)(b+d)(2\beta k\theta+\mu+d)+\sqrt{\Delta} > 0,$$

hence, $f'(0) > 0$. Through a similar derivation, we obtain that

$$f''(\theta) = \frac{2}{\langle k\rangle}\sum_{k=1}^{n} kp(k)\frac{B^2\frac{dA}{d\theta} - 2AB\frac{dB}{d\theta}}{B^4}, \tag{2.20}$$

where

$$\frac{dA}{d\theta} = \frac{4\gamma^2(\sigma+d)^2\beta^2(b+d)^2(\mu+d)\alpha\omega bk}{\Delta}C < 0,$$

since

$$C = -\langle k\rangle\sqrt{\Delta} - \frac{\alpha\omega(\mu+d)[b-2(b+d)\theta]^2\langle k\rangle^2\beta\gamma(\sigma+d)}{\sqrt{\Delta}} < 0.$$

And

$$\frac{dB}{d\theta} = 2\gamma(\sigma+d)(b+d)\beta[k + \frac{\alpha\omega(\mu+d)\langle k\rangle[b-2(b+d)\theta]}{\sqrt{\Delta}}],$$

then

$$A\frac{dB}{d\theta} = 4\gamma^3\beta^3(\sigma+d)^3(b+d)^2bk[k + \frac{\alpha\omega(\mu+d)\langle k\rangle[b-2(b+d)\theta]}{\sqrt{\Delta}}]^2 > 0,$$
$$B^2\frac{dA}{d\theta} - 2AB\frac{dB}{d\theta} = B(B\frac{dA}{d\theta} - 2A\frac{dB}{d\theta}) < 0,$$

thus $f''(\theta) < 0$, that is to say, $f(\theta)$ is a convex function for $\theta \in [0, \frac{b}{b+d}]$. Furthermore, when $R_0 > 1$,

$$\begin{aligned} f(\frac{b}{b+d}) &= 1 - \frac{1}{\langle k\rangle}\sum_{k=1}^{n} kp(k)\frac{\beta kb\gamma(\sigma+d)}{\beta kb\gamma(\sigma+d) + \gamma(\sigma+d)(\mu+d)(b+d)} \\ &> 1 - \frac{\sum_{k=1}^{n} kp(k)}{\langle k\rangle} = 0, \end{aligned}$$

$$\begin{aligned} f(0) &= 1 - \frac{2}{\langle k\rangle}\sum_{k=1}^{n} kp(k)\frac{\beta kb\gamma(\sigma+d)}{2\gamma(\sigma+d)(\mu+d)(b+d)} = 1 - \frac{\beta b\sum_{k=1}^{n} k^2p(k)}{(\mu+d)(b+d)\langle k\rangle} \\ &= 1 - R_0 < 0. \end{aligned}$$

So there exists a unique positive equilibrium $E^*(X_1^*, I_1^*, ..., X_n^*, I_n^*, M^*)$ of system (2.3). The proof is complete. □

3. Models with demographics and nonlinear infectivity on weighted networks

In this section, we study the binge drinking model (2.1) with different weights of the uncorrelated network and nonlinear infectivity $\varphi(k) = k^a (0 < a < 1)$. Firstly, we introduce the new model on the fixed weighted network, then model (2.1) is modified as follows

$$\begin{aligned} \frac{dS_k(t)}{dt} &= b(1 - S_k - X_k - I_k) - kS_k\Theta_k(t) - \alpha S_kM + \sigma X_k + \mu I_k - dS_k, \\ \frac{dX_k(t)}{dt} &= \alpha S_kM - \sigma X_k - dX_k, \\ \frac{dI_k(t)}{dt} &= kS_k\Theta_k(t) - \mu I_k - dI_k, k = 1, 2, ..., n, \\ \frac{dM(t)}{dt} &= \omega\sum_{k=1}^{n} p(k)I_k - \gamma M. \end{aligned} \tag{3.1}$$

$kS_k\Theta_k(t)$ denotes newly drinkers per unit time, where $\Theta_k(t) = \sum_i p(i|k)\frac{\varphi(i)}{i}\beta_{ik}I_i(t)$ $= \sum_i \frac{i^a p(i)}{\langle k\rangle}\beta_{ik}I_i(t)$ is the probability that the drinking behaviors transmit through

a link. β_{ik} represents the transmission rate from nodes with degree i to nodes with degree k. S_k, X_k, I_k, M and all other parameters have the same meaning as model (2.1).

There are many forms of weighted expressions on complex networks. In this paper, we use nodes' degrees to express edges' weight. The edges' weight between two nodes with degree i and k can be expressed as a function $\omega(i,k) = f(i)f(k)$, where $f(i)$ is an increasing function of degree i since the individuals whose degree is large will have great influence on the social network, thus, their weight should be great. In this paper, we take $f(i) = i^h$ [45], h depends on the networks, e.g., for the Escherichia coli matabolic network $h = 0.5$, for the US airport network $h = 0.8$ [25].

ψ_k is the weight of nodes with degree k, it is the sum of links' weights connected with the node, i.e., $\psi_k = k\sum_i p(i|k)\omega(i,k) = k^{h+1}\langle k^{h+1}\rangle/\langle k\rangle$. For the i-degree node, we assume it has a fixed total transmission rate which is given by βi [6, 45], and the transmission rate through an edge from the i-degree node to the k-degree node is measured by the proportion of the edge's weight accounting for the i-degree node's weight, i.e., $\beta_{ik} = \beta i\frac{\omega(i,k)}{\psi_i} = \beta k^h\langle k\rangle/\langle k^{h+1}\rangle$. It means that the greater proportion of ψ_i the weight $\omega(i,k)$ of a link accounting for, the greater chance the k-degree node will be impacted to drinking. Thus $\Theta_k = \beta k^h\sum_i i^a p(i)I_i/\langle k^{h+1}\rangle$.

Using the F-V method [9], we get the production of new binge drinkers $\mathscr{F}$ and the rate of transfer of individuals $\mathscr{V}$ as follows

$$\mathscr{F} = \begin{pmatrix} (\frac{b}{b+d} - X_1 - I_1)\Theta_1 \\ 2(\frac{b}{b+d} - X_2 - I_2)\Theta_2 \\ \vdots \\ n(\frac{b}{b+d} - X_n - I_n)\Theta_n \\ 0 \\ 0 \\ \vdots \\ 0 \\ 0 \end{pmatrix}_{2n+1}, \mathscr{V} = \begin{pmatrix} (\mu+d)I_1 \\ (\mu+d)I_2 \\ \vdots \\ (\mu+d)I_n \\ (\sigma+d)X_1 - \alpha(\frac{b}{b+d} - X_1 - I_1)M \\ (\sigma+d)X_2 - \alpha(\frac{b}{b+d} - X_2 - I_2)M \\ \vdots \\ (\sigma+d)X_n - \alpha(\frac{b}{b+d} - X_n - I_n)M \\ \gamma M - \omega\sum_{k=1}^{n} p(k)I_k \end{pmatrix}_{2n+1}.$$

Calculating the Jacobian matrices of $\mathscr{F}$ and $\mathscr{V}$ at $E_1 = (0,\ 0,\ \ldots,\ 0,\ 0,\ 0)$ as follows

$$F = D\mathscr{F}(E_0) = \begin{pmatrix} F_{11} & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}_{(2n+1)\times(2n+1)},$$

$$V = D\mathscr{V}(E_0) = \begin{pmatrix} V_{11} & 0 & 0 \\ 0 & V_{22} & V_{23} \\ V_{31} & 0 & V_{33} \end{pmatrix}_{(2n+1)\times(2n+1)},$$

where

$$F_{11} = \frac{\beta b}{(b+d)\langle k^{h+1}\rangle} \begin{pmatrix} p(1) & 2^a p(2) & \cdots & n^a p(n) \\ 2^{h+1}p(1) & 2^{h+1}2^a p(2) & \cdots & 2^{h+1}n^a p(n) \\ \vdots & \vdots & \ddots & \vdots \\ n^{h+1}p(1) & n^{h+1}2^a p(2) & \cdots & n^{h+1}n^a p(n) \end{pmatrix}_{n\times n},$$

$$V_{23} = \left(-\tfrac{\alpha b}{b+d} \ -\tfrac{\alpha b}{b+d} \ \cdots \ -\tfrac{\alpha b}{b+d}\right)_n^T,$$

$$V_{31} = \left(-\omega p(1) \ -\omega p(2) \ \cdots \ -\omega p(n)\right)_n,$$

$V_{11} = (\mu+d)E, V_{22} = (\sigma+d)E, V_{33} = \gamma$, E represents a unit matrix and 0 represents a zero matrix. The basic reproduction number R_1 of model (3.1) is calculated as follows

$$R_1 = \frac{\beta b \left\langle k^{a+h+1}\right\rangle}{(b+d)(\mu+d)\left\langle k^{h+1}\right\rangle}.$$

Theorem 3.1. *The alcohol free equilibrium E_1 of system (3.1) is locally asymptotically stable if $R_1 < 1$, but unstable if $R_1 > 1$.*

Secondly, we investigate the drinking model on the adaptive weighted network which considers the action of individuals' health conscious. When the phenomenon of alcoholism has become more and more serious, individuals will take some measures to avoid being infected to drink. Then the weight functions represented by the density of binge drinkers at time t becomes more significantly [45]. The edges' weight $\omega'(i,k)$ between two nodes with degree i and k on the adaptive weighted networks can be expressed as

$$\omega'(i,k) = \omega_0 i^h \exp(-c(i)I(t))k^h \exp(-c(k)I(t)),$$

where $c(k) = k^\rho$ is a non-decreasing function of k.

The node's weight with degree k on the adaptive weighted networks is improved as follows

$$\psi_i' = \omega_0 i^{h+1}\left[\exp(-c(i)I(t))\right]\left\langle k^{h+1}\exp(-c(k)I(t))\right\rangle / \langle k\rangle.$$

The corresponding transmission rate β_{ik}' on the adaptive weighted networks becomes

$$\beta_{ik}' = \frac{\beta k^h \langle k\rangle \exp(-c(k)I(t))}{\langle k^{h+1}\exp(-c(k)I(t))\rangle}.$$

Substituting β_{ik}' into the expression of the probability Θ_k' that the drinking behaviors transmit through a link on the adaptive networks, then we have that

$$\Theta_k' = \frac{\beta k^h \exp(-c(k)I(t))}{\langle k^{h+1}\exp(-c(k)I(t))\rangle}\left\langle k^a I_k(t)\right\rangle.$$

When $c(k) \neq 0$, $h \neq 0$, the network is the adaptive weighted network. When $c(k) = 0$, $h \neq 0$, then $\Theta_k' = \Theta_k$, the network is the fixed weighted network. When $c(k) = 0$, $h = 0$, the network is the unweighted network. The adaptive factor can not change the value of the basic reproduction number, but it brings a great effect to the drinking behavior [45].

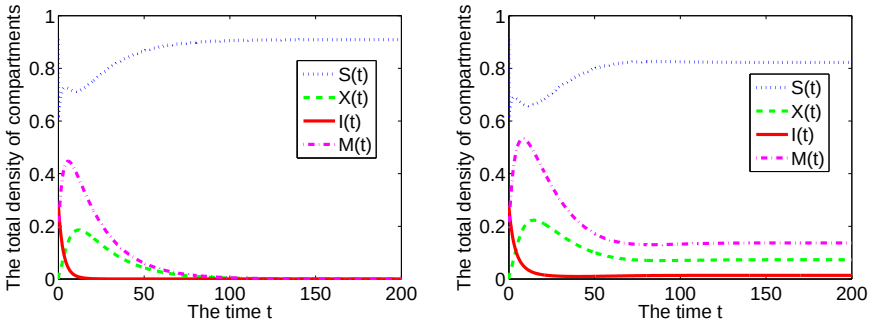
4. Numerical simulation and sensitivity analysis

In this section, we will simulate the drinking behaviors on the unweighted, fixed and adaptive networks, respectively, and compare the impact of infectivity exponent a on the binge drinking models. In the scale-free networks, we take 100 nodes to simulate the drinking model, $p(k) = 18k^{-3}$, other parameters are chosen in Table 2.

Table 2. The parameter values of the model.

Parameter	Data estimated	Data sources
b	0.2 $year^{-1}$	*estimate*
d	0.02 $year^{-1}$	*estimate*
β	0.02 $year^{-1}$	[16]
μ	0.4 $year^{-1}$	[16]
α	0.1 $year^{-1}$	[16]
σ	0.2 $year^{-1}$	[16]
ω	0.5 $year^{-1}$	[43]
γ	0.05 $year^{-1}$	[43]

Figure 2 simulates the trend of compartments' density of S_k, X_k, I_k, M changing with time on the unweighted networks with the linear infectivity. When $\beta = 0.02$, we have $R_0 = 0.4146 < 1$, and $I_k(t) \to 0$, which means the phenomenon of alcohol abuse will disappear (figure 2(a)). When $\beta = 0.05$, we get $R_0 = 1.0366 > 1$, and S_k, X_k, I_k, M all tend to a nonzero constant when $t \to +\infty$ (figure 2(b)), i.e., the phenomenon of alcohol abuse will be permanent, and eventually reaches a steady state.

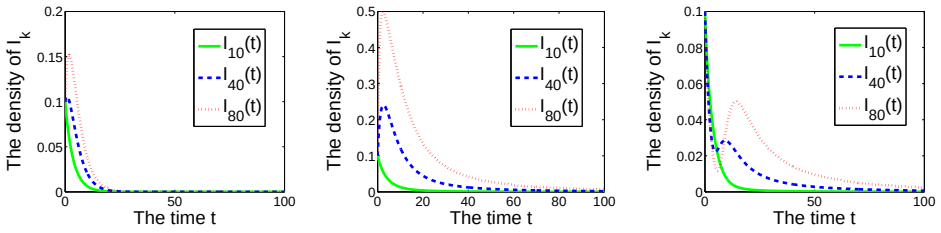


(a) $R_0 < 1$ (b) $R_0 > 1$

Figure 2. The trend of S_k, X_k, I_k, M on the unweighted networks with the linear infectivity.

The basic reproduction number is small than one ($R_0 = 0.4146$ in the unweighted networks; $R_1 = 0.9457$ in the fixed and adaptive weighted networks), $a = 1$ and $\beta = 0.02$ in figure 3. It simulates the trend of I_k ($k = 10, 40, 80$) changing with time on different networks. We find that I_k all tend to 0, but the peak of I_k with the same degree is the highest in the fixed weighted networks (figure 3(b)) and is the lowest in the adaptive weighted networks (figure 3(c)). It means that the drinking behavior is the most easily to spread in the fixed weighted networks, furthermore, the self-protection awareness of health drinkers guides them to reduce the contact

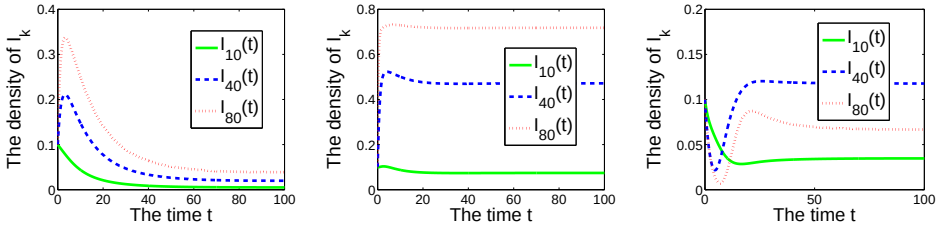
with alcoholics, then the risk of be infected will become low in the adaptive weighted networks.



(a) unweight (b) fixed weight (c) adaptive weight

Figure 3. The trend of I_k $(k = 10, 40, 80)$ on different networks, when the basic reproduction number is small than one.

Figure 4 simulates the trend of I_k $(k = 10, 40, 80)$ changing with time on different networks in the case of $a = 1$, $\beta = 0.05$ and the basic reproduction number is great than one ($R_0 = 1.0366$ in the unweighted networks; $R_1 = 2.3643$ in the fixed and adaptive weighted networks). When $t \to +\infty$, I_k all tend to a nonzero positive constant, then the binge drinking will form the endemic disease. But the drinking behavior is the most easily to break out in the fixed weighted networks, the result is accordant with figure 3.



(a) unweight (b) fixed weight (c) adaptive weight

Figure 4. The trend of I_k $(k = 10, 40, 80)$ on different networks, when the basic reproduction number is great than one.

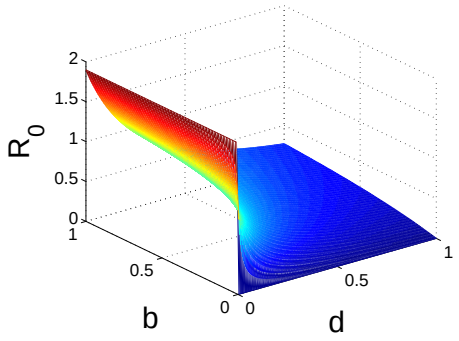
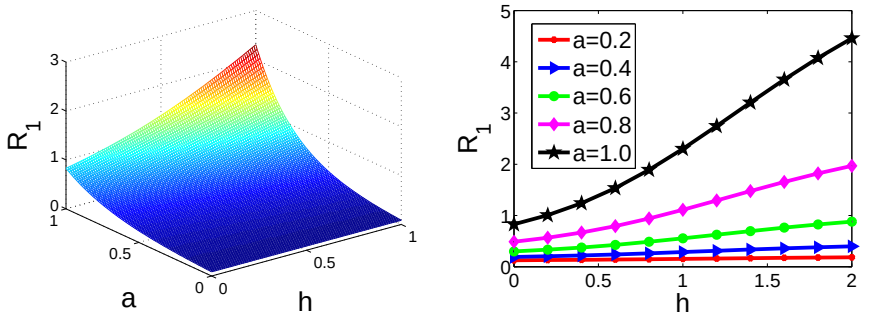


Figure 5. the relationship among R_0, birth rate b and death rate d.

Next, we give the sensitivity analysis of R_0 and R_1 with models' parameters. Figure 5 shows the relationship among R_0, birth rate b and death rate d. By Figure 5, we know that R_0 increases with the increasing of the value of b, and increases with the decreasing of the value of d.

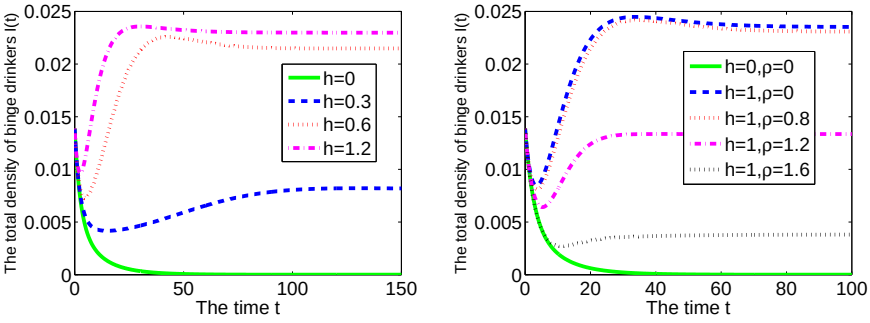
The relationship among R_1, infectivity exponent a and weighted exponent h is shown in figure 6. The result shows that R_1 increases with the increasing of the value of a and h, but it is more sensitivity on a. It implies that the infectivity exponent has a greater effect on the spread of drinking than that of the weighted exponent.



(a) (b)

Figure 6. The relationship among R_1, infectivity exponent a and weighted exponent h.

We show the influence of the fixed and adaptive weight on the total density of heavy drinkers $I(t)$ in figures (7(a)) and (7(b)), where $\beta = 0.04$, $a = 1$. The results display that $I(t)$ is greater when the fixed weighted exponent is greater, and the phenomenon of alcoholism is more serious. On the contrary, the adaptive weight exponent is greater, the rate of be infected will be smaller. It means that the self-protection consciousness plays a good role in reducing the drinking behavior.



(a) (b)

Figure 7. The influence of the fixed and adaptive weight on the total density of heavy drinkers $I(t)$, respectively.

5. Conclusions and discussions

In this paper, we investigate binge drinking models incorporating the population factor on different weighted networks. Firstly, we analyze the dynamics of the drinking model with the linear infectivity $\varphi(k) = k$ on the unweighted network. The basic reproduction number R_0, the uniqueness and stability of equilibria are obtained. Secondly, we modify the model by introducing the nonlinear infectivity $\varphi(k) = k^a (0 < a < 1)$ and two kinds of weights. Finally, we simulate the theoretical results and discuss the spread of drinking behaviors on different networks. We also study the impact of infectivity exponent.

Our results show that the spread of drinking behaviors on the fixed weighted network is the most easily to break out and the infectivity exponent has a greater effect on the drinking behaviors than that of the weight exponent.

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