

INFLUENCE OF ENVIRONMENTAL FACTORS ON COLLEGE ALCOHOL DRINKING PATTERNS

RIDOUAN BANI, RASHEED HAMEED AND STEVE SZYMANOWSKI

Department of Mathematics, Northeastern Illinois University
5500 N. St. Louis Ave, Chicago, IL 60625-4699, USA

PRISCILLA GREENWOOD

Department of Mathematics, University of British Columbia
1209 Math Annex, Vancouver, BC V6T 1Z2, Canada

CHRISTOPHER KRIBS-ZALETA

Department of Mathematics, The University of Texas at Arlington
Box 19408, Arlington, TX 76019-0408, USA

ANUJ MUBAYI¹

Department of Mathematics, Northeastern Illinois University
5500 N. St. Louis Ave, BBH 214A, Chicago, IL 60625-4699, USA

Mathematical, Computational & Modeling Science Center
Arizona State University, Tempe

Prevention Research Center, Berkeley

ABSTRACT. Alcohol abuse is a major problem, especially among students on and around college campuses. We use the mathematical framework of [16] and study the role of environmental factors on the long term dynamics of an alcohol drinking population. Sensitivity and uncertainty analyses are carried out on the relevant functions (for example, on the drinking reproduction number and the extinction time of moderate and heavy drinking because of interventions) to understand the impact of environmental interventions on the distributions of drinkers. The reproduction number helps determine whether or not the high-risk alcohol drinking behavior will spread and become persistent in the population, whereas extinction time of high-risk drinking measures the effectiveness of control programs. We found that the reproduction number is most sensitive to social interactions, while the time to extinction of high-risk drinkers is significantly sensitive to the intervention programs that reduce initiation, and the college drop-out rate. The results also suggest that in a population, higher rates of intervention programs in low-risk environments (more than intervention rates in high-risk environments) are needed to reduce heavy drinking in the population.

1. Introduction. Alcohol abuse has been on the rise, especially among college students. Alcohol-related injuries and deaths per 100,000 college students increased by 6% from 1998 to 2001 [7]. The proportion of students from ages 18-24 who were reported driving under the influence of alcohol also increased from 26.5% to

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¹**Corresponding Author**

31.4% during the same period [7]. In 1998, a special task force was created by the National Advisory Council of the National Institute on Alcohol Abuse and Alcoholism (NIAAA) especially for the research and review of college drinking [8]. Customs that promote college drinking are embedded in various levels of students' environments, for example, advertisements of alcohol industry sponsors during college sports events and promotion of alcohol by establishments near campus that sell alcohol. College students come into contact with college drinking whether they drink or not. Social interactions can be defined as the acts, actions, or practices of two or more people mutually oriented toward each other. Social behavior affects and takes into account others' subjective experiences [22].

Both diseases and habits in human populations follow dynamics determined by social interactions [12, 13, 14]. It has been observed that social contexts and activities are the components and proxy for influences in drinking—both at the individual and social levels. Environmental and peer influences combine to create a culture of drinking. There have been studies that have evaluated alcohol drinking environmental programs [25] although their impact and effect were captured over short time scales, using specific (and small) populations, or without considering the impact of social interactions. Still, these studies suggest that the use of multiple, environmental strategies are promising for reducing high risk alcohol use and alcohol-related problems among college students. However, their implementation depends on the risk-levels of the various college environments.

There have been only a handful of modeling studies in the literature that capture the impact of social interactions on the dynamics of alcohol drinking. [6] conducted a study using inner-city adolescents; the study examined the role of family drinking norms to predict adolescent drinking decisions. Their results suggest the incorporation of the social influence into the alcohol prevention programs. [21] used a meta-analytic review to compare thirteen studies that used different variables associated with the modeling of alcohol consumption. Analyses identified variables that moderate the effect of modeling on alcohol consumption, including the drinking history of the participant, the drinking task used and the nature of the interaction between model and participants. A simple small-world network based model capturing alcohol-dependence in the population is studied in [3]. This study uses a combination of network and epidemiological theory to link individuals in a population. All the individuals in this network included a characteristic defined as resistivity to alcohol drinking behavior as well as their linking structure. The individual's drinking behavior is therefore affected by resistivity and social interactions causing them to be alcohol dependence or free from it. A treatment is defined that reduces individual's intensity for alcohol dependence. This study concludes that the application of the treatment after a critical level can be useful in reducing alcohol dependence in a population, and this can be improved by increasing connections of the individuals within their network, that also causes a reduction in alcohol dependence at the community level with fewer levels of treatments. The study in [23] uses a dynamic mathematical model to adequately predict the actual drinking patterns of students in college campuses. The authors used data from a variety of campuses surveyed in the Social Norms Marketing Research Project study and predicted the impact on drinking patterns of several interventions to address heavy episodic drinking on different types of campuses. [24] conducted a modeling based drinking ecological study that included 74 larger cities in Los Angeles County to assess the geographic association between city-specific rates of assaultive violence and

alcohol-outlet density. The results suggested that sociodemographic factors alone accounted for 70% of the variance in the rate of assaultive violence in a multiple regression model.

In the literature, the mathematical models that study the dynamics of substance abuse in the population mostly follow a deterministic approach, but in reality the system under contemplation is influenced by intrinsic stochastic factors. Social and environmental systems naturally contain uncertainties. Methods are developed to incorporate such uncertainties so that they can be quantified. Stochasticity naturally arises from social, environmental and demographical factors, which can be incorporated into the population models. [18] studied extensively the role of stochasticity in epidemic models where he used the approximations of quasi-stationary distribution (stationary distribution conditional on non-extinction), and times of extinction derived from simple SIR-type stochastic epidemiological models. He underscored the fact that it is extremely complicated to show explicit results for stochastic factors involving large populations. However, approximation of a quasi-stationary distribution leads to a legitimate approximation of the expected time to extinction, starting from this distribution.

This paper builds on those prior studies and poses the question of which social factors most influence alcohol drinking patterns and what strategies will reduce the social interaction of serious drinkers so that the population of heavy drinkers can become negligible. We use the expressions of “time to extinction” and “critical community size” [11] and extend this work by performing uncertainty and sensitivity analysis on them as well as deriving other important quantities to identify factors that may be crucial in designing control policy in colleges. The goal is to compare the impact of different environmental alcohol intervention programs on alcohol drinking distributions.

The paper is structured as follows: In Section 1.1 a review of the dynamical systems model from [16] is presented. Section 2 describes the methods used to calculate the relevant mathematical quantities such as reproduction number, time to extinction, and critical community size. Section 3 explains the uncertainty and sensitivity analyses on the quantities of interest. The relevant quantities include drinking reproduction number, quasi-stationary distribution (QSD), time to extinction of moderate and heavy drinkers (starting from QSD) from the population in the presence of environment-dependent interventions, and critical community size (defined as population size above which stochastic fadeout of a high-risk drinking outbreak over a given period of time). Numerical results from the analysis are in Section 4. The results and their implications are discussed in Section 5.

1.1. Mathematical framework. We use the mathematical framework of [16], which models a population of typical college alcohol drinkers. The population in the model is stratified into S light drinkers, that consume at least once a month but not more than three drinks in one sitting, M moderate drinkers, that drink at least once a month and can consume three to five drinks per sitting, and H heavy drinkers, that drink three to four drinks at least in a week and can consume five or more drinks in one sitting at least once in a month. Figure 1 illustrate the model. The model captures two drinking environments defined as low-risk E_1 and high-risk E_2 . M drinkers are divided into subpopulations M_1 and M_2 based on the two distinct drinking environments. M_1 drinkers tend to encounter S drinkers in low-risk environments E_1 . M_2 drinkers tend to encounter H drinkers in high-risk environments E_2 , M_2 also encounters S in a non-drinking environment. High-risk

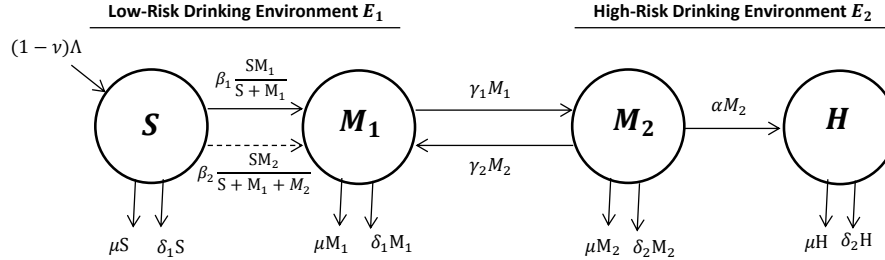


FIGURE 1. Model flowchart where light drinkers (S), moderate in low-risk (M_1), moderate in high-risk (M_2) and heavy drinkers (H) are represented

drinking environment can be venues such as off-campus parties, fraternity parties, pubs, bars while low drinking environments are restaurants, residence halls, and outdoors. We assume that individuals in the S and H drinking classes tend to hang out primarily in low-risk and high-risk drinking environments, respectively. However, M drinkers may move into both drinking environments. S move to M_1 under the social influences of moderate drinkers M ($M = M_1 + M_2$), β_1 is the rate of moving to M_1 after interacting with M_1 , and β_2 is the rate of moving to M_1 after interacting with M_2 and M_2 move to H under a natural drinking progression with a rate α . S can move to H only after passing through the moderate drinking stage. The recruitment into the population is only through S -class and is captured in the rate $(1 - \nu)\Lambda$. The exit from the modeling population occurs at a per capita departure rate μ , intervention exit rate for all classes of drinkers from low-risk and high-risk environment are δ_1 and δ_2 respectively. The per-capita rate of movement of moderate drinkers from E_1 to E_2 is γ_1 and from E_2 to E_1 is γ_2 . The definitions of the variables and parameters are in Table 2.

The model is derived using a stochastic process, $X(t) = (S(t), M_1(t), M_2(t), H(t))$, which is governed by a system of stochastic differential equations (SDE) [16]. The process of entry into the system (or inflow) is a Poisson process with fixed rate parameter Λ . The inflow may be reduced, as a result of a prevention program, by the factor of $(1 - \nu)$. The parameter ν quantifies the effectiveness of a prevention program, with $\nu = 1$ implying perfect prevention. Intervention programs are composed of two Poisson-type processes which are environmentally dependent with rate parameter δ_1 times the class sizes in E_1 and δ_2 times the class sizes in E_2 , at time t . Other transitions are also Poisson-type processes with rate parameters depending on the components of the stochastic process $X(t)$. In fact, these transition rates are conditional rates. The conditional transition rates in an interval, $(t, t + \Delta t)$, of the stochastic process $(S(t), M_1(t), M_2(t), H(t))$ are defined in Table 2. The probability of more than one transition in a time interval Δt is of order $o((\Delta t)^2)$ and therefore neglected for small Δt . In the model, during a time interval $(t, t + \Delta t)$ change in stochastic process is given by

$$\Delta X_t = (\Delta S_t, \Delta M_{1t}, \Delta M_{2t}, \Delta H_t).$$

TABLE 1. Transitions and their rates

Index (i)	Transitions	Conditional rates given $X_t = (S, M_1, M_2, H)$
1	$S \rightarrow S + 1$	$(1 - \nu)\Lambda + o(\Delta t)$.
2	$S \rightarrow S - 1$	$\beta_1 \frac{SM_1}{(S+M_1)} + (\mu + \delta_1)S + \beta_2 \frac{SM_2}{(S+M_1+M_2)} + o(\Delta t)$.
3	$M_1 \rightarrow M_1 + 1$	$\beta_1 \frac{SM_1}{(S+M_1)} + \gamma_2 M_2 + \beta_2 \frac{SM_2}{(S+M_1+M_2)} + o(\Delta t)$.
4	$M_1 \rightarrow M_1 - 1$	$\gamma_1 M_1 + (\mu + \delta_1)M_1 + o(\Delta t)$.
5	$M_2 \rightarrow M_2 + 1$	$\gamma_1 M_1 + o(\Delta t)$.
6	$M_2 \rightarrow M_2 - 1$	$\gamma_2 M_2 + \alpha M_2 + (\mu + \delta_2)M_2 + o(\Delta t)$.
7	$H \rightarrow H + 1$	$\alpha M_2 + o(\Delta t)$.
8	$H \rightarrow H - 1$	$(\mu + \delta_2)H + o(\Delta t)$.

Each component of ΔX_t is the sum of its conditional expected value and a conditional centered random increment, where the conditioning is on $X(t)$. That is,

$$\begin{aligned}
 \Delta S_t &= E[\Delta S_t] + (\Delta S_t - E[\Delta S_t]) &= E[\Delta S_t] + (\Delta Z_1 - \Delta Z_2 - \Delta Z_3), \\
 \Delta M_{1t} &= E[\Delta M_{1t}] + (\Delta M_{1t} - E[\Delta M_{1t}]) &= E[\Delta M_{1t}] + (\Delta Z_2 - \Delta Z_4 - \Delta Z_5), \\
 \Delta M_{2t} &= E[\Delta M_{2t}] + (\Delta M_{2t} - E[\Delta M_{2t}]) &= E[\Delta M_{2t}] + (\Delta Z_4 - \Delta Z_6 - \Delta Z_7), \\
 \Delta H_t &= E[\Delta H_t] + (\Delta H_t - E[\Delta H_t]) &= E[\Delta H_t] + (\Delta Z_6 - \Delta Z_8).
 \end{aligned}
 \tag{1}$$

Here, for example, the conditionally centered random increment $\Delta S_t - E[\Delta S_t]$ is a result of a change in one of its three related transition processes (inflow-, outflow-, influence-processes). The increment ΔZ_i ($i=1,2, \dots,8$) in System (1) represents the i^{th} conditionally centered locally Poisson increment that is related to the change in one of the four components of $X(t)$ (described in Table 1). For example, the centered Poisson increment ΔZ_2 corresponds to transitions representing newly generated moderate drinkers (i.e., transition from S to M_1). This same increment appears in the corresponding representation of ΔM_i with the opposite sign.

On scaling System (1) by a sufficiently large (but finite) deterministic stationary total population size (N), we can replace centered Poisson increments by $G_i \Delta W_i$, for $i = 1, 2, \dots, 8$, where each $G_i \sqrt{\Delta t}$ is the approximate conditional standard deviation of the increment of respective transition and each W_i is a standard Wiener process, that is, $\Delta W_i \sim \mathcal{N}(0, \Delta t)$ [9]. For infinitesimal small increments of time, the system of SDE describing temporal evolution of the process $\frac{X_t}{N} = x_t = (s_t, m_{1t}, m_{2t}, h_t)$, is obtained.

2. Methods. We collect here the relevant quantities obtained from the model. The uncertainty and sensitivity analyses are carried out on the two quantities (or metrics), namely, the drinking reproduction number and the time to extinction of the high-risk drinkers in the presence of interventions.

2.1. Drinking reproduction number. The drinking reproduction number of the model, denoted by R_d , can be derived using the deterministic model. R_d is interpreted as the expected number of secondary moderate M_1 -drinkers produced, in a population of light drinkers, by the social interaction of a typical mobile (moving

TABLE 2. Definitions of Variables and Parameters

Variables & Parameters	Definition	Units
E_1	Low-risk drinking environments	...
E_2	High-risk drinking environments	...
S	Light or Susceptible drinkers	Number of people
M_1	Moderate drinkers in low-risk environment	Number of people
M_2	Moderate drinkers in high-risk environment	Number of people
H	Heavy drinkers	Number of people
β_1	Social interaction parameter influencing S in low-risk environment. This parameter is a combination of average interaction rate and probability of successful conversion given a contact between S and M_1 .	Interactions per person per year
β_2	Social interaction parameter influencing S in high-risk environment. This parameter is a combination of average interaction rate and probability of successful conversion given a contact between S and M_2 .	Interactions per person per year
μ	Graduation and drop out rate	per person per year
α	Progression rate from M_2 to H - drinking state in high-risk environment	per person per year
γ_1	Transition rate for M -drinkers from low-risk environment to high-risk environment	per person per year
γ_2	Transition rate for M -drinkers from high-risk environment to low-risk environment	per person per year
δ_1	Intervention exit rate for all classes of drinkers in low-risk environment	per person per year
δ_2	Intervention exit rate for all classes of drinkers in high-risk environment	per person per year
ν	Intervention proportion targeted to reduce initiation of alcohol drinking (referred sometime in the text as prevention proportion)	Dimensionless
Λ	Recruitment rate of drinkers in the population	per year

between drinking environments) drinker. The analysis of the corresponding deterministic model suggests that *if $R_d \leq 1$ then eventually only light drinkers will persist in the population, and if $R_d > 1$, all three types of drinkers exist in the population for all times but the size of number of H - and M -drinkers eventually will depend on how far the value R_d is above one.* Hence, R_d captures whether there will be a large outbreak of heavy drinkers in the population and if yes, then what will be the size of it over time. The R_d for the model is:

$$R_d(\delta_1, \delta_2) = \frac{\beta_1(1 - \tilde{\gamma}) + \beta_2\tilde{\gamma}}{\mu_1(\delta_1)(1 - \tilde{\gamma}) + (\mu_2(\delta_2) + \alpha)\tilde{\gamma}} \quad (2)$$

where $\mu_1(\delta_1) = \mu + \delta_1$, $\mu_2(\delta_2) = \mu + \delta_2$, and $\tilde{\gamma} \equiv \frac{\gamma_1}{\mu_2 + \gamma_1 + \gamma_2 + \alpha}$. Nontrivial steady state (which exists and stable when $R_d > 1$) is (s^*, m_1^*, m_2^*, h^*) , where

$$m_2^* = \theta_2 h^*, \quad m_1^* = \tilde{\xi} \theta_2 h^*, \quad s^* = \tilde{\Lambda} - \tilde{\eta} h^*, \tag{3}$$

with $\theta_2 = \frac{\mu_2}{\alpha}$, $\sigma = \frac{\mu_2 + \alpha}{\mu_1}$, $\tilde{\xi} = \frac{1 - \tilde{\gamma}}{\tilde{\gamma}}$, $\tilde{\eta} = \theta_2(\sigma + \tilde{\xi})$, $\tilde{\Lambda} = \frac{(1 - \nu)\zeta}{\mu_1}$, $\hat{H} = h^* \frac{\theta_2}{\tilde{\Lambda}}$ and

$$\tilde{a}\hat{H}^2 - \tilde{b}\hat{H} + \tilde{c} = 0, \tag{4}$$

$$\begin{aligned} \tilde{a} &= (\sigma + \tilde{\xi})(\sigma - 1)(R_d - 1) + \tilde{\xi}(\sigma - 1) + \frac{\beta_2}{\mu_1}, \quad \tilde{b} = (R_d - 1)[(\sigma - 1) + (\sigma + \tilde{\xi})] + \frac{\beta_2}{\mu_1(\sigma + \tilde{\xi})} + \tilde{\xi}, \\ \tilde{c} &= (R_d - 1), \quad \tilde{N} = \frac{(1 - \nu)\Lambda}{\mu}, \quad s^* = \frac{S^*}{N}, \quad m_1^* = \frac{M_1^*}{N}, \quad m_2^* = \frac{M_2^*}{N} \quad \text{and} \quad h^* = \frac{H^*}{N}. \end{aligned}$$

2.2. Quasi-stationary distribution of drinkers. If $R_d > 1$, all sample paths of the stochastic model will eventually converge to the “trivial state” ($M_1 = M_2 = H = 0$) even though solutions of the deterministic model converge to a nontrivial steady state. This fact is because the solution of the stochastic model consists of set of distributions of the drinking states index by time, where extinction (or zero) state is possible at all times but with varying probabilities at each time. On the other hand, the solution of the corresponding deterministic model approximate the expected value of the solution (set of distributions index by time) of the stochastic model. The deterministic model predicts that the proportion of moderate and heavy drinkers will eventually approach a positive fixed level for $R_d > 1$, while the stochastic model predicts that eventually moderate and heavy drinkers will become extinct because of the presence of randomness (though the time to extinct might be extremely large). [15] defined the quasi-stationary distribution as the eventual distribution of the system, conditioned on the processes M and H not having gone extinct [16] and its approximation is as follows.

For sufficiently large \tilde{N} , the distribution of the scaled process

$$\sqrt{\tilde{N}} \left(s_t - s^*, m_{1t} - m_1^*, m_{2t} - m_2^*, h_t - h^* \right)$$

can be approximated by the stationary distribution of the stochastic process that has local drift, J (Jacobian matrix of the deterministic model at the endemic state), and covariance matrix, C . This stationary distribution is a multivariate normal with mean 0 and covariance Σ given by the relation.

$$J\Sigma + \Sigma J^T = -C, \tag{5}$$

[1, 18] where $\Sigma = (\sigma_{ij})_{i,j=1,2,3,4}$ represents the covariance between i^{th} and j^{th} of the process $X(t)$ at quasi-stationary,

$$J = \begin{bmatrix} -(P + \mu + \delta_1) & -Q & -T & 0 \\ P & Q - (\mu + \delta_1 + \gamma_1) & T + \gamma_2 & 0 \\ 0 & \gamma_1 & -(\mu + \delta_2 + \gamma_2 + \alpha) & 0 \\ 0 & 0 & \alpha & -(\mu + \delta_2) \end{bmatrix}, \tag{6}$$

and

$$C = \begin{bmatrix} \frac{(1 - \nu)\Lambda}{N} + \frac{\beta_1 s^* m_1^*}{(s^* + m_1^*)} + \frac{\beta_2 s^* m_2^*}{(s^* + m_1^* + m_2^*)} + (\mu + \delta_1) s^* & \dots \\ - \left(\frac{\beta_1 s^* m_1^*}{(s^* + m_1^*)} + \frac{\beta_2 s^* m_2^*}{(s^* + m_1^* + m_2^*)} \right) & \dots \\ 0 & \dots \\ 0 & \dots \end{bmatrix}$$

$$\begin{array}{ccc}
 \dots & - \left(\frac{\beta_1 s^* m_1^*}{(s^* + m_1^*)} + \frac{\beta_2 s^* m_2^*}{(s^* + m_1^* + m_2^*)} \right) & \dots \\
 \dots & \left(\frac{\beta_1 s^* m_1^*}{(s^* + m_1^*)} + \frac{\beta_2 s^* m_2^*}{(s^* + m_1^* + m_2^*)} \right) + (\gamma_1 m_1^* + \gamma_2 m_2^*) + (\mu + \delta_1) m_1^* & \dots \\
 \dots & - (\gamma_1 m_1^* + \gamma_2 m_2^*) & \dots \\
 \dots & 0 & \dots \\
 \dots & 0 & 0 \\
 \dots & - (\gamma_1 m_1^* + \gamma_2 m_2^*) & 0 \\
 \dots & (\gamma_1 m_1^* + \gamma_2 m_2^*) + \alpha m_2^* + (\mu + \delta_2) m_2^* & -\alpha m_2^* \\
 \dots & -\alpha m_2^* & \alpha m_2^* + (\mu + \delta_2) h^*
 \end{array} \Bigg], \tag{7}$$

$$\begin{aligned}
 \text{with } P &= \frac{\beta_1 (m_1^*)^2}{(s^* + m_1^*)^2} + \frac{\beta_2 (m_1^* + m_2^*) m_2^*}{(s^* + m_1^* + m_2^*)^2}, \\
 Q &= \frac{\beta_1 (s^*)^2}{(s^* + m_1^*)^2} - \frac{\beta_2 s^* m_2^*}{(s^* + m_1^* + m_2^*)^2}, \\
 \text{and } T &= \frac{\beta_2 s^* (s^* + m_2^*)}{(s^* + m_1^* + m_2^*)^2}.
 \end{aligned}$$

Since the covariance matrix Σ is symmetric, equation (5) can be reduced to the following linear system

$$\begin{array}{cccccc|cccc}
 2j_{11} & 2j_{12} & 2j_{13} & 0 & 0 & \dots & \sigma_{11} & -c_{11} \\
 j_{21} & (j_{11} + j_{22}) & j_{23} & 0 & j_{12} & \dots & \sigma_{12} & -c_{12} \\
 0 & j_{32} & (j_{11} + j_{33}) & 0 & 0 & \dots & \sigma_{13} & 0 \\
 0 & 0 & j_{43} & (j_{11} + j_{44}) & 0 & \dots & \sigma_{14} & 0 \\
 0 & 2j_{21} & 0 & 0 & 2j_{22} & \dots & \sigma_{22} & -c_{22} \\
 0 & 0 & j_{21} & 0 & j_{32} & \dots & \sigma_{23} & -c_{23} \\
 0 & 0 & 0 & j_{21} & 0 & \dots & \sigma_{24} & 0 \\
 0 & 0 & 0 & 0 & 0 & \dots & \sigma_{33} & -c_{33} \\
 0 & 0 & 0 & 0 & 0 & \dots & \sigma_{34} & -c_{34} \\
 0 & 0 & 0 & 0 & 0 & \dots & \sigma_{44} & -c_{44}
 \end{array} =$$

where $J = (j_{ij})$ and $C = (c_{ij})$. Since J and C are known, we can compute the matrix Σ . Hence, the approximate mean and covariance of the quasi-stationary distribution of the process X_t are

$$\tilde{N}(s^*, m_1^*, m_2^*, h^*) \quad \text{and} \quad \tilde{N}^2 \Sigma,$$

respectively. The mean, variance and coefficient of variation of light (S), moderate ($M = M_1 + M_2$) and heavy (H) drinking states are

$$\tilde{\mu}_S = Mean(S) = \tilde{N}s^*, \quad \tilde{\sigma}_S^2 = Var(S) = \tilde{N}^2\sigma_{11}, \quad \text{and} \quad CV(S) = \frac{\tilde{\sigma}_S}{\tilde{\mu}_S} \quad (8)$$

$$\tilde{\mu}_M = \tilde{N}(m_1^* + m_2^*), \quad \tilde{\sigma}_M^2 = \tilde{N}^2(\sigma_{22} + 2\sigma_{23} + \sigma_{33}), \quad \text{and} \quad CV(M) = \frac{\tilde{\sigma}_M}{\tilde{\mu}_M} \quad (9)$$

$$\tilde{\mu}_H = \tilde{N}h^*, \quad \tilde{\sigma}_H^2 = \tilde{N}^2\sigma_{44}, \quad \text{and} \quad CV(H) = \frac{\tilde{\sigma}_H}{\tilde{\mu}_H}. \quad (10)$$

The coefficient of variation indicates how “far away” from the absorption the epidemic process is at equilibrium. Covariance between states can also be computed and are:

$$Cov(S, M_1) = \tilde{N}\sigma_{12}, \quad Cov(S, M_2) = \tilde{N}\sigma_{13}, \quad \text{and} \quad Cov(M_1, M_2) = \tilde{N}\sigma_{23}. \quad (11)$$

The correlation matrix $\rho = (\rho_{ik})$ of quasi-stationary process is

$$\rho = (V^{1/2})^{-1}\Sigma(V^{1/2})^{-1} \quad (12)$$

where $V^{1/2} = (v_{ij}^{1/2})$ is the standard deviation matrix such that $v_{ii}^{1/2} = \sqrt{\sigma_{ii}}$ and $v_{ij}^{1/2} = 0$ for $i \neq j$. The quasi-stationary distribution is used in modeling long-term behavior of a stochastic system conditioned on non-extinction.

2.3. Time to extinction of high-risk drinkers. The random variable describing time to extinction starting with the quasi-stationary distribution is denoted by T_Q . The quasi-stationary state is defined by the state of the process X_t conditioning on non-extinction of the M and H sub-processes. The behavior of the stochastic process used here appears to be stationary over a finite time scale but it seems to terminate eventually in some sense [20]. The absorption state is defined as the state where the two components M_1 and M_2 are zero, after reaching this state if there is any remaining at the component H it will exit the system and become zero. Thus, by eventual termination (or extinction) of the model’s sample paths we mean arriving at the state where the components M and H are zero. The first time a sample path reaches this state is referred as time to extinction of the sample path.

The time it takes to move from the quasi-stationary state to any state where $M_1 = M_2 = 0$ is exponentially distributed. The probability that the process is in state X at time t , given that it has not yet become absorbed into the absorbing state (or state of extinction) is given by

$$\begin{aligned} q_{S, M_1, M_2, H}(t) &= P\{S_t = S, M_{1t} = M_1, M_{2t} = M_2, H_t = H \mid M_{1t} = M_{2t} \neq 0\} \\ &= \frac{p_{S, M_1, M_2, H}(t)}{(1 - p_{*, 0, 0, *}(t))}. \end{aligned} \quad (13)$$

A process that has been running for a long time and has not gone to the absorbing state is approximately in the quasi-stationary distribution $Q = \{q_{S, M_1, M_2, H}\}$, where

$$q_{S, M_1, M_2, H} = \lim_{t \rightarrow \infty} q_{S, M_1, M_2, H}(t).$$

Using a jump process (with rates mentioned in Table 1), and starting from the quasi-stationary state, various mathematical quantities can be derived.

For example, the expected time spent in states $(M_1, M_2) = (0, 1)$, $(M_1, M_2) = (1, 0)$, and $M = 1$ are derived as

$$E(V^{01}) = p(cL_1 + l_2L_2)(1 - A) + p\tilde{g}(cL_1 + b_1L_2)A, \tag{14}$$

$$E(V^{10}) = g\tilde{p}(cL_1 + b_2L_2)(1 - A) + g(cL_1 + l_1L_2)A, \text{ and} \tag{15}$$

$$E(W) = (c(p + g\tilde{p})L_1 + (pl_2 + g\tilde{p}b_2)L_2)(1 - A) \tag{16}$$

$$+ (c(g + p\tilde{g})L_1 + (gl_1 + p\tilde{g}b_1)L_2)A, \tag{17}$$

respectively, where $M = M_1 + M_2$, $A = \frac{M_1^*}{M_1^* + M_2^*}$, $p = \frac{\mu + \delta_2 + \alpha + \beta_2 \frac{S^*}{S^* + 1}}{\gamma_2 + \mu + \delta_2 + \alpha + \beta_2 \frac{S^*}{S^* + 1}}$, $g = \frac{\mu + \delta_1 + \beta_1 \frac{S^*}{S^* + 1}}{\gamma_1 + \mu + \delta_1 + \beta_1 \frac{S^*}{S^* + 1}}$, $c_1 = \frac{1}{\gamma_1}$, $c_2 = \frac{1}{\gamma_2}$, $\tilde{p} = 1 - p$, $\tilde{g} = 1 - g$, $\tilde{h} = \tilde{p}\tilde{g}$, $c = c_1 + c_2$, $b_1 = c_1 + l_2$, $b_2 = c_2 + l_1$, $l_1 = \frac{1}{\mu + \delta_1 + \beta_1 \frac{S^*}{S^* + 1}}$, $l_2 = \frac{1}{\mu + \delta_2 + \alpha + \beta_2 \frac{S^*}{S^* + 1}}$, $L_1 = \frac{\tilde{h}}{(1 - \tilde{h})^2}$, and $L_2 = \frac{1}{(1 - \tilde{h})}$. The marginal quasi-stationary probability of state $M = 1$ denoted by $q_{*,\bar{1},*}$, can be expressed as

$$q_{*,\bar{1},*} = \frac{1}{\tilde{\sigma}_M} \left(\frac{\phi\left(\frac{1 - \tilde{\mu}_M}{\tilde{\sigma}_M}\right)}{1 - \Phi\left(\frac{0.5 - \tilde{\mu}_M}{\tilde{\sigma}_M}\right)} \right) \tag{18}$$

where the function $\phi(\cdot)$ is the standard normal density, $\Phi(\cdot)$ is the cumulative distribution function, $\tilde{\mu}_M = \tilde{N}(m_1^* + m_2^*)$, and $\tilde{\sigma}_M^2 = \tilde{N}^2(\sigma_{22} + 2\sigma_{23} + \sigma_{33})$. The expected value of T_Q is [15]

$$\tau_Q = E[T_Q] = \frac{1}{q_{*,\bar{1},*}L} \tag{19}$$

where $\frac{1}{L} = \frac{E[W]}{(\mu + \delta_2 + \alpha)E[V^{01}] + (\mu + \delta_1)E[V^{10}]}$.

2.4. Critical community size of a population of drinkers. The expected time to extinction, when the initial distribution is the quasi-stationary distribution (Equation (19)), is $E(T_Q)$. It depends on model parameters. Since the non-trivial steady state is a function of \tilde{N} , probabilities (p , g , A and $q_{*,\bar{1},*}$) and times (l_1 and l_2) are also functions of \tilde{N} . Hence, $E[W]$, $E[V^{01}]$ and $E[V^{10}]$ are functions of \tilde{N} . Therefore, if other parameters are fixed then τ_Q can be written as a function of \tilde{N} . As defined by [1] in an epidemic context, The *Critical Community Size* \tilde{N}_c is an upper bound on the community size (that is, total modeling population) for the heavy drinking epidemic to have a fair chance of going extinct before a given time. More precisely, the critical community size, $\tilde{N}_c(t^*, p^*)$, depends on the time horizon t^* and extinction probability p^* , and satisfies $P(T_Q \leq t^*) = p^*$. Since T_Q is exponentially distributed, we can obtain \tilde{N}_c by:

$$1 - e^{-\lambda(\tilde{N}_c)t^*} = p^*, \quad \text{where } \lambda(\tilde{N}_c) = \frac{1}{\tau_Q(\tilde{N}_c)}.$$

Hence, if τ_Q is a bijective function of \tilde{N}_c , then by the implicit value theorem we get

$$\tilde{N}_c = \tau_Q^{-1} \left(-\frac{t^*}{\ln(1 - p^*)} \right). \tag{20}$$

One cannot invert τ_Q explicitly. However, \tilde{N}_c can be computed numerically. The computation of \tilde{N}_c involves the following steps. The critical community size (i.e.,

$\tilde{N}_c(t, p)$), which depends on the time horizon t and extinction probability p , is the solution \tilde{N} that satisfies $P(T_Q \leq t) = p$, i.e.,

$$1 - e^{-\lambda(\tilde{N})t} = p \tag{21}$$

$$q_{*,\bar{1},*}(\tilde{N}) L(\tilde{N}) = \frac{1}{t} \ln \left(\frac{1}{1-p} \right) \tag{22}$$

where $q_{*,\bar{1},*}(\tilde{N}) = \frac{\tilde{N}(\sigma_{22} + \sigma_{33} + 2\sigma_{23}) \left(1 - \Phi \left(\frac{0.5 - \tilde{N}(m_1^* + m_2^*)}{\tilde{N}(\sigma_{22} + \sigma_{33} + 2\sigma_{23})} \right) \right)}{\phi \left(\frac{(1 - \tilde{N}(m_1^* + m_2^*))}{\tilde{N}(\sigma_{22} + \sigma_{33} + 2\sigma_{23})} \right)}$,
 $L(\tilde{N}) = \frac{(\mu + \delta_2 + \alpha)E_1(\tilde{N}) + (\mu + \delta_1)E_2(\tilde{N})}{E_3(\tilde{N})}$, $E_1 = E(V^{01})$, $E_2 = E(V^{10})$, and $E_3 = E(W)$.

3. Analysis. We carry out global uncertainty and sensitivity analyses on two mathematical quantities: drinking reproduction number (Equation (2)) and expected time to extinction (Equation (19)) with respect to the model parameters. In these analyses, model parameters are random variables and our aim is to study the effect of variations in parameters on the changes in model outcomes like R_d and τ_Q .

We employ the Latin Hypercube Sampling method (LHS), where sampling is carried out on a high-dimensional parameter space. In this method, the parameter space is divided into N equal probability intervals. Using data from [12], a probability distribution is assigned to each of the model parameters. Table 3 and Figure 2 show the distribution of the parameters assumed in our analyses [12]. 10,000 sample sets from the parameter space (each set is comprised of a sample from each of the parameters involved in the output variables) were chosen from equal probability interval areas and used to compute estimates of the output variables (R_d and τ_Q).

We consider three different scenarios based on the intervention programs in the two drinking environments: *Case 1* ($\delta_1 < \delta_2$), where stronger intervention programs are implemented in high-risk environments rather than low-risk environments; *Case 2* ($\delta_1 = \delta_2$) represents the scenario of intervention programs in both types of drinking environments being equal; and *Case 3* ($\delta_1 > \delta_2$) is related to the case where stronger intervention programs are implemented in the low-risk environment rather than high-risk environment.

Since the non-trivial steady state exists and is stable if $R_d > 1$ (as mentioned in Section 2.1), the uncertainty and sensitivity analysis of time to extinction τ_Q is carried out by first computing a distribution of β_2 using assigned distributions of other parameters and the condition

$$\beta_2 > \frac{\mu_1(1 - \tilde{\gamma}) + (\mu_2 + \alpha)\tilde{\gamma} - \beta_1(1 - \tilde{\gamma})}{\tilde{\gamma}}$$

ensuring estimates of β_2 such that $R_d > 1$.

3.1. Uncertainty analysis. In the uncertainty analysis, we investigate and quantify uncertainties in the relevant output variables as a function of uncertainty in measurement of input variables (or model parameters) [10, 12, 18]. The idea is to explore the different parameters in a systematic way. A procedure is designed and performed to determine an effect, or to estimate how the numerical value of an output variable will be affected by errors due to methodology, presence of confounding effects and measurements. Experimental uncertainty estimates are needed to assess the confidence in the estimates of output variables.

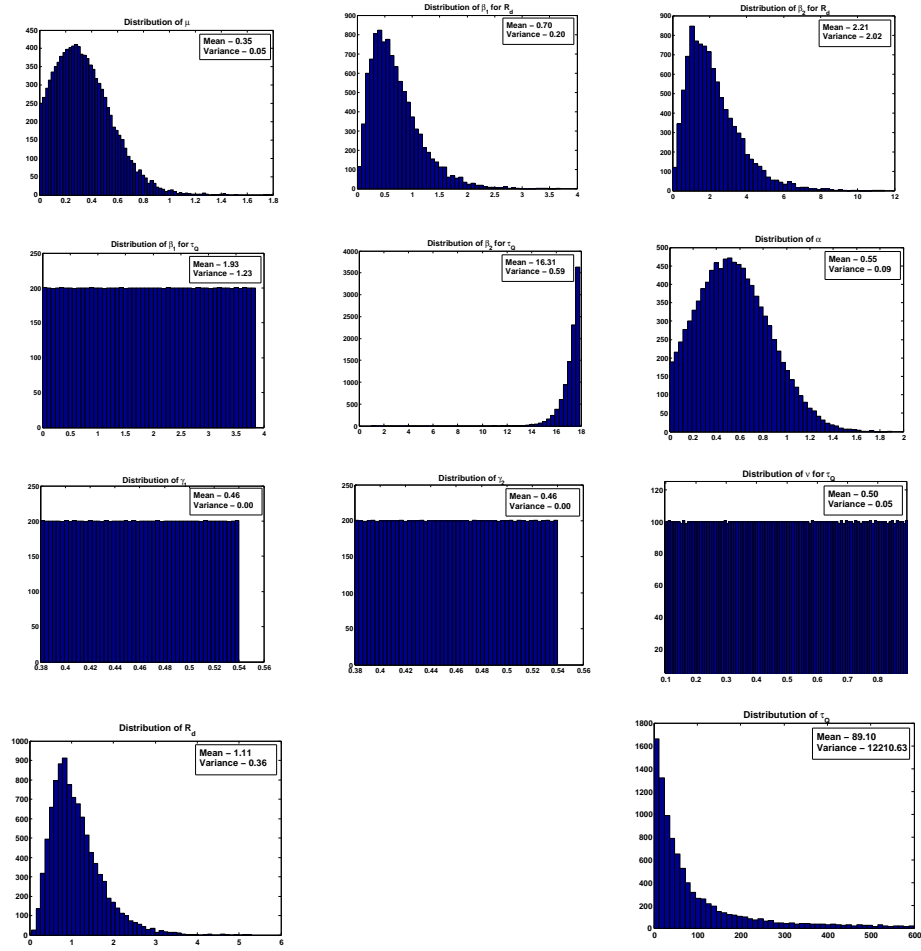


FIGURE 2. Assumed probability distributions of input parameters (μ , β_1 , β_2 , α , γ_1 , γ_2 and ν) in the uncertainty and sensitivity analyses. Since time to extinction (τ_Q) is computed for $R_d > 1$, the distributions of β_1 and β_2 are modified to ensure $R_d > 1$. The distributions of output variables (R_d and τ_Q) from the analyses are also shown.

3.2. Sensitivity analysis. Uncertainty and sensitivity analyses are run in tandem. Sensitivity analysis identifies factors that contribute mostly to the output variability. We use a sensitivity procedure to determine how different values of an input variable will affect a particular output variable under a given set of assumptions and in the presence of other input variables [12]. We compute partial rank correlation coefficients (PRCC) of the outputs with respect to each of the parameters involved. The PRCC is a sensitivity index that lies between -1 and 1 and is used to compare the effect of input parameters on an output variable. Sensitivity indexes are shown in bar graphs with black and gray representing negative- and positive-sensitivity,

respectively. If the absolute value of a sensitivity index is greater than 0.5, then we consider the parameter to be significantly sensitive. The computed PRCC values are in Table 5.

TABLE 3. Distribution and values of parameters used in the analysis of R_d and τ_Q . The distributions that are used here include Normal (\mathcal{N}), Gamma (Γ), and Uniform (\mathcal{U}) distribution.

Parameter	Parameter distribution/values of in R_d analysis	Parameter distribution/values in τ_Q analysis
μ	$\mathcal{N}_{[0,\infty)}(Mean=0.35, Var=0.11)$	$\mathcal{N}_{[0,\infty)}(Mean=0.37, Var=0.04)$
β_1	$\Gamma(k = 2.49, \theta = 0.28)$	$\mathcal{U}(a = 0.001, b = 3.85)$
β_2	$\Gamma(k = 2.47, \theta = 0.89)$	$\mathcal{U}(a = 0, b = 17.42)$
α	$\mathcal{N}_{[0,\infty)}(Mean=0.55, Var=0.09)$	$\mathcal{N}_{[0,\infty)}(Mean=0.25, Var=0.03)$
γ_1	$\mathcal{U}(a = 0.38, b = 0.54)$	$\mathcal{U}(a = 0.30, b = 0.62)$
γ_2	$\mathcal{U}(a = 0.38, b = 0.54)$	$\mathcal{U}(a = 0.38, b = 0.62)$
ν	...	$\mathcal{U}(a = 0.10, b = 8.99)$
δ_1 (when $\delta_1 < \delta_2$)	0.4	0.4
δ_2 (when $\delta_1 < \delta_2$)	0.8	0.8
δ_1 (when $\delta_1 = \delta_2$)	0.5	0.5
δ_2 (when $\delta_1 = \delta_2$)	0.5	0.5
δ_1 (when $\delta_1 > \delta_2$)	0.8	0.8
δ_2 (when $\delta_1 > \delta_2$)	0.4	0.4
ζ	...	3

TABLE 4. University of California UC Digest 2003: University of California system schools and intake. We assume that annual intake of schools is 1.5 times fall semester intake. College intake Data is mixed with college drinking Data from [26, 4, 5] data suggested that 65% of freshman are drinkers.

UC Digest 2003			Using assumptions of [3]	
School	Total undergrad.	Total first year admitted in fall	Drinkers admitted in a year	of drinkers admitted per year
UC Berkeley	24,636	3,814	3,718	15
UCLA	25,432	4,246	4,139	16
UC Davis	23,458	4,412	4,301	18
UC SD	21,369	3,981	3,881	18
UCI	21,854	4,048	3,946	18
UC Riverside	14,973	3,270	3,188	21
				17.6

4. **Results.** The uncertainty and sensitivity analyses were carried out by fixing some known parameters, whereas assuming distribution for parameters for which exact value with precision were not found. We begin with estimation of fixed parameters from data, as well as estimation of distribution of unknown parameters.

4.1. **Point and distribution estimates of parameters.** We considered schools in the University of California system as our modeling population and estimated relevant parameters using data from University of California (UC) digest [26, 4, 5]. The parameter Λ is assumed to be equal to ζN , where ζ is a fraction of the total population (N) of the UC system. The estimate of the parameter ζ is 0.17, which

TABLE 5. Sensitivity indices (PRCC values) from the sensitivity analysis.

		μ Exit rate	β_1 Social interactions in E_1	β_2 Social interactions in E_2	α Progression rate $M_2 \Rightarrow H$	γ_1 Transition rate $E_1 \rightarrow E_2$	γ_2 Transition rate $E_2 \rightarrow E_1$	ν Intervention proportion to reduce initiation
R_d	$\delta_1 < \delta_2$	-0.4359	0.6886	0.4859	-0.1275	0.0156	0.0019	...
	$\delta_1 = \delta_2$	-0.4359	0.6471	0.5255	-0.1614	0.0288	-0.0029	...
	$\delta_1 > \delta_2$	-0.3862	0.6487	0.5546	-0.1739	0.0438	-0.0071	...
τ_Q	$\delta_1 < \delta_2$	-0.9389	0.0304	-0.0100	-0.0684	0.0055	0.0083	-0.6104
	$\delta_1 = \delta_2$	-0.9401	0.0173	-0.00005	-0.0527	-0.0028	-0.0059	-0.6159
	$\delta_1 > \delta_2$	-0.9395	0.0037	-0.0099	-0.0868	0.0063	-0.0109	-0.6087

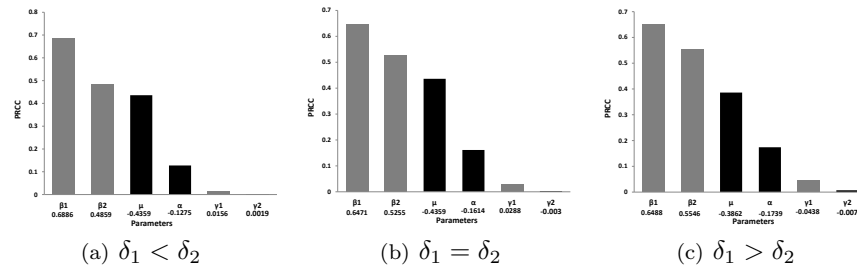


FIGURE 3. Influence of parameters on R_d

is obtained from Table 4. The parameter Λ is estimated using ζ and the total population of the schools mentioned in Table 4.

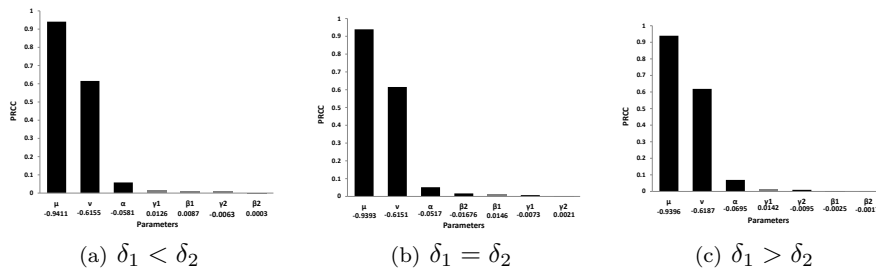


FIGURE 4. Influence of parameters on τ_Q

We also estimate the average intervention rate for Case 2 ($\delta_1 = \delta_2 = \delta$). The total population of the deterministic system is $s^* + m_1^* + m_2^* + h^* = 1 - \frac{\delta}{\bar{\gamma}\alpha}$ and so $\delta \leq \bar{\gamma}\alpha$. Efficacy reviews of brief interventions reveal that if applied to the total population at risk, they would reduce the overall prevalence of high-risk drinking by 35 to 50 percent, equivalent to a 14 to 18 percent improvement in the rate of recovery without intervention at all. We assume 70% adherence and potential intervention coverage of 50% in the population of drinkers and estimated remission rates to be between 4.9 and 6.4 percent higher than natural history rates. Hence, we consider $1 - \frac{\delta}{\bar{\gamma}\alpha} = \frac{(14+18)*100}{2}$, that is, $\delta = 0.1225$. The end points of intervals for parameter-distribution are assumed using values of the parameters from [15]. Note,

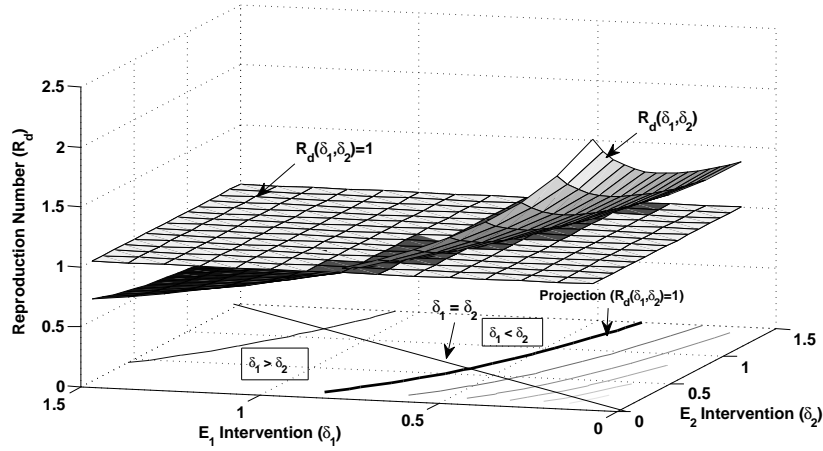


FIGURE 5. The variability of R_d as a function of δ_1 and δ_2

the parameter estimates used in the study is merely to provide its relative (to other parameters) significance as an example and its value needs to be understood from the perspective of sensitivity analysis results.

Figure 2 shows assumed distributions of the parameters and estimated distributions of the output variables. The distribution of output variables for R_d and τ_Q , when the $\delta_1 = \delta_2$, are also in this figure. The expected value and variance for the distribution of parameters are taken from [15]. The statistical summary of the distribution is represented in Table 7, and Table 8.

4.2. Uncertainty and sensitivity analysis of R_d . The analysis of the deterministic model confirms that the high-risk drinking can be reduced quickly if R_d is decreased below 1. Hence, it is natural to study how parameters, especially interventions programs (captured by parameters δ_1 and δ_2), reduce R_d . Figure 5 shows changes in the mean value of R_d when δ_1 and δ_2 are varied, while other parameters are fixed. It seems that for small values of δ_1 , the mean estimates of R_d remain greater than one under fixed δ_2 values. Similar trends are observed for small values of δ_2 under fixed δ_1 . When δ_1 or δ_2 increases it follows that the mean of R_d decreases, however the decrease of R_d is faster with an increase in δ_1 as compared to δ_2 . These results suggest that when intervention programs are applied in both low and high-risk environments, smaller values of δ_2 have more effect on reducing the high-risk drinkers in the populations, but large values of δ_1 are needed to contain the high-risk drinkers faster and to get lasting effect. Hence, the results conclude targeting of the high-risk environments by interventions when the funding are limited and focusing on low-risk environments, too, if funding is available. Table 6 provides summary statistics of the R_d distribution for the three cases in the sensitivity analysis (Figure 3).

We perform sensitivity analysis to see how the δ'_i s $i = 1, 2$ impacted our output variables when they are equal as well as when they are not equal. We notice, in all the three cases ($\delta_1 < \delta_2$, $\delta_1 = \delta_2$ and $\delta_1 > \delta_2$), that the reproduction number R_d is most sensitive to β_1 and β_2 (Figures 3(a)-3(c)); in fact, β_1 and β_2 are the only two significantly sensitive parameters. β_1 is positively highly correlated with

the reproduction number R_d in all the three cases, but when $\delta_1 > \delta_2$, the difference between the PRCCs of β_1 and β_2 is smaller. If the intervention rates in the high-risk environments are large (that is, $\delta_1 < \delta_2$), then sensitivity results suggest increases in social interactions in low-risk environments will increase the estimates of R_d more as compared to increases in social interactions in high-risk environments. This could be because E_1 is a provider of drinkers for E_2 environments. In Figure 3(c), when the intervention focus is on low-risk environment (that is, $\delta_1 > \delta_2$), the value of the PRCC between R_d and β_1 decreases compared to case when $\delta_2 > \delta_1$. The stronger interventions in E_1 made the social interaction in E_1 unimportant.

As Figure 6 shows, if the intervention rates are less than approximately 0.6, the expected value of R_d values tend to be greater than 1 and the variability on R_d estimates is high. Alcohol abuse soars in the population because of low (and hence ineffective) intervention levels. The parameter δ_1 seems to make more impact on R_d values than δ_2 , which is because it is more effective to influence moderate drinkers in a low-risk environment than in a high-risk environment.

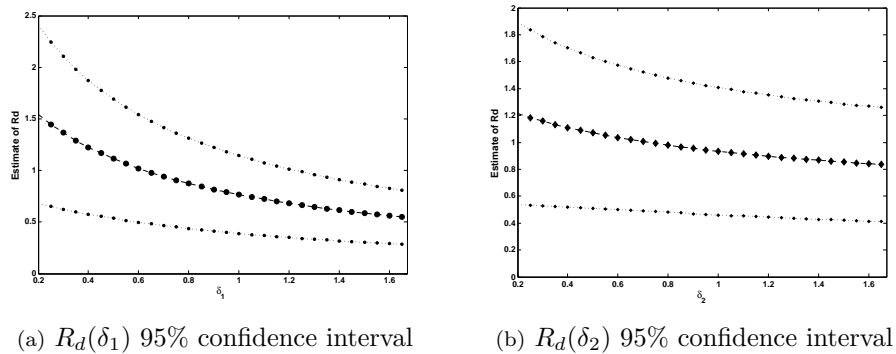


FIGURE 6. The confidence interval of the mean of R_d , (a) δ_2 is fixed and δ_1 vary, (b) δ_1 is fixed and δ_2 vary.

4.3. Uncertainty and sensitivity analysis of τ_Q . The time to extinction (τ_Q) is the time it takes the population of high-risk drinkers to die out in the presence of intervention. Evaluating τ_Q we see in Figures 4(a) - 4(c), the parameters μ and ν are highly correlated with time to extinction in all three cases ($\delta_1 < \delta_2, \delta_1 = \delta_2$ and $\delta_1 > \delta_2$), while social interactions parameters (β_1 and β_2) becomes influential. Since ν captures the prevention programs, efforts to educate college students against alcohol use and abuse can reduce the alcohol user population. The social interaction parameters are not the greatest influencers in these scenarios. The parameter μ is the parameter of college students leaving the population of drinkers through voluntary quitting drinking or exiting (transferring to an other school, dropping out or graduating) college. Time to extinction τ_Q is related to the reproduction number R_d , not explicitly, but indirectly. When R_d increases, time to extinction (τ_Q) increases. The parameters β_1 and β_2 does not affect τ_Q , as in the cases of R_d . The variability in τ_Q estimates are higher when $\delta_2 > \delta_1$ than when $\delta_1 > \delta_2$.

4.4. Changes in critical community size of drinkers, \tilde{N}_c . Critical community size is the minimal population size of high-risk drinkers below which the drinkers

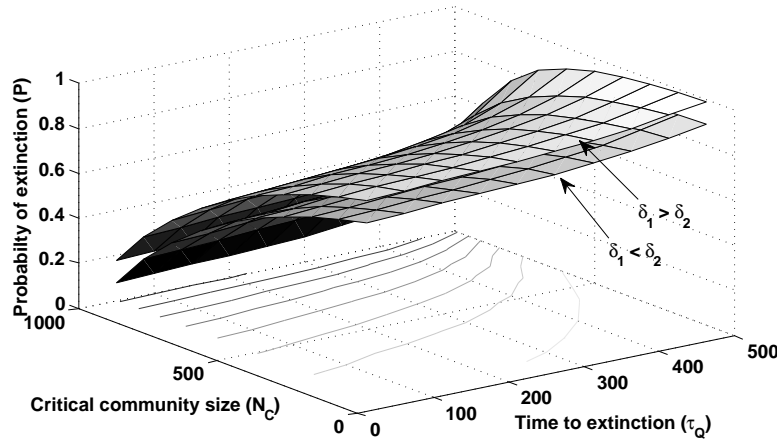


FIGURE 7. Critical community size of drinkers as a function of probability of extinction and time of extinction

population goes to extinction. Figure 7 shows the effect of probability of extinction of high-risk drinkers on critical community size of drinker's population for a given time horizon. The results suggest that for the given probability of extinction, the critical community size of drinkers increases with increases in time horizon (Figure 7). For fixed time period of extinction, critical community size decreases exponentially with increase in likelihood of extinction in that period. The probability of extinction are more higher when the $\delta_1 > \delta_2$, as a result the the effectiveness of interventions programs in low-risk environment are more than high-risk environment.

5. Discussion. The long term success of intervention programs for reducing heavy alcohol drinkers in a college population depends on the availability of resources and is directly related to our ability to identify where and how much to intervene. In this study, we use model based uncertainty and sensitivity analyses on the derived metrics that measure the temporal impact of the intervention programs. The usefulness of these analyses is immense as these methods not only help in identifying important environmental factors to consider when implementing intervention programs but also help in deciding how much to focus on these factors. The measures that are found to be important through these analyses usually require precise quantification from field data. We primarily identify factors that are most sensitive to the two metrics, namely, drinking reproduction number and time to extinction of high-risk drinkers.

Mathematical analysis suggests that heavy drinking can be reduced if the drinking reproduction number (R_d), which depends on social and environmental factors, can be brought below one. That is, the value of the estimate of R_d for a population predicts whether the extinction or the persistence of heavy drinkers will happen in that population. Uncertainty analysis results for R_d using some preliminary data from colleges in California indicate that the average intervention rate in a population should be better than 0.6 per year (i.e., on an average drinkers should avail interventions within about 1 year and 9 months) to bring R_d below one. Moreover, a higher rate of intervention in the low-risk environments is much more effective

than the same rate of intervention in the high-risk environments, when interventions are being implemented in both the environments. The sensitivity analysis of R_d indicates that social interactions are the most significant factors influencing R_d estimates irrespective of size of intervention rates in the two drinking environments.

Uncertainty and sensitivity analyses were also carried out on the metric representing time to extinction of high-risk drinkers in the presence of intervention programs. This metric provides an indirect measure of the success of the intervention programs in a college population where heavy drinking is a norm. The parameter related to intervention programs that reduces initiation of drinkers (ν) is significantly negatively correlated with the time to extinction of heavy drinkers (τ_Q). That means, that time to extinction of high-risk drinkers decreases with increase in the level of interventions targeted to reduce rate of recruitment of new alcohol drinkers. In addition, the parameter (μ), representing the graduation and dropout rate of the drinkers, is found to be the second most sensitive to τ_Q . That is, improving graduation and dropout rates among college students may also reduce time to extinction of high-risk drinkers from the population. These sensitivity results of τ_Q remain consistent even with contrasting variations in the intervention rates between the two environments. In fact, τ_Q is insensitive to all other social interactions and drinking progression parameters. This suggest that high-risk drinking can be effectively controlled faster by reducing drinking initiation rate or improving graduation and dropout rates in a college population.

In the context of infectious diseases, [2] introduced the concept of a critical community size, below which a population cannot sustain the disease for long term without external inputs of infection. We compute estimates of critical community sizes of drinkers for various values of time horizon for extinction and likelihood of extinction within the time horizon. Our results suggest that critical community size decreases exponentially with increase in the probability of extinction of high-risk drinkers for a given time horizon of extinction irrespective of rates of intervention programs. However, the probability of extinction is higher when intervention programs are implemented with higher rates in the low-risk environment than intervention rates in the high-risk environment. In general, we showed that higher interventions rates in low-risk environments are likely to be more effective than in others as they are the feeder for high-risk environments and early stage intervention may reduce social influence of heavy drinkers drastically.

In order to parameterize the model of this study, data that quantify environment-dependent social interactions, interventions, and dropout rates of different types of college drinkers, need to be collected. It appears that such data from a single study are unavailable. The primary goal of this study was to identify the average impact of environment-dependent intervention programs at a *population level* using various metrics. We do not attempt to evaluate any particular data sets. Hence, we did not focus on estimation of parameters and ignored several factors, for example, individual variations in alcohol consumption, including psychological and biological factors which may make the individual more prone to heavy alcohol use. Moreover, in the model, we assumed *constant (averaged)* social interaction rates between drinkers, however, social contacts of a drinker changes continuously and such changes may influence the results. Certainly implications from incorporating these factors in the model need to be studied and will be incorporated in our future research. An example of such a model might be an individual-based model capturing various psychological and biological factors as well as a changing social network.

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Appendix

TABLE 6. Statistical summary of the output variables

		Mean	Minimum	First Quartile	Median	Third Quartile	Maximum	Std Dev
R_d	$\delta_1 < \delta_2$	1.1198	0.0493	0.6932	1.0034	1.4233	4.7000	0.5958
	$\delta_1 = \delta_2$	1.1145	0.0520	0.6894	0.9963	1.4137	5.2946	0.5974
	$\delta_1 > \delta_2$	0.9072	0.0433	0.5703	0.8152	1.1490	4.2771	0.4732
τ_Q	$\delta_1 < \delta_2$	105.59	1.05	21.68	58.66	140.27	658.79	121.52
	$\delta_1 = \delta_2$	89.10	1.01	18.72	48.41	116.93	599.69	105.88
	$\delta_1 > \delta_2$	88.74	1.00	17.62	45.96	110.89	597.91	111.13

TABLE 7. Distribution Statistical Summary for calculating R_d

Parameter	Mean	Minimum	First Quartile	Median	Third Quartile	Maximum
μ	0.3516	0.0012	0.18166	0.3235	0.4853	1.8770
β_1	0.6994	0.0048	0.3714	0.6078	0.9426	3.6930
β_2	2.2149	0.0017	1.576	1.9189	2.9443	11.4635
α	0.5547	0.0002	0.3237	0.5341	0.7529	1.9024
γ_1	0.4600	0.3800	0.4200	0.4600	0.5000	0.5399
γ_2	0.4600	0.3800	0.4200	0.4600	0.5000	0.5399

TABLE 8. Distribution Statistical Summary for calculating τ_Q

Parameter	Mean	Minimum	First Quartile	Median	Third Quartile	Maximum
μ	0.3448	0.0011	0.1779	0.3171	0.4782	1.7952
β_1	1.9255	0.0012	0.9632	1.9254	2.8879	3.8498
β_2	16.3140	0	14.0960	17.4270	17.4270	17.4270
α	0.2521	0.0001	0.1269	0.2333	0.3546	1.1136
γ_1	0.4600	0.3000	0.3799	0.4600	0.5399	0.6199
γ_2	0.4600	0.3800	0.3801	0.4599	0.5401	0.6199
ν	0.5000	0.1000	0.2999	0.4999	0.7000	8.9999

E-mail address: r-bani@neiu.edu; r-hameed@neiu.edu; s-szymanowski@neiu.edu

E-mail address: pgreenw@math.ubc.ca; kribs@uta.edu; a-mubayi@neiu.edu