Sensitivity Analysis of Drinking Dynamics: From Deterministic to Stochastic Formulations

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Abstract

The similarities between drinking behavior and contagion are used to explore —under two types of social structure in the population: homogenous and heterogeneous mixing— the role of nonlinear social interactions on the dynamics of alcohol consumption; quantifying how some model parameters influence the latter dynamics. A deterministic model, assuming homogeneous mixing, is used to derive relative sensitivity functions which explain how the recovery and relapse rates affect the establishment of problem drinkers. A continuos-time Markov chain model is derived from the deterministic model; stochastic simulations are used to quantify how histograms of the number of problem drinkers depend on the recovery and relapse rates, as these rates are gradually incremented. The impact of lowering the relapse rate, as a result of successful treatment, at various intervention times, is assessed by stochastic simulations of drinking dynamics among communities with small-world structure; reductions in the average number of problem drinkers are obtained —with some community structures showing more vulnerability to higher levels of prevalence than others. We conclude from sensitivity analyses (of deterministic and stochastic models) that, either: increasing the recovery rate; or lowering the relapse rate, are measures with positive effects —they tend to reduce the number of problem drinkers.

Keywords: drinking dynamics; deterministic model; stochastic model; small-world network; sensitivity analysis.

1 Introduction

The National Institute of Alcohol Abuse and Alcoholism estimates 18 million Americans suffer from alcohol abuse and alcohol dependence; the economic toll taken by problems related to excessive alcohol consumption, in the U.S. (United States) society, adds up to $185 billion annually [1]. Excessive alcohol use is the third leading cause of death in the U.S.; causing nearly 80,000 fatalities every year, during 2001-05 [2]. This latter number suggests that current prevention and control efforts, including various forms of treatment and education programs targeting children [3] and adolescents [4], are yet to be improved.

Excessive alcohol use can cause anxiety, tension and intoxication, which consequently may reflect in the malfunction of several organs, including the stomach, liver, and heart [5]. On the other hand, alcohol addiction often obeys physical as well as psychological factors with four salient features [6]:

(i) Strong need or desire to drink.

(ii) Lack of control once drinking starts.

(iii) Withdrawal symptoms such as anxiety, nausea, and perspiration when alcohol intake is interrupted.

(iv) Increasing alcohol tolerance.

Social factors —such as peer pressure [7], availability of alcohol [8], and life style— can amount to the drinking behavior that eventually develops into addiction. The mechanisms

associated with the dynamics of drinking at the population level are complex and seem to be highly influenced by drinking environments —such as nightclubs, fraternity parties, local bars, and sports events— as well as by the time spent in each type of drinking environment [9]. Indeed, cultural norms play a fundamental role on the dynamics of drinking [9]; while, in the context of infectious diseases or contagion, various factors such as changing social landscapes (heterogeneous mixing), for sexually transmitted diseases [10], and increased travel patterns, for emerging and re-emerging diseases [11, 12], affect transmission dynamics.

Theoretical epidemiologists [13, 14] define a contact as either: (i) the actual event of a transmission opportunity, or (ii) the pairing of two individuals during which multiple transmission opportunities may occur. Moreover, epidemiologists note that the most important aspects of contacts for infection transmission are [14]: the number of contacts per unit of time; and the number of different individuals with whom these contacts occur.

Motivated by the similarities between the time evolution of drinking behavior and contagion we model the former as a contact process: defining contacts as interactions that promote drinking behavior to be passed on from person to person [15]. More precisely, problem drinking is modeled as a state that results from intense as well as frequent interactions between individuals in three drinking states: moderate drinkers, problem drinkers, and temporarily recovered [15]. Even though, alcohol use is not a communicable disease, throughout this study we consider it as a social contact process and refer to it as "drinking contagion" as well as "contagion among drinking communities". Epidemiological contact

models have proved successful when applied to study patterns of social influence, including the spread of scientific ideas and innovations [16], as well as problem behaviors fueled by social influence, such as: eating disorders [17], alcohol use [15], violence [18], crime [19], and use of illicit drugs [20, 21].

We consider two types of community structure: well-mixed and small-world. In contrast to [22], a recent contribution pertaining small-world network theory and alcohol epidemiology, we build compartmental models ranging from deterministic homogeneous mixing to stochastic homogeneous mixing to stochastic heterogenous mixing; examining the sensitivity of drinking dynamics to the rates of recovery and relapse. Despite the difference in approaches, those proposed here versus those in [22], the results are consistent; simple mathematical formulations with social structure prove useful while informing the effects of potential treatment and prevention programs.

The paper is organized as follows: In Section 2, sensitivity analysis of a deterministic model is carried out; in Section 3 the role of stochastic effects is incorporated; and in Section 4 the role of stochastic effects and community structure (small-world networks) on drinking dynamics are studied. The relevance of the results of these analyses is discussed in the last section.

2 A Deterministic Model for Well-mixed Drinking Communities

A classical SIR (Susceptible-Infected-Recovered) epidemiological framework is used to describe the dynamics of drinking in as simple settings as possible [23, 15]. We revisit the formulation proposed in [15], where the population under consideration is, at any time t, divided into three drinking classes: moderate and occasional drinkers [24, 1], $S(t)$; problem drinkers (also referred to as heavy drinkers [25, 1]), $D(t)$; and temporarily recovered individuals (those who are at risk of relapse), $R(t)$. (The definitions of state variables and parameters are given in Table 1.) It is assumed the drinking community has individuals who interact (or contact each other) at random. In other words, the likelihood that an individual will have contacts with members of each class is given by the size of the class divided by the total population size. For example, the chance of contacting a problem drinker is $D(t)/N$. It is also assumed that all drinking environments are identical (other heterogeneous environments are considered in [9]). The drinking dynamics within a homogeneously mixing (also referred to as well-mixed) population are given by the following set of nonlinear differential equations:

$$\frac{dS}{dt} = \mu N - \beta S(t)\frac{D(t)}{N} - \mu S(t), \tag{1}$$

$$\frac{dD}{dt} = \beta S(t)\frac{D(t)}{N} + \rho R(t)\frac{D(t)}{N} - (\mu + \phi)D(t), \tag{2}$$

$$\frac{dR}{dt} = \phi D(t) - \rho R(t)\frac{D(t)}{N} - \mu R(t), \tag{3}$$

$$N \quad = \quad S(t) + D(t) + R(t), \tag{4}$$

where $1/\mu$ is the average residence time of an individual in this drinking community; β denotes the effective transmission rate (progression from moderate to problem drinker); ϕ is the per-capita recovery rate, implying that $1/(\mu + \phi)$ is the mean length of time spent by an individual in class D; while ρ denotes the community driven relapse rate.

The question of whether or not problem drinkers become established in this community pertains to the number of conversions from S to D when $S \approx N$ (total population), a concept known as reproductive number [23, 14], defined —in a population with recovery— by

$$\mathcal{R}_\phi \equiv \mathcal{R}(\phi) = \frac{\beta}{\mu + \phi}, \tag{5}$$

which when evaluated at $\phi = 0$ gives the so-called basic reproductive number, $\mathcal{R}_0 \equiv \mathcal{R}(0) = \beta/\mu$ [15]. Typically a disease (problem drinker behavior) becomes established — establishment refers to local stability of a nontrivial equilibrium, for additional details see [15]— in the population whenever the reproductive number is greater than one, $\mathcal{R}_\phi > 1$; and under this scenario we explore the role played by recovery —assuming it is highly influenced by treatment— and relapse in the number of problem drinkers. It is of particular interest to address how $D(t)$ changes in response to changes in ϕ and ρ; which, in the case of the deterministic model defined by equations (1)–(3), can be quantified by calculating

$$\frac{\partial D(t)}{\partial \phi} \quad \text{and} \quad \frac{\partial D(t)}{\partial \rho}. \tag{6}$$

Sensitivity analysis theory [26, 27] suggests a way of calculating numerical solutions to

Table 1: State variables and parameters of the contagion model in well-mixed drinking communities.

State variable	Description
$S(t)$	Number of occasional and moderate drinkers at time t
$D(t)$	Number of problem drinkers at time t
$R(t)$	Number of temporarily recovered individuals at time t
Parameter	**Description**
β	Effective transmission rate (average number of interactions per occasional and problem drinker per unit of time)
ρ	Community-driven relapse rate (average number of effective interactions per problem drinker and recovered individual per unit of time)
ϕ	Per-person recovery rate
μ	Per-person departure rate from the drinking environment
N	Total population size

the partial derivatives in equation (6). Before we outline this computation we introduce some notation. The vector

$$\theta = (N, \mu, \beta, \rho, \phi),$$

denotes the model parameters. On the other hand, $u(t;\theta) = (S(t;\theta), D(t;\theta), R(t;\theta))^T$ denotes the state variable vector, at time t for a given θ. We also assume $g = (g_1, g_2, g_3)$ is the vector function whose entries are given by the expressions on the right sides of equations (1)–(3), and write

$$\frac{du}{dt} = g(u(t;\theta);\theta). \tag{7}$$

Since the function g is differentiable, taking the partial derivatives $\partial/\partial\theta$ of both sides of equation (7) we obtain the differential equation

$$\frac{d}{dt}\frac{\partial u}{\partial \theta} = \frac{\partial g}{\partial u}\frac{\partial u}{\partial \theta} + \frac{\partial g}{\partial \theta}. \tag{8}$$

In equation (8) the 3×3 matrix $\partial g/\partial u$ is equal to

$$\begin{bmatrix} -\left(\frac{\beta}{N} + \mu\right) & -\frac{\beta}{N}S & 0 \\ \\ \frac{\beta}{N}D & \frac{\beta}{N}S + \frac{\rho}{N}R - (\mu + \phi) & \frac{\rho}{N}D \\ \\ 0 & \phi - \frac{\rho}{N}R & -\left(\frac{\rho}{N}D + \mu\right) \end{bmatrix},$$

while $\partial u/\partial\theta$ is a 3×5 matrix given by

$$\frac{\partial u}{\partial \theta} = \begin{bmatrix} \frac{\partial S}{\partial N} & \frac{\partial S}{\partial \mu} & \frac{\partial S}{\partial \beta} & \frac{\partial S}{\partial \rho} & \frac{\partial S}{\partial \phi} \\ \frac{\partial D}{\partial N} & \frac{\partial D}{\partial \mu} & \frac{\partial D}{\partial \beta} & \frac{\partial D}{\partial \rho} & \frac{\partial D}{\partial \phi} \\ \frac{\partial R}{\partial N} & \frac{\partial R}{\partial \mu} & \frac{\partial R}{\partial \beta} & \frac{\partial R}{\partial \rho} & \frac{\partial R}{\partial \phi} \end{bmatrix}.$$

Numerical values of $\partial u/\partial\theta$ are calculated by solving (7) and (8) simultaneously; we define $x(t) = \frac{\partial u}{\partial \theta}(t;\theta)$, let the parameters be evaluated at some known values, $\theta = \hat{\theta}$, and solve the following differential equations from $t = t_0$ to $t = t_n$,

$$\frac{du}{dt} = g(u(t;\theta);\theta) \tag{9}$$

$$\frac{d}{dt}x(t) = \frac{\partial g}{\partial u}x(t) + \frac{\partial g}{\partial \theta} \tag{10}$$

$$x(0) = 0. \tag{11}$$

Relative sensitivity functions, $s_\phi(t)$ and $s_\rho(t)$, defined as

$$s_\phi(t) = \frac{\phi}{D(t;\hat{\theta})}\frac{\partial D}{\partial \phi}(t;\hat{\theta}) \tag{12}$$

$$s_\rho(t) = \frac{\rho}{D(t;\hat{\theta})}\frac{\partial D}{\partial \rho}(t;\hat{\theta}), \tag{13}$$

are dimensionless variables, that can be used to compare, within the same scale, the degree of sensitivity of $D(t)$ with respect to the recovery rate ϕ and the relapse rate ρ.

Figure 1 displays numerical solutions to equations (9)–(10), obtained while using known parameter values suggested in [15] (the units are written in square brackets); $N = 2500$; $\beta = 0.65$ [years^{-1}]; $\rho = 0.10$ [years^{-1}]; $\phi = 0.10$ [years^{-1}]; and $\mu = 0.10$ [years^{-1}]. In Figure

1(a), the number of, at time t: occasional and moderate drinkers, $S(t)$, problem drinkers, $D(t)$, and temporarily recovered individuals, $R(t)$, are shown in squares, crosses, and triangles, respectively. Figure 1(b) displays the relative sensitivity functions; $s_\phi(t)$(stars) and $s_\rho(t)$ (circles), versus time t. Neither of these functions changes sign, during the time window under consideration, suggesting monotonic behavior in $D(t)$ originates from fluctuations in the parameters. On one hand, it is seen the function $s_\rho(t)$ is positive ($s_\rho(t) > 0$ for $0 < t < 25$), implying that the number of problem drinkers, $D(t)$, is increasing relative to increments in the relapse rate ρ. On the other hand, $s_\phi(t) < 0$ for $0 < t < 25$, suggests that variations in the recovery rate ϕ, cause the number of problem drinkers to decline. This type qualitative behavior (Figure 1(b)) is reassuring, since one would expect the establishment of problem drinkers to benefit from increments in the community-based relapse rate, while soaring recovery rates (reflecting in part the effect of treatment) should be detrimental for such establishment.

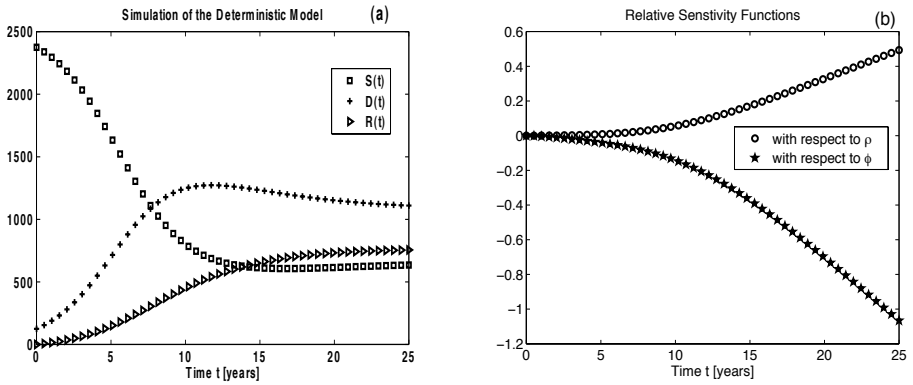

Figure 1: Numerical solutions of the deterministic model for drinking dynamics. Panel (a) shows $S(t)$ (squares), $D(t)$ (crosses), and $R(t)$ (triangles), as functions of time t, respectively. Panel (b) displays the relative sensitivity functions, $\frac{\rho}{D(t)}\frac{\partial D(t)}{\partial \rho}$ (circles) and $\frac{\phi}{D(t)}\frac{\partial D(t)}{\partial \phi}$ (stars), versus time t. The baseline values used to obtain these numerical solutions are: $N = 2500$; $\beta = 0.65$; $\rho = 0.10$; $\phi = 0.10$; and $\mu = 0.10$.

3 A Stochastic Model in Well-mixed Drinking Communities

In this Section we derive a stochastic model from the deterministic formulation described by equations (1)–(4). The goal is to quantify the variability on drinking dynamics due to stochastic effects; intending to highlight some of the differences and similarities between the deterministic and stochastic approaches.

The derivation of the stochastic model is standard —see for instance[28, 29]— and our analysis here is primarily based on simulations. Transitions between drinking classes involve discrete events which change the number of individuals in every class, one at a time. For example, when a drinking contagion event occurs, the number of moderate drinkers is decreased by one, while the number of problem drinkers increases by one. The probability an event takes place during an infinitesimal time interval $[t, t + dt]$ is calculated from the average rates in the deterministic model (the probability equals the rate multiplied by the length of the time interval). In the example mentioned above, the event occurs at a rate equal to $\beta S(t)D(t)/N$, then the probability it happens during $[t, t+dt]$ is $(\beta S(t)D(t)/N)\, dt$. All the events, their rates of occurrence, and the probabilities at which they take place are listed in Table 2.

It is assumed the events (see first column in Table 2) are described by independent Poisson processes [28]. The term

$$E = \mu N + \mu S + \mu D + \mu R + \beta SD/N + \phi D + \rho RD/N, \tag{14}$$

denotes the rate at which all events occur at time t. For simplicity in notation, we omit the

Table 2: Possible events in the drinking model, their rates and probabilities of their occurrence in time interval $[t, t+dt]$. For simplicity, the dependence on t is omitted, writing S, D, and R, instead of $S(t)$, $D(t)$, and $R(t)$, respectively.

Event	Transition	Rate at which event occurs	Probability of transition in $[t, t+dt]$
Recruitment	$S \to S+1$	μN	$\mu N dt$
Moderate drinker removal	$S \to S-1$	μS	$\mu S dt$
Problem drinker removal	$D \to D-1$	μD	$\mu D dt$
Recovered removal	$R \to R-1$	μR	$\mu R dt$
Drinking contagion	$S \to S-1$, $D \to D+1$	$\beta S \frac{D}{N}$	$\beta S \frac{D}{N} dt$
Recovery	$D \to D-1$, $R \to R+1$	ϕD	$\phi D dt$
Relapse	$D \to D+1$, $R \to R-1$	$\rho R \frac{D}{N}$	$\rho R \frac{D}{N} dt$

dependence on t and we write S, D, and R, instead of $S(t)$, $D(t)$, and $R(t)$, respectively. The time between events is exponentially distributed with mean $1/E$; in fact, the time at which the next event happens is found by sampling from an exponential distribution with mean $1/E$.

To decide which event takes place (once it is known an event occurs), we divide up the interval $(0, E)$ into lengths that correspond to the relative occurrence probabilities of the various events. In other words, given that an event has occurred, the probability that it is a recruitment is $\mu N/E$, the probability that it is a moderate drinker removal is $\mu S/E$, the probability that it is a problem drinker removal is $\mu D/E$, etc. A random number U is sampled from the uniform distribution on $(0, 1)$ and an event is selected if this sampled number falls within the subinterval in $(0, E)$ corresponding to such event. For instance, we decide that a recruitment has occurred if $0 < U < \mu N/E$, a moderate drinker removal if U lies between $\mu N/E$ and $(\mu N + \mu S)/E$, a problem drinker removal if U lies between $(\mu N + \mu S)/E$ and $(\mu N + \mu S + \mu D)/E$, and so on.

The average behavior of the stochastic model described in Table 2 is compared to its deterministic analog when stochastic realizations and numerical solutions are computed using the same parameter values ($N = 2500$; $\beta = 0.65$; $\rho = 0.10$; $\phi = 0.10$; and $\mu = 0.10$). In Figure 2 it is observed the dynamics of the deterministic model (black circles) agrees with the mean (over 100 realizations) dynamics of the stochastic model (grey curves), as is expected when $\mathcal{R}_\phi > 1$ (albeit extinction in the stochastic model is theoretically possible; see [28]). A fundamental difference between the deterministic model (equations (1)–(4))

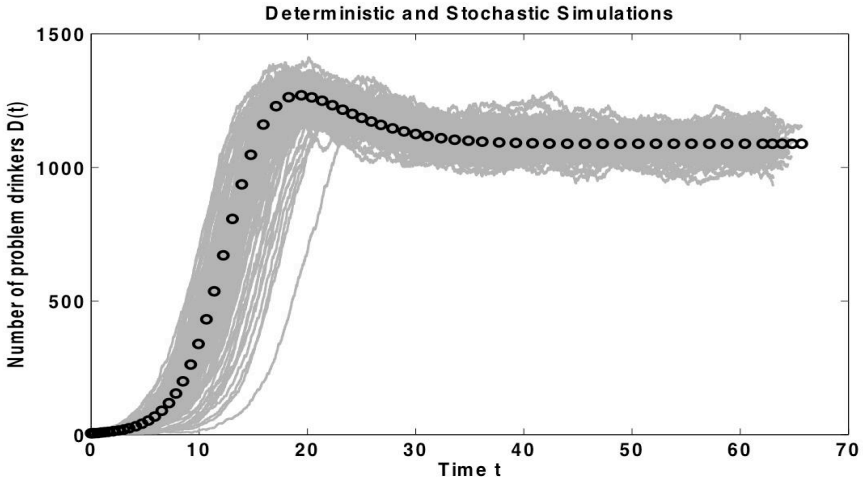

Figure 2: Results from numerical simulations. 100 stochastic realizations (grey curves) and numerical solutions of the deterministic (black circles) problem drinker class $D(t)$ versus time t. For these simulations the following values of parameters were used: $N = 2500$; $\beta = 0.65$; $\rho = 0.10$; $\phi = 0.10$; and $\mu = 0.10$. The initial number of problem drinkers was $D(t_0) = 5$.

and the stochastic model (Table 2) is that for the same set of parameters: the latter model gives rise to several distinct time trajectories, each of them referred to as a realization, while the former model gives an only possible outcome. The variability of the number of problem drinkers, at a particular time point, can be quantified from the realizations of the simulated stochastic model. For instance, letting T denote a stoppage time (if t_j denotes the time at which the jth event takes place, see equation (14), then define $T = t_n$, for some n, as the stoppage time of the simulations —for those displayed in this Section, we used $n = 10000$), then the variability of $D(T)$ can be quantified by calculating its mean and variance from the realizations. To illustrate the variability of $D(T)$ (number of problem drinkers at a stoppage time T) we show a histogram in Figure 3, resulting from 100 stochastic realizations.

It is of interest to investigate how the stochastic drinking dynamics are affected by the recovery and relapse rates. We address it using simple numerical experiments, which quantify the dependence of $D(T)$ on ρ (relapse rate) and ϕ (recovery rate assumed to be fueled by treatment). More precisely, most of the model parameters are held fixed while one parameter is varied, and 25 realizations of the stochastic model are calculated: ϕ is varied from $\phi = 1.0 \times 10^{-8}$ to $\phi = 5.0 \times 10^{-1}$; ρ is varied from $\rho = 1.0 \times 10^{-8}$ to $\rho = 5.0 \times 10^{-1}$; while $N = 2500$; $\beta = 0.65$; $\rho = 0.10$ (while varying ϕ); $\phi = 0.10$ (while varying ρ); and $\mu = 0.10$. Figure 4(a) displays the mean and standard deviation of $D(T)$ versus ϕ; the mean values appear as solid squares, while the mean plus one standard deviation is shown as the upper dashed curve, and the mean minus one standard deviation is displayed as the

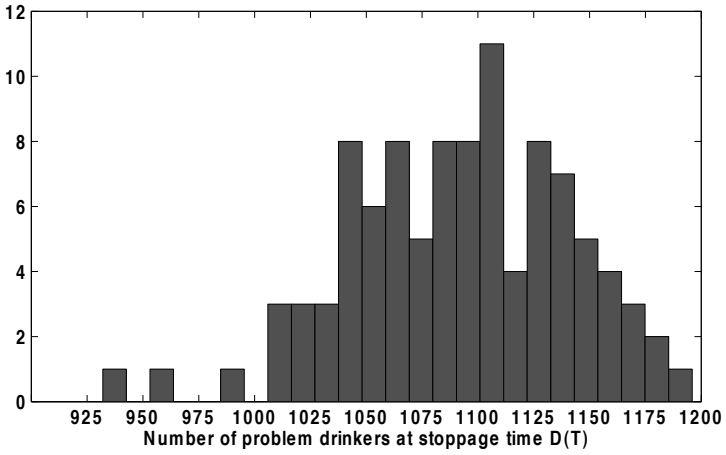

Figure 3: Histogram of $D(T)$, number of problem drinkers at stoppage time T, resulting from 100 stochastic realizations.

lower dashed curve. It is seen that the mean $D(T)$ decreases as a function of the recovery rate ϕ, this is consistent with the information conveyed by the relative sensitivity function $s_\phi(t)$ in Figure 1(b). On the other hand, Figure 4(b) displays the mean $D(T)$, and the mean $D(T)$ plus (upper dashed curve) and minus (lower dashed curve) one standard deviation, as functions of ρ. It is clear the mean $D(T)$ is an increasing function of the relapse rate ρ, which is consistent with the positivity of $s_\rho(t)$ in Figure 1(b).

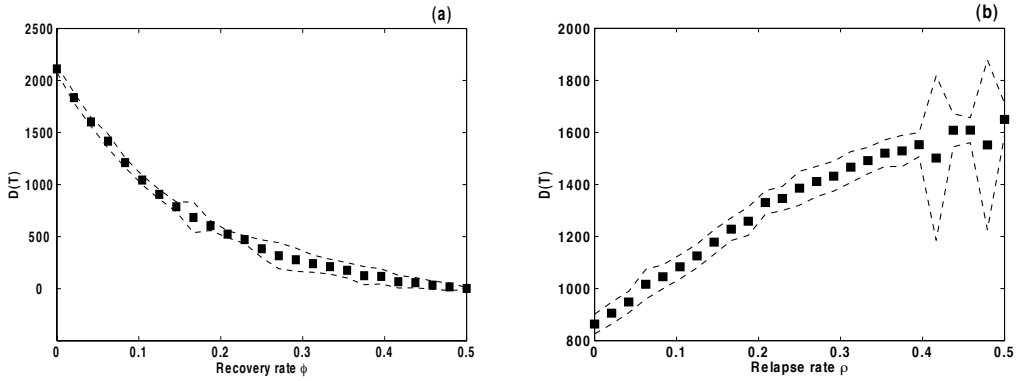

Figure 4: Results from 25 stochastic simulations while varying the recovery rate ϕ and the relapse rate ρ. Panel (a) displays the mean number (solid square) of problem drinkers at a stoppage time T, $D(T)$, along with the mean plus/minus one standard deviation (dashed curves), for each value of ϕ used in the simulations; from $\phi = 1.0 \times 10^{-8}$ to $\phi = 5.0 \times 10^{-1}$. Panel (b) shows the mean $D(T)$, and the mean plus (upper dashed curve) and minus (lower dashed curve) one standard deviation, versus ρ; from $\rho = 1.0 \times 10^{-8}$ to $\rho = 5.0 \times 10^{-1}$. The other parameters were held fixed at the following values: $N = 2500$; $\beta = 0.65$; $\rho = 0.10$ (while varying ϕ); $\phi = 0.10$ (while varying ρ); and $\mu = 0.10$.

4 A Stochastic Model for Structured (Small-world) Drinking Communities

The fact that several processes (including drinking) are highly dependent on the contacts and types of contacts between heterogeneous individuals has motivated the study of epidemics on networks [30, 31, 32]. A network (graph) is a set of nodes and connections between them (edges). Random graphs are natural representations of the space (landscape) of contacts between individuals in a population; by letting each node represent an individual and allowing a random connection between two nodes resemble a potential contact [33].

Watts and Strogatz [34] introduced a parametrization of families of networks that interpolate between two architectures (topologies): from a regular lattice to a random graph. Their proposed parametrization is stated as an algorithm (used to construct networks) which is initialized with a one-dimensional periodic ring lattice of N nodes, each of them connected to the closest $\langle k \rangle$ neighbors (two nodes are neighbors if there is an edge connecting them). The network continues to be updated by re-wiring each edge to a randomly selected node with probability p (referred to as the disorder parameter). When the disorder parameter satisfies $p \to 0$, then the algorithm leaves the lattice intact, while $p \to 1$ implies all edges are rewired and the resulting network is equivalent to a random graph [35]. Watts and Strogratz showed that a few random long-range connections would drastically reduce the average distance between any pair of nodes [34] —a property that enhances transmis-

sion. This property is known as the small-world effect and was discovered by the social psychologist S. Milgram as a result of letter-forwarding experiments [36]. In addition, these networks (constructed with the Watts-Strogatz algorithm) have also a property called clustering, which pertains to how the neighborhoods of connected nodes overlap (cliquishness), in other words, whether or not "friends of friends are friends of each other" [34].

In this study we use the terms network and community interchangeably. Moreover, community structure is modeled by random graphs; with nodes representing individuals and random edges denoting social connections to other individuals which may lead to a conversion into a problem drinker. Nodes can be in one of three distinct states: moderate drinkers, problem drinkers, and temporarily recovered. They transition between states according to probabilities which are functions of time and the number of neighbors in particular states. Suppose there is a community with N nodes; a given node i (where $1 \leq i \leq N$) has $\delta(i,t)$ neighbors who, at time t, are in the state called problem drinker. We define the probability node i changes:

1. from moderate drinker into problem drinker as $1 - \exp(-\beta\delta(i,t))$, where β denotes the transmission rate;

2. from problem drinker into temporarily recovered as $1 - \exp(-\phi)$, where ϕ denotes the recovery rate;

3. from temporarily recovered into problem drinker as $1-\exp(-\rho_\tau(t)\delta(i,t))$, where $\rho_\tau(t)$ denotes a time-dependent relapse rate.

Table 3: State variables, parameters, events, and transition probabilities of the drinking contagion model in small-world communities.

State variable	Description
$\delta(i,t)$	Number of problem drinker neighbors of node i at time t
$S_p(t)$	Total number of moderate drinkers at time t in a community parametrized by p
$D_p(t)$	Total number of problem drinkers at time t in a community parametrized by p
$R_p(t)$	Total number of temporarily recovered individuals at time t in a community parametrized by p
Parameter	**Description**
β	Transmission rate
ϕ	Per-person recovery rate
$\rho_\tau(t)$	Time-dependent relapse rate

Event	Probability of transition
Node i changes from *moderate* into *problem* drinker	$1 - e^{-\beta\delta(i,t)}$
Node i switches from *problem drinker* into *temporarily recovered*	$1 - e^{-\phi}$
Node i changes from *temporarily recovered* into *problem drinker*	$1 - e^{-\rho_\tau(t)\delta(i,t)}$

Table 4: Parameter values utilized in simulations of drinking contagion in small-world communities.

Parameter	Description	Baseline value
$\langle k \rangle$	Average connectivity per node	6
N	Community size	1000
β	Transmission rate	0.12
ϕ	Per-person recovery rate	0.7
$\rho_\tau(t)$	Time-dependent relapse rate	$\rho_\tau(t) = 0.90$ whenever $t < \tau$ $\rho_\tau(t) = 0.12$ if $t \geq \tau$
T	Stoppage time	4000
$D_p(0)$	Initial number of problem drinkers chosen uniformly at random in every community	5
	Number of stochastic realizations	20

This formulation (see Table 3) determines a stochastic process and the terms $S_p(t)$, $D_p(t)$, and $R_p(t)$ are random variables denoting, at time t, the total number of moderate drinkers, problem drinkers, and temporarily recovered, respectively, in a community parametrized by the disorder parameter p.

We simulate drinking dynamics among small-world communities, by calculating stochastic realizations of the random networks [34] and the stochastic process defined in Table 3, while using the parameter baseline values summarized in Table 4. We relax the assumption about time between events being exponentially distributed; instead we let time between events have length one, with arbitrary time units.

To contrast the role played by a time-dependent relapse rate, we consider both $\rho_\tau(t) \equiv 0$ and $\rho_\tau(t) \neq 0$. In the case $\rho_\tau(t) \equiv 0$ —that is, assuming nobody relapses after drinking stops— the model reduces to a network SIR epidemic model [31] with a well known qualitative behavior; the number of problem drinkers grows from a small number until it peaks at a maximum to later fade out, since eventually everyone recovers. This feature derives from the deterministic [23] and stochastic [28] single-outbreak SIR model, only being enhanced by the community structure.

Histograms of $D_p(T)$ and $R_p(T)$, where T denotes a stoppage time in the simulations (see Table 4), are computed for each value of p; for instance, Figure 5 displays the histograms obtained over 20 realizations with $p = 3.02 \times 10^{-4}$. To assess the role community structure plays in both of these quantities we examine, in Figure 6, how the average and variance (over 20 realizations) of $D_p(T)$ and $R_p(T)$ vary with respect to p [37, 38]. Figure

6(a) shows that $D_p(T) = 0$, for all p, implying that every problem drinker node eventually becomes temporarily recovered across all community structures. However, the average size, at a stoppage time T, of the temporarily recovered class $R_p(T)$, displayed in Figure 6(b), varies substantially for various community structures; random networks with $1.00 \times 10^{-4} < p < 1.00 \times 10^{-2}$ have mean $R_p(T)$ values under 200 (less than 20%), while communities with $1.00 \times 10^{-1} < p < 1.00$ display average $R_p(T)$ values between 600 and 800 (from 60% to 80%). The results of Figure 6(b) suggest that in the absence of demographics and relapse, the community structure does affect the total number of temporarily recovered individuals; small average distance between nodes (in the community) promotes drinking contagion.

The next case considered here uses a nontrivial time-dependent relapse rate, $\rho_\tau(t) \neq 0$, defined as

$$\rho_\tau(t) = \begin{cases} 0.90 & \text{if } t < \tau \\ 0.12 & \text{if } t \geq \tau, \end{cases} \tag{15}$$

where τ denotes a time at which the relapse rate drops as a result of treatment. The values of the relapse rate were chosen to have the average relapse probability change, at $t = \tau$, from

$$1 - e^{-\rho_\tau(t)\langle k \rangle} \approx 1 \tag{16}$$

to

$$1 - e^{-\rho_\tau(t)\langle k \rangle} \approx 0.50, \tag{17}$$

where $\langle k \rangle$ denotes the average number of connections per node in a one-dimensional lattice,

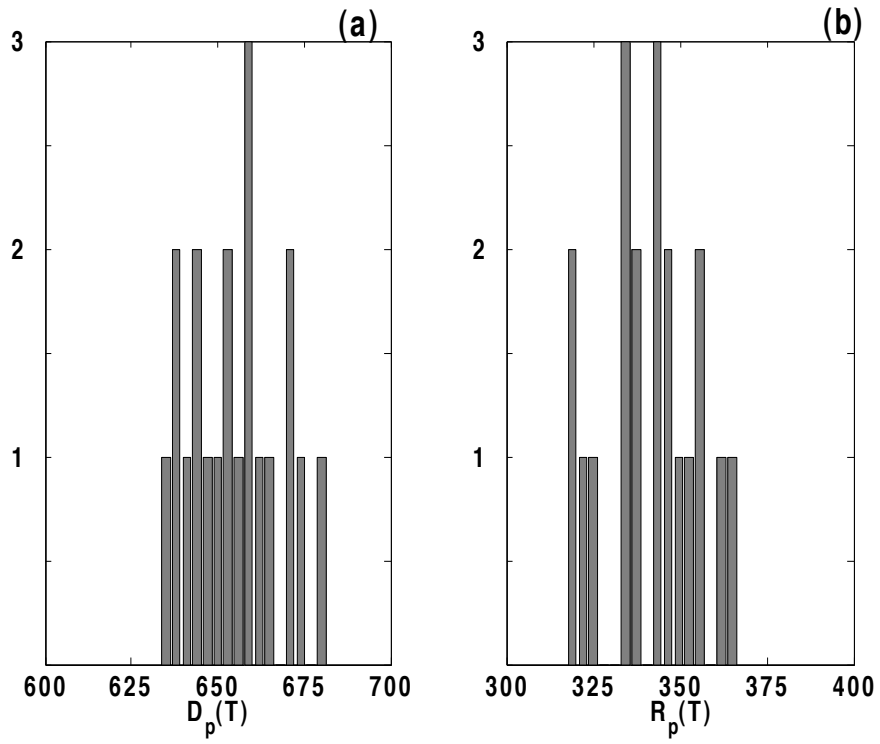

Figure 5: Histograms of the total number of problem drinkers and temporarily recovered individuals, $D_p(T)$ and $R_p(T)$, respectively, at a stoppage time T. Samples obtained from 20 stochastic realizations in simulated communities with $p = 3.02 \times 10^{-4}$ (the parameter baseline values used in these simulations are displayed in Table 4).

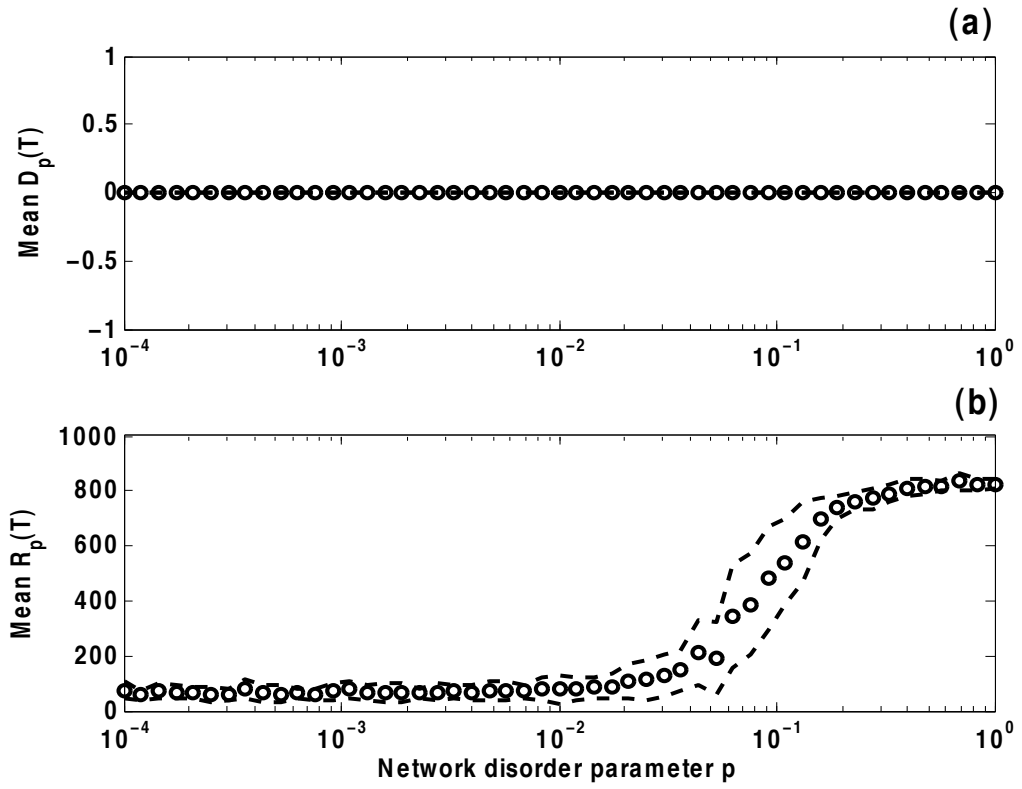

Figure 6: Average and variance of $D_p(T)$ and $R_p(T)$ as functions of the simulated community architecture parametrized by p (logarithmic scale). The mean (circles) and mean plus and minus one standard deviation (dash curves) are computed from 20 stochastic realizations for each fixed value of p. Panels (a) and (b) display results of simulated contagion in small-world communities in the absence of relapse, $\rho_\tau(t) \equiv 0$.

$p = 0$ (see Table 4; for the simulations we set $\langle k \rangle = 6$). The average relapse probability of equation (16) illustrates a worst-case scenario in which temporarily recovered nodes become problem drinkers with probability nearly one. Figure 7 displays results for a worst-case scenario using $\tau = \infty$ (i.e. the relapse rate remains constant). Under this scenario we find community structure does not affect the average size of either the problem drinker or the temporarily recovered class; all communities seem to be equally at risk of supporting endemic levels of problem drinkers (these levels are above 60%, 600 out 1000, of the community size). It is seen in Figure 7 that on average $D_p(T) + R_p(T) = 1000$; implying that every node in the simulated communities has been converted into a problem drinker at least once, leaving the total number of moderate drinkers nearly empty.

The sensitivity of the prevalence levels of problem drinkers, at time T, with respect to the recovery and relapse rates is investigated by simulating drinking dynamics, while increasing the values of ϕ and ρ, respectively. More precisely: the recovery rate is increased from $\phi = 1.0 \times 10^{-4}$ to $\phi = 3.0 \times 10^{-1}$; the relapse rate is increased from $\rho = 1.0 \times 10^{-1}$ to $\rho = 2.5$; while all the other parameters are held fixed at values displayed in Table 4. It can be observed in Figure 8(a) that when displayed as a function of the recovery rate ϕ, the mean $D_p(T)$ decreases to nearly one-half, for $p = 0$ and $p = 1$, respectively. Hence, increasing the recovery rate when the average relapse probability is extremely high ($1 - e^{-\rho_\tau(t)\langle k \rangle} \approx 1$) promotes reductions in the number of problem drinkers (and therefore gains in the number of temporarily recovered individuals, not shown here); very encouraging results from the point of view of treatment and intervention. In Figure 8(b) we see that

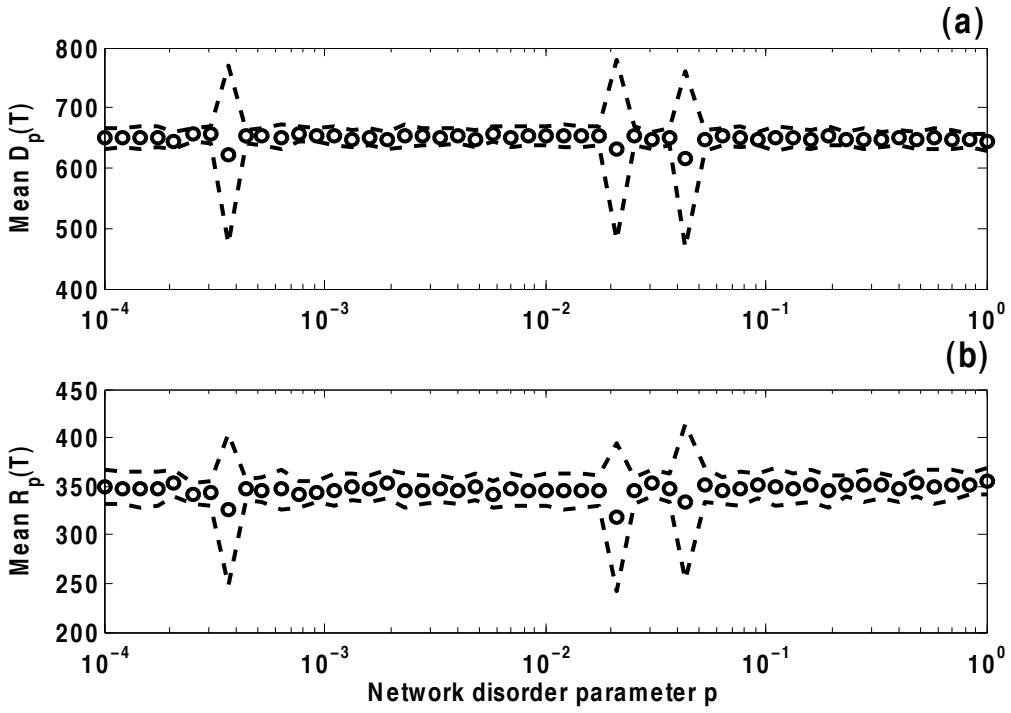

Figure 7: Dependence of the average and variance of $D_p(T)$ and $R_p(T)$ on the community structure p (logarithmic scale). Average (circles) and one standard deviation added to and subtracted from the average (dash curves) are calculated from 20 stochastic realizations for each fixed value of p. The results shown in Panels (a) and (b) assess a "worst case scenario" of having on average every temporarily recovered node going into relapse with probability nearly one, in symbols $1 - e^{-\rho_\tau(t)\langle k\rangle} \approx 1$.

as ρ increases the average $D_p(T)$ increases as well. Network structure does not affect the sensitivity of ϕ, however, it does affect the sensitivity of ρ: Figure 8(b) suggests that small average distance between nodes, in the drinking community, results in larger average numbers of problem drinkers —compare the mean $D_p(T)$ at $p = 1$ (circles) and $p = 0$ (squares) for $0.10 < \rho < 0.25$.

The sensitivity of the mean $D_p(T)$ with respect to the relapse rate, Figure 8(b), suggests that while using a time-dependent relapse rate $\rho_\tau(t)$, with $\tau < \infty$, one would expect significant outcomes. To explore numerically the role of applying successful treatment programs in drinking communities, which prevent temporarily recovered individuals from relapse, at distinct times τ, we carry out simulations for $\tau = 3,\ 5,\ 7,\ 10$. Figure 9 displays the effect of how long it takes to launch treatment programs that reduce relapse; it suggests that for $\tau = 3,\ 5,\ 7,\ 10$, there is not much differences in the average $D_p(T)$ and $R_p(T)$. However, these averages give evidence of improvement when compared to those obtained with $\tau = \infty$. Therefore, the treatment programs promoting a lower relapse are beneficial, even when it seems too late.

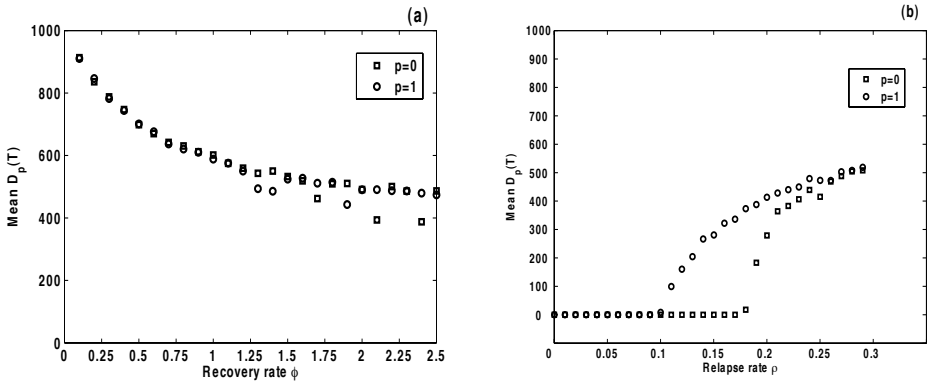

Figure 8: Dependence of the average $D_p(T)$ on the recovery rate, ϕ, and the relapse rate, ρ. The averages of 10 stochastic realizations are displayed for simulated communities obtained at $p = 0$ (squares) and $p = 1$ (circles). Panels (a) and (b) provide basic measures on the sensitivity of the mean $D_p(T)$ to variations in ϕ and ρ, respectively. It is clear from Panel (a) that increments in the recovery rate ϕ lead to a decay in the mean $D_p(T)$; while Panel (b) suggests that a decay in the mean $D_p(T)$ is expected from decreasing the relapse rate ρ. It is seen in Panel (b) that simulated communities obtained at $p = 1$ (circles) seem to accumulate larger numbers of problem drinker nodes relative to those of communities with $p = 0$, for $0.10 < \rho < 0.25$. On the other hand, Panel (a) shows that both types of community structure ($p = 0$ and $p = 1$) have the same decreasing behavior in $D_p(T)$ as ϕ is increased.

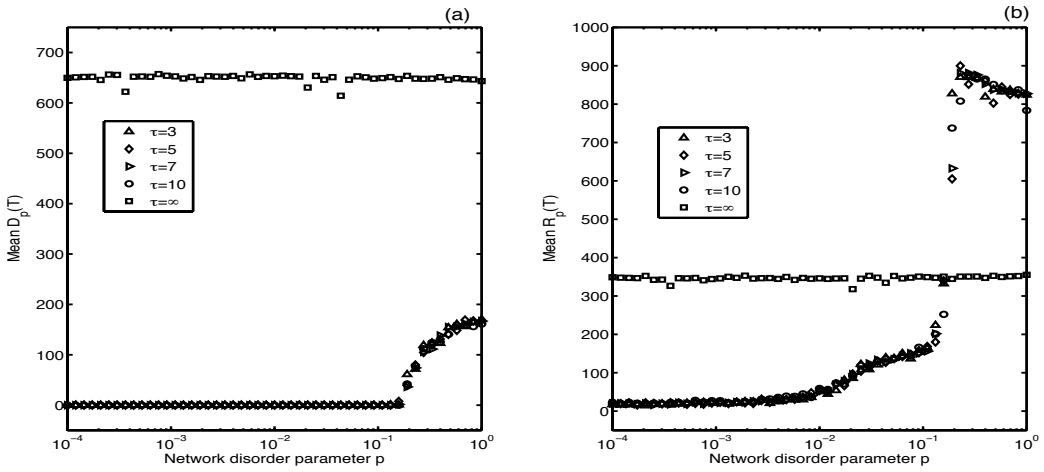

Figure 9: Average $D_p(T)$ and $R_p(T)$ as functions of the community structure parameterized by p. Panels (a) and (b) display the results obtained from using a time-dependent relapse rate $\rho_\tau(t)$. The relapse rate is decreased from 0.90 to 0.12 at time $t = \tau$. In this way on average every node diminishes its probability to change from temporarily recovered into problem drinker by half. In symbols, the probability $1 - e^{-0.90\langle k\rangle} \approx 1$ decreases to $1 - e^{-0.12\langle k\rangle} \approx 0.5$. Panels (a) and (b) show the averages sampled by applying this relapse reduction at distinct times: $\tau = 3$ (upward triangles); $\tau = 5$ (diamonds); $\tau = 7$ (right triangles); $\tau = 10$ (circles). The averages displayed in Figure 7 are shown here as $\tau = \infty$ (squares).

5 Discussion

Under the assumption of homogeneous mixing we model drinking dynamics as a contact process [15, 39]; using a simple deterministic formulation based on nonlinear differential equations. The sensitivity equations of the deterministic model are derived and numerical solutions, obtained using known parameter values, are suggestive of the roles played by the relapse and recovery rates in the prevalence of problem drinkers. The numerical solutions displayed in Figure 1(b) suggest the establishment of problem drinkers would benefit from increments in the relapse rate, while increasing recovery rates should be detrimental for such establishment.

The deterministic model, introduced in Section 2, is extended into a stochastic model; by allowing the contact process to evolve according to stochastic rules. This stochastic formulation permits to quantify the variability in the size of the problem drinker class and using numerical studies we confirm that: (i) the deterministic model matches the average behavior of the stochastic model; (ii) the mean $D(T)$ decreases as a function of the recovery rate ϕ; and is an increasing function of the relapse rate ρ.

In the last part of this study, we explore the sensitivity of drinking dynamics to the recovery and relapse rates, while modeling heterogenous mixing with random networks — unlike in [22] where the effect of community structure is explored using a simpler drinking contagion model with only two states as well as a different network model called Rewired Connected Caveman Network. Random networks are constructed with the Watts-Strogatz

algorithm [34] and are parameterized by a disorder parameter p. For $p = 0$, we have a situation where individuals only interact locally, that is, only with the nearest neighbors, while when $p = 1$ they basically interact with everybody in the community. According to the simulations under a worst-case scenario —having a high average relapse probability, $1 - e^{-\rho_\tau(t)\langle k\rangle} \approx 1$, with $\tau = \infty$— all community structures are equally exposed to develop nearly the same average endemic levels of problem drinkers. On the other hand, improvements (reductions in mean $D_p(T)$) are seen when simulating treatment programs that promote lowering the relapse rate at various times ($\tau = 3, 5, 7, 10$); network structure plays a role in the latter scenario, the mean $D_p(T)$ and $R_p(T)$ attain higher levels as $p \to 1$ (see Figure 9).

In summary, sensitivity analyses —Figure 1 (deterministic model for well-mixed communities), Figure 4 (stochastic model for well-mixed communities), and Figure 8 (stochastic model for small-world communities)— consistently suggest that increments in the recovery rate, as well as reductions in the relapse rate, have a positive effect; they tend to promote reductions in the number of problem drinkers. We conclude it is in the best interest of treatment programs to concentrate efforts preventing temporarily recovered individuals from relapse into drinking.

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