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A DOSE-RESPONSE PERSPECTIVE ON COLLEGE DRINKING AND RELATED PROBLEMS

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Abstract

AIMS—In order to examine the degree to which heavy drinking specifically contributes to risks for problems among college drinkers this paper develops and tests a dose-response model of alcohol use that relates frequencies of drinking specific quantities of alcohol to the incidence of drinking problems.

METHOD—A mathematical model was developed that enabled estimation of dose-response relationships between drinking quantities and drinking problems using self-report data from 8,698 college drinkers across 14 campuses in California, USA. The model assumes that drinking risks are a direct monotone function of the amount consumed per day and additive across drinking days. Drinking problems accumulate across drinking occasions and are the basis for cumulative reports of drinking problems reported by college drinkers.

RESULTS—Statistical analyses using the model showed that drinking problems were related to every drinking level, but increased 5-fold at three drinks and more gradually thereafter. Problems were most strongly associated with occasions on which three drinks were consumed, and over half of all reported problems were related to occasions on which 4 or fewer drinks were consumed. There were some important differences in dose-responsiveness between men and women and between different groups of “light,” “moderate,” and “heavier” drinkers.

CONCLUSIONS—Many problems among college students are associated with drinking relatively small amounts of alcohol (2 – 4 drinks). Programs to reduce college drinking problems should emphasize risks associated with low drinking levels.

Keywords

College Drinking; Dose-Response; Alcohol Problems; Epidemiology; Prevention

The past 60 years of alcohol research has set a high standard for establishing reliable and valid measures of alcohol use(1–3). During this time, researchers have seen the emergence of surveillance systems that permit large-scale studies of drinking in the general population(4), college youth(5), and among addicted and dependent drinkers(6). Measures used in these surveys include estimates of drinking frequency, typical or average quantities of use, total volume, and measures of heavy or “binge” drinking. More recently, daily drinking measures have attracted particular interest, providing counts of drinks consumed per day using individual diaries or electronic recording procedures(7,8). There are important scientific reasons for the use of these different measures, however, concomitant with the emergence of these different measures has been debate about which are best to use(9).

When the purpose of research is to establish the social or demographic correlates of drinking, using drinking measures as dependent variables in statistical models, the selection of a particular measure may not be of great theoretical importance. As long as the measure is reliable and valid(10) and there is a coordinated understanding of how it relates to other measures in some framework(11), then the drinking measure need only reflect the interests of the researcher. However, when drinking measures are related to the incidence or prevalence of related problems, using drinking measures as independent variables in statistical models, additional theoretical issues arise. Under these circumstances drinking measures are related to drinking problems by some implicit or explicit dose-response model. Some of these dose-response relationships may be quite distal (e.g., when total volume is related to myocardial infarction) and others quite proximal to the problem under consideration. But when a thorough theoretical and empirical assessment of dose-response relationships is desired, the assessment will require, first, the determination of distributions of alcohol use at different drinking levels, second, the specification of theoretical mechanisms by which these levels of use are related to problems and third, adequate assessments of problems specifically related to alcohol use.

Interpreting Risks Related to Drinking

An important question to ask with regard to any drinking measure is “At what specific drinking level or levels do alcohol problems arise?” The answer to this question for all traditional drinking measures is that no specific levels are indicated. For example, it is often assumed that a positive correlation between average drinking quantities and drinking problems indicates that more problems arise with greater use. However, drinkers may increase average quantities of use by drinking more often at low or high drinking levels. Since a shift from drinking 1 to 2 drinks 5 times a week has the same impact on the average as a shift from drinking 4 to 5 drinks (in each case 5 drinks are added), and since problems may arise at both low and high levels, the source of greater drinking problems with increasing average levels of use is unknown. A positive correlation between average drinking levels and drinking problems is uninformative on this point.

For similar reasons, other drinking measures fail to inform our understanding of relationships between drinking and problems. Measures of drinking frequency aggregate across all drinking events, neglect the role of drinking quantities, and tell us little about the doses of alcohol which place drinkers at risk. Measures of typical amounts consumed average across drinking events and neglect distributions of use; someone who drinks 1 drink on 7 occasions and 9 drinks on 1 occasion is indistinguishable from another who consumes 2 drinks on 8 occasions. Measures of total volume are functions of drinking frequencies and quantities and inherit the problems of both. And, finally, frequencies and probabilities of “binge” or heavy drinking increase with greater frequencies and quantities of use, and problems ascribed to “binge” drinking may arise from use at lower drinking levels. A positive correlation between “binge” drinking measures and drinking risks is uninformative on this point.

The obvious first step to solving some of these problems is to collect drinking data that allow researchers to assess frequencies of drinking at varying dosage levels and relate these measures to the problems that arise. To date, several methods have been developed that allow the acquisition of suitable data: daily drinking diaries(8) and other interactive data acquisition systems(7), graduated frequency methods that distinguish days on which different amounts were consumed developed by Greenfield and colleagues in the U.S.(9) and Mäkelä and colleagues in Finland(13), and an alternative technique developed by us (11) that asks respondents to report the numbers of days on which they consumed 1 or more, 2 or more, and so on, “continued” drinks, and uses a mathematical model of drinking to

extract information about drinking frequencies at different levels of use. This technique produces reliable and valid estimates of drinking patterns(1) and the mathematical model used to approximate drinking distributions accurately represents data collected using graduated frequency methods(13).

Assessing Dose-Response

With these data in hand, a theoretical model is required that relates frequencies of use at different drinking levels to drinking problems. One representation of the problem to be solved is shown in Figure 1. To the top left is a distribution of the frequencies at which 1, 2, 3 or more drinks were consumed over the past month. To the right of the figure is a list of hypothetical risks associated with each of these drinking levels. We assume that drinking at each level leads to some, perhaps fractional, average number of problems so that whenever 3 drinks are consumed N_3 problems occur. Thus, if the drinker consumes 3 drinks on 5 occasions, then $5 \times N_3$ problems accumulate. The total number of problems related to drinking is the weighted sum of problems related to drinking at all levels. The fundamental theoretical problem is how to determine specific risks for each drinking level.

Whenever a college drinker is asked a question like, “How often in the past month have you had a hangover after drinking?” she or he will summarize all hangovers related to all levels of drinking in the past month. No information will be provided about the amount consumed on any occasion on which a hangover occurred. However, some reasonable assumptions can be made by which risks related to specific drinking levels can nevertheless be recovered. The theoretical linkages for one such model are outlined in the table at the bottom of Figure 1. The table presents observable (in **bold**) and theoretically inferred (in *italics*) aspects of drinking that lead to reports of problem rates. The observable characteristics of drinking are that drinkers consume varying amounts of alcohol (column 1), drink these amounts on different numbers of occasions (column 2), and summarize the number of problems over this time (sum of column 4). The theoretically inferred aspects of drinking are that each drinking quantity is associated with a specific risk (entries in column 3) under the condition that the sum of the cross-products of columns 2 and 3 are equal to the observable sum of column 4. Empirical constraints on plausible theoretical entries in the table are the observed distribution of drinking frequencies (column 2) and the total number of problems (sum of column 4). The theoretical challenge is to fill in column 3, the dose-response of problems to use at each specific drinking level. Gruenewald, et al.(14) showed that this could be achieved by assuming a specific form for the dose-response function (e.g., linear in column 3 of this table), then fitting the model to data given the observed constraints.

This paper applies a more thoroughly developed nonlinear model of dose-response to data from a very large survey of college drinkers in California to assess the performance of this theoretical model and to elucidate dose-response functions in this important population of drinkers. We consider all problems related to alcohol as reported on the survey, problems reported specifically by men and women, “moderate” and “serious” problems, and problems that arise in “light”, “moderate” and “heavier” drinkers. The goals of the paper are to (1) outline the theoretical model, (2) demonstrate its application to the assessment of risks related to college drinking, (3) estimate problem risks in this group and determine drinking levels at which college students are most at risk for problems, and (4) compare the statistical performance of these theoretically-derived models to those of simpler “binge” drinking models.

METHODS

The dose-response model introduced here assumes that risks for alcohol problems are (a) additive across drinking levels, (b) distributed according to a constrained set of parametric functions, and (c) may be constant or censored at low levels of drinking (e.g., risks may equal zero at low levels of alcohol use). Dose-response may be constant, censored or uncensored, monotone increasing or decreasing, accelerating or decelerating. A fuller explication of the model is available from the authors upon request. The essence of the model is summarized here in three equations.

It is assumed that in each drinking day risks depend upon the number of drinks consumed (drinking exposures, E_i). Cumulative risks for problems, R , over days, T , are a linear function of background risks (risk on non-drinking days, E_0) and risks associated with days on which $i = 1, 2, 3, \dots, n$ drinks are consumed:

$$R = \alpha_0 E_0 + \alpha_1 E_1 + \alpha_2 E_2 + \alpha_3 E_3 + \dots + \alpha_n E_n \quad (1)$$

The E_i are the numbers of days on which i drinks were consumed and α_i are risks related to drinking at each drinking level. The hypothetical drinker in Figure 1 has $E_1 = 3$ days $\alpha_1 = 0.1$ and, consequently, $\alpha_1 E_1 = 0.3$, the cumulative impact of this individual's three single-drink days on total problems, R .

The vector α_i represents the contribution of each drinking level to accumulated risks, R , over days, T ; dose-response. The elements of this vector are constrained here to a specific set of shapes, each related to a different hypothesis about the distribution of drinking risks. The simplest hypothesis is that problems occur on non-drinking days at rate $\alpha_0 = c_1$ and the use of alcohol does not contribute to these risks, $\alpha_{i>0} = c_1$. The next simplest hypothesis is that drinking contributes to these risks regardless of drinking level so that $\alpha_0 = c_1$ and $\alpha_{i>0} = c_1 + c_2$. Drinking days are more risky than non-drinking days by c_2 units. The next simplest model is a "binge" drinking model in which at a certain number of drinks, $i \geq \kappa$, a change in dose-response is observed such that $\alpha_{i \geq \kappa} = c_1 + c_2 + c_3$, with c_3 representing the added risks entailed by "binge" drinking. Thus, problems uniquely related to "binge" drinking can be distinguished from those related to other drinking levels.

More realistic models allow continuous variation in risks at various drinking levels. These models incorporate the assumption that the weights, α_i , are a function of drinking levels:

$$\alpha_i = \beta + [\delta(i - \kappa + 1)^\zeta | i \geq \kappa] \quad i = 1, 2, 3, \dots, n \quad (2)$$

Treating risks on non-drinking days, α_0 , as fixed, this function states that the additional risks related to drinking days are a function of background drinking risks β plus risks related to drinking at or beyond a specified level κ (the censoring interval). δ is the magnitude of increase at threshold κ , and ζ is a power exponent allowing for possible nonlinear dose-response. If $\zeta = 1$, risks increase linearly. If $\zeta > 1$, risks increase in an accelerated manner. If $\zeta < 1$, risks increase in a decelerated manner. It is also possible that risks remain constant over drinking levels, $\zeta = 0$ (the "binge" drinking model), or decrease over levels conditional upon the values of β and δ .

Given equations (1) and (2), total risks reported over a given time period are:

$$R = \alpha_0 T + \sum \alpha_i E_i \quad i = 1, 2, 3, \dots, n \quad (3)$$

Accumulated risks, R , over time, T , are a function of risks on non-drinking, $\alpha_0 T$, and drinking days, $\sum \alpha_i E_i$. Given estimates of drinking exposures, E_i , and problems related to these exposures, R , then dose-response functions from drinking data can be inferred by fitting equation (3) to these data.

Data Sources

Data were collected from a stratified random sample of undergraduate students at 14 California public university and college campuses in fall, 2003. A web-based survey collected demographic information, measures of drinking patterns, and alcohol-related problems caused by the students' own drinking. Of 28,157 students invited to participate, surveys were collected for 14,072, representing a 50% response rate. The analyses reported below exclude abstainers as well as respondents with incomplete or inconsistent data, resulting in a final sample of 8,698 drinkers. Post-stratification weights were used in all statistical analyses, adjusting for campus size, gender and racial composition (White, African American, Asian, and Hispanic). Drinking patterns were assessed over the past 28 days or, for low frequency drinkers, since the beginning of the school semester. These items asked respondents to indicate the number of occasions on which they consumed 1 or more, 2 or more, 3 or more, 6 or more, and 9 or more drinks of alcohol. Answers to these questions were used to estimate numbers of drinking occasions at each drinking level during the previous 28 days using a suitable mathematical model of drinking patterns(11). Replicating previous work, the average R^2 of model fits to these drinking data was 0.98, reflecting the high validity and reliability of this procedure (1). A drink was defined as a 12-ounce glass or bottle of beer, a 5-ounce glass of wine, or a 1-ounce shot of spirits.

Three problem measures were created from a standard set of 29 drinking problems reported by each college drinker. This instrument provided an overall assessment of drinking problems commonly encountered by college drinkers and was constructed to parallel standard instruments that have been used for the past two decades.(5) For the purpose of developing and testing the proposed dose-response model it was felt the use of a standardized instrument of this sort would adequately approximate an overall assessment of risk and, suitably, not depart from the kinds of risks often associated with binge drinking. The 29 problems were from a number of domains presumably affected by alcohol: physiological (e.g., hangovers, forgetting); school related (e.g., missing a class, getting behind in school work); psychological (e.g., doing something you later regretted); behavioral (e.g., driving a car after drinking or while intoxicated); interpersonal (e.g., pressuring or forcing someone to have sex); medical (e.g., getting hurt or injured); legal (e.g., getting into trouble with campus authorities or police). Students were asked to report the number of times each of these problems had occurred between the survey date and the start of the current semester or quarter. These reports were converted to problem rates per 28 days and the overall rate of drinking problems was the sum of these 29 specific indicators. Two additional measures subdivided this set of items into problem groups representing moderate (hangovers, missing class, getting behind in school work, arguing with friends, and vomiting) and serious (requiring medical treatment for an alcohol overdose, passing out, blackouts, physical fights, and pressuring or forcing someone to have sex) problems.

Finally, the population of college drinkers was partitioned into "light," "moderate," and "heavier" drinkers using criteria established by the National Institute on Alcohol Abuse and Alcoholism. "Light" drinkers were those who consumed on average 3 or fewer drinks per week (54.0% of drinkers in this sample), "moderate" drinkers were those who consumed more than 3 drinks per week but less than 7 drinks per week for women or 14 drinks per week for men (28.5%), and "heavier" drinkers were those who averaged higher counts of weekly drinks (17.5%).

Analysis Procedures

Censored regression models were employed to estimate the parameters of different dose-response models related to respondents' drinking problems. Since accumulated problems were expressed as rates over 28 days (an interval scale with non-negative fractional values), censored regression models, TOBITs, were required for unbiased analysis of dose-response relationships(15). The TOBIT regressions were corrected for heteroskedasticity related to frequencies of use and dosage level(16). A sequence of TOBIT models was executed for all problems and groups examined: (1) The constant risks model without censoring assumed that problem risks were related to drinking but did not increase with dosage level. (2) The "binge" model with censoring assumed constant risks related to drinking, but that these risks increased in a stepwise manner at some threshold; the "binge" drinking criterion. (3) The linear model without censoring assumed constant risks related to drinking and a linear increase in risks beyond the first drink, replicating Gruenewald, et al.(14). (4) The nonlinear model without censoring assumed constant risks related to drinking and a nonlinear increase in drinking risks beyond the first drink. (5) The nonlinear model with censoring assumed constant risks related to drinking, but that these risks increased in a nonlinear manner starting at some criterion drinking level κ , referred to as the censoring interval for dose-response effects.

Poisson and negative binomial models were considered as alternative statistical approaches to analyses of these count data. They were not used because respondents reported counts of problems since the beginning of the current semester, and conversion to a common 28-day metric resulted in fractional problem rates. In addition, current software implementations of these models do not generally provide sufficient analytic flexibility to account for the several sources of heteroskedasticity we expected to encounter in these analyses. Our previous work(1,14) indicated that heteroskedasticity increases as a function of background drinking risks (reflected in drinking frequencies) and dosage levels (related to average quantities). Independent of the impacts of different distribution theory upon effects estimates, the results of dose-response analyses should otherwise be indifferent to these statistical issues.

Model estimation proceeded sequentially. Model 1 was directly estimated by including a constant term and frequency of drinking among the independent measures. Model 2 required the addition of a parameter representing the censoring interval, κ . The value for the censoring interval was estimated using a simplex search through plausible integer values of κ from 1 drink (no censoring) through 12 drinks, iterating through successive TOBIT models with different scoring functions. Since no censoring was assumed, Model 3 could be directly estimated by including constant, frequency, and the difference between total volume and frequency estimates among the independent measures(17). Model 4 added a term for nonlinear dose-response, ζ , estimated using a simplex search through plausible values from 0.01 to 2.0 with a resolution of 0.01 units. "Best" models were those that minimized Rao's likelihood ratio chi-square statistics. Two-tailed 95% confidence bounds for ζ were estimated by comparing likelihood ratio chi-square values along this dimension of the parameter space(18). Model 5 added a test for censoring to the nonlinear model (see Model 2). Confidence intervals for κ were all less than ± 1.0 unit indicating strong discontinuities in risks at these points in the parameter space. (In practice, since only integer values of κ could be considered, confidence bounds for κ cannot be given.) Finally, examinations of fit statistics across the parameter spaces for κ and ζ from Models 2, 4 and 5 demonstrated single global minimums for these parameter estimates.

These fit procedures do not provide direct methods by which standard errors of dose-response estimates can be assessed. For this purpose, parametric bootstrap estimates were

generated (10,000 iterations) using parameters and standard errors directly estimated from the models using the procedures given here(19).

RESULTS

Table 1 presents the statistical performance of the five TOBIT models used to assess risks related to all drinking problems reported by college drinkers. The left portion of the table presents the model number and name, estimated pseudo- R^2 value(20), likelihood ratio chi-square statistic, G^2 , for the best fitting model, and associated degrees of freedom, df. The right portion of the table presents statistical comparisons between different models using likelihood-ratio statistics. Even the most simple dose-response model, the constant model without censoring, correlated well with rates of problem outcomes in college students, as evidenced by the pseudo- R^2 of 0.493. This observation shows that a measure of overall exposure to alcohol use correlates well with drinking problems since drinking frequencies are correlated with exposures to use at all levels. The model comparisons to the right of the table provide further clarification of this relationship by demonstrating substantive dose-response effects related to levels of use. For example, the “binge” drinking model (Model 2), with an estimated threshold $\kappa = 3$, provided the best fit within this class of models showing that heavier drinking contributed disproportionately to problem rates. As shown in column 1 to the right of the table, Models 3 through 5 provided significantly better fits than Model 1. The performances of Models 3 and 4 relative to the “binge” drinking model (Model 2) cannot be directly compared (the models are not hierarchically nested), but both provided substantively better fits to the problem data than Model 1 (G^2 improvements of 510.6 and 606.8 units, respectively). Nonlinear Model 5 with censoring at $\kappa = 3$ accounted for the largest proportion of problem-outcome variance of all models tested.

The top portion of Table 2 presents TOBIT parameter estimates with their 95% confidence bounds for the five models presented in Table 1. Reading from left-to-right the table includes parameter estimates for drinking risks, β , the value of δ for the “binge” drinking model (assuming $\zeta = 0.00$), and the slope, δ , and exponent, ζ , of the linear (assuming $\zeta = 1.00$), and nonlinear (allowing $\zeta \neq 1.00$) dose-response functions (Models 3, 4 and 5). The value of β represents risks associated with drinking on any day on which one or more drinks was consumed. These risks were significantly greater than zero for all but Model 4, indicating that some risks for problems attended all drinking levels. The value of κ represents the value of the censoring interval for “binge” drinking, estimated to be equal to 3 drinks in Models 2 and 5. The associated impact of drinking 3 or more drinks on any occasion is captured by the value of δ . There is an abrupt increase in drinking risks at three drinks; about a 5-fold increase over background risks in both analyses. The value of δ in Models 3, 4, and 5 represents the slope of the dose-response function; it is positive and significant for all models in which it was included. Finally, the value of ζ represents the curvature of the dose-response function; equal by definition to 1.00 in Model 3 and greater than 0.00 but less than 1.00 in both Models 4 and 5, indicating decelerating dose-response over greater numbers of drinks.

All models included a correction for heteroskedasticity related to drinking frequency (background drinking risks). Model 2 included an additional heteroskedasticity control related to heavy drinking frequencies (defined by the “binge” criterion), while Models 3, 4 and 5 included terms related to linear or nonlinear dose-response. Heteroskedasticity was positive and significant for all control variables in these models, reflecting increases in the variance of problem responses over greater drinking frequency and dosage.

The bottom sections of Table 2 present parameter estimates from separate analyses for men and women, “moderate” and “serious” problem groups, and for “light,” “moderate” and

“heavier” drinkers, each estimated using the nonlinear model that provided best fit. These separate analyses showed that background drinking risks were often significantly positive and all nonlinear dose-response functions were decelerating. Models for women and light drinkers were uncensored ($\kappa = 2$), while heavier drinkers had the highest censoring threshold ($\kappa = 4$). In one interesting case the dose-response function for “light” drinkers was linearly increasing.

Figure 2 presents the dose-response functions for all students (top left, based on results from Model 5) and men and women separately (top right, based on Models 6 and 7). Dose-response for the total sample was significantly greater than zero at low levels of drinking, increased at three drinks and rose in a decelerating manner thereafter. Dose-response functions for men and women were overall quite similar, although men show less dose-response at the second drink and women show somewhat more dose-response at higher levels (9 or more). The middle graph in each set shows the observed distributions of drinking levels in the total population (left) and men vs. women (right). Women drank somewhat less frequently than men at every drinking level, particularly from 4 to 9 drinks. The dose-response model allows us to multiply these average frequencies at various drinking levels by estimated dose-response to obtain estimates of population risks related to drinking at each level, shown at the bottom of the figure. Across all college students it is evident that drinking problems were associated with every drinking level but were most-closely linked to days consuming 3 drinks (the mode). The bulk of college drinking problems were associated with consumption of 3 to 6 drinks of alcohol. Male and female risks for alcohol problems diverged considerably, with women showing more risks at 2 drinks, both genders exhibiting peak drinking risks again at 3 drinks, and risks for men exceeding those of women at higher drinking levels.

The left side of Figure 3 displays the dose-response functions for the entire sample but separately considers “moderate” and “serious” problems as defined above. The right side of the table looks at total problems for subgroups of college students characterized as “light” vs. “moderate” vs. “heavier” drinkers. The plots for “moderate” and “serious” problems both exhibited a discontinuity in dose-response at 3 or more drinks. “Serious” problems, however, were unrelated to drinking at low levels, 1 or 2 drinks, and were more dose-responsive than “moderate” problems at higher drinking levels. Combined with information about the distribution of drinking levels, middle graph, it is also evident that “moderate” problems were more associated with drinking at low levels and “serious” problems somewhat more associated with drinking at higher levels. However, the similarities between these functions are more striking than their differences.

The plots for “light,” “moderate” and “heavier” drinkers presented to the right of the figure demonstrate the power of the current technique to distinguish relationships of drinking to problems in these groups. As shown in the upper graph, dose-response was very different for these drinkers. Dose-response for “light” drinkers was linearly increasing from the first drink and greater than that for “moderate” and “heavier” drinkers across all drinking levels. “Moderate” and “heavier” drinker dose-response functions started off at about the same level, but risks increased for “moderate” drinkers at 3 drinks and for “heavier” drinkers at 4 drinks. Risks increased thereafter with little difference between these two dose-response functions. As shown by the middle graph, the drinking patterns of these three groups were, of course, very different. Very little “heavy” drinking (i.e., greater than 4 drinks) took place among “light” drinkers, but considerable “light” and “heavy” drinking took place among both “moderate” and “heavier” drinkers. Population risks related to drinking levels were also very different between these groups, reflecting differences in drinking patterns and dose-response. Although “moderate” and “heavier” drinkers exhibited similar dose-response at

higher drinking levels (top graph), “heavier” drinkers consumed at high levels much more often and exhibited many more problems (2 to 10 times greater at 4 or more drinks).

DISCUSSION

The results of this study demonstrate that theoretical dose-response models can provide sound bases for quantitative assessments of drinking risks (Figure 1). These models have three unique features. First, they assume that risks are additive across drinking events. Second, they treat numbers of occasions on which different quantities of alcohol are consumed as separate drinking exposures. Third, they model dose-response using a flexible set of analytic functions. The major advantage of this approach is that a large variety of dose-response relationships can be represented and statistically evaluated while also providing a framework for understanding empirical correlations between commonly used drinking measures and related problems. Another advantage is that quantitative analyses based on these models provide comprehensive assessments of distributions of risks related to drinking. The model departs from the usual theoretical standards for psychological and sociological explanations of drinking patterns and problems to focus upon the quantitative machinery that links drinking exposures to incidence and prevalence of harms related to drinking in human populations.

The empirical work presented here demonstrates that these dose-response models can provide informative assessments of relationships between drinking patterns and problems. These studies showed that moderate levels of drinking (2 – 5 drinks) were most strongly related to drinking problems among college students. Censored nonlinear dose-response models best fit the data for all college students (Table 1), with censored dose-response below 3 drinks, a large increase at that point, and increasing but decelerating risks thereafter (Figure 2). Women’s and men’s drinking risks were very similar, with women’s risks more dose-responsive at low levels of alcohol use (Figure 2). Men’s higher accumulated rates of problems above 3 drinks resulted less from dose-response differences than from their greater propensity to drink at higher levels despite these risks. These statistical analyses of alcohol measures also indicate decelerating increases in levels of harm over greater average drinking quantities.⁽⁹⁾ But, uniquely, the current models provide an analytic expression of risks related to individual rather than aggregate drinking levels.

Estimates of dose-response for both moderate and serious problems were quite low at 1 – 2 drinks and increased considerably at 3 drinks (Figure 3). However, serious problems were more dose-responsive at higher drinking levels. This observation might suggest that heavier drinking is related to impairments in judgment that lead to more serious problems⁽²¹⁾, or that the contexts of heavier drinking (e.g., bars, parties) may place drinkers at greater risks for these outcomes⁽²²⁾. Since the same drinkers were included in analyses of moderate and serious problems, differences in population risks are solely attributable to differences in dose-response.

Finally, estimates of dose-response for different drinker classes were, as one would expect, very different (Figure 3). “Light” drinkers were very dose-responsive, demonstrating linear increasing risks from the first drink on. In agreement with behavioral economic models of drinking⁽²³⁾, the consumption of “light” drinkers appeared to be problem limited, restricting their drinking to quantities at which few problems were reported. Also in agreement with behavioral economic models, “moderate” and “heavier” drinkers were less dose-responsive, and thus drank more frequently at higher drinking levels. Although dose-response for “moderate” and “heavier” drinkers was very similar at these levels, “heavier” drinkers consumed 4 or more drinks far more frequently and exhibited 5.7 times as many total problems from these high-drink days. This observation suggests a pattern of shifting dose-

response and negative consequences of use expected from neurocognitive models of addiction; a shift from “impulsive” to “compulsive” use unconstrained by negative outcomes, a pattern of use also characteristic of alcohol dependence(24,25).

This study demonstrates how one specific theoretical dose-response model can lead to innovative empirical analyses of drinking patterns and problems. Applications of the current model have two advantages over purely statistical analyses of these data. First, it simultaneously relates reported problems to drinking at many different levels (here 1 through 12 drinks). Simpler models that relate problems to the number of heavy drinking occasions (e.g., above a 5-drink binge threshold) will be biased to the extent that heavy drinking days are correlated with lighter drinking days. Simplifying assumptions about the shape of the dose-response function allows for parsimonious descriptions of the relationship between drinking and problems. Second, the assumption of additive risks across occasions makes it possible to combine occasion-specific dose-response with population-level drinking propensities to estimate the proportion of all reported problems that are due to drinking at various levels. Prior clinical and experimental studies have demonstrated that individual drinkers experience many more problems at higher ethanol blood alcohol concentrations(24), but such analyses do not account for the lower frequency at which drinkers consume to high drink levels. Two factors underlie the current study’s contrasting finding that population-aggregated drinking problems decline at high drinking levels (Figure 3, bottom). First, the decelerating population dose-response function indicates that problems per occasion rise less quickly at higher drinking levels, as might be expected if heavier drinkers tend to be those individuals who are less sensitive to the effects of alcohol. Second, and more importantly, very heavy-drinking occasions are relatively rare, reducing the population-level impact of such occasions.

The current study provides the only available method by which survey data may be used to control for low level risks in assessments of problems typically associated with binge drinking. Figure 4 compares results from Models 1 through 5 explaining total problems from the full drinker sample. Despite considerable model differences in dose-response (top panel), all models found that population risks peaked well below binge drinking levels (bottom panel).. Peak aggregate risks occurred at 2 drinks in Model 1 (constant) and at 3 drinks for all other models. Extensive re-specifications of these models using different censoring intervals for Models 2 and 5 failed to improve fit to these data and only those models which performed very poorly shifted apparent peak drinking risks away from the 3-drink level.

The Importance of “Binge” Drinking

The dose-response analyses of drinking in this college population might be interpreted to suggest unique risks related to “binge” drinking. Looking at the college drinking population as a whole, low risks were exhibited at low drinking levels (0.2 problems per drinking day at 1 and 2 drinks), there was a 5-fold increase at 3 drinks (to 1.0 problems per day), and a gradual progression in risks over higher drinking levels (to 1.7 problems per day at 12 drinks). Similar threshold-like increases occurred at 2 drinks among women and 3 drinks among men, with each of these “binge” criteria being 2 drinks below those assumed in the college drinking literature (i.e., 4 or 5 drinks for women or men, respectively). Although drinking at high drinking levels obviously increases risks for alcohol problems among college students, the formal analytic framework presented here identifies optimum “binge” drinking criteria clearly below those often used to identify problematic college drinking. Using the best fitting model and accumulating problems across drinking levels 1 through 12 in Figure 2, only 47% of drinking problems were related to days with 5 or more drinks. This suggests that a very substantial number of problems occur at non-“binge” drinking levels.

Implications for Prevention Policy

From a public health perspective, two recommendations based on the findings of the current study could be made. First, it is important that those groups and individuals who can shape alcohol policy have the benefit of complete and accurate information about college drinking risks. These risks are often attributed to “binge” drinking. However, substantial risks appear to arise at lower drinking levels. Therefore, the advantages of reducing “binge” drinking among college students may be quite modest, especially if reductions in “binge” drinking are offset by shifts to moderate levels at which problems still occur. Second, health education messages about drinking should be realistic if they are intended to inform students about drinking risks. The focus upon “binge” drinking may distract students from recognizing the most frequent source of alcohol problems in college, drinking at relatively moderate levels.

Limitations

This paper investigates just one possible implementation of dose-response models. One limitation discovered with the current formulation is that the results were highly sensitive to specification of censoring points. It is possible that two censoring thresholds (e.g., $\kappa = 3$ and $\kappa = 4$) could provide very similar fit statistics yet substantially different plots of the dose-response function. This might be avoided with the use of continuous logistic and asymmetric logistic functions in dose-response models.

Another area for improvement is distinguishing between types of risks. The survey used in this study asked only about problems that the respondent attributed to his or her own drinking. For many problems, like arguments with friends, it is essential to distinguish background non-drinking problem risks from background drinking risks, which requires suitable survey items.

A third area for improvement in these models regards the introduction of contextual effects into model estimates of dose-response. Considerable research demonstrates that drinking patterns are associated with the use of different drinking contexts.(11,22,26) For example, if heavy drinkers are more likely to drink in bars, then the dose-responsiveness of problems associated with drinking at bars such as aggression and violence will reflect this. Ideally, an extended model would segregate context effects on problems from those directly related to drinking levels.

A fourth area for improvement will be to discern separate dose-response effects for different domains of drinking problems. Earlier work using a simple linear model suggests great differences in dose-response across different problems (e.g., arguments with friends, unplanned sexual activity), with risk distributions for serious problems tending to be shifted to the right(14). Individual problem categories might have considerably different risk profiles than were estimated in the current study for aggregated groups of problems. This might occur because many risks related to drinking are avoidable, such as those associated with operating heavy machinery. Although heavy equipment use might be extremely dangerous after many drinks, the associated population risks may be quite low if alcohol impaired individuals avoid such activities.

Finally, observed effects sizes will be affected by self-report biases,(27,9). To the extent that such biases can be quantified, the current modeling approach enables corrected estimation of dose-response functions using a variety of statistical approaches(28) (e.g., Bayesian models with corrections for measurement error; Congdon, 2001). Exploration of these biases is an essential task for alcohol epidemiology.

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Figure 1. An example of a distribution of drinking levels (left), associated drinking problems (right), and a table for computing dose-response and drinking risks (bottom).

Figure 2. Dose-response functions (top), drinking exposures by drinking level (middle), and drinking risk distributions (bottom) for the full sample (left) and men versus women separately (right).

Figure 3. Dose-response functions (top), drinking exposures by drinking level (middle), and drinking risk distributions (bottom) by seriousness of problem (left) and drinker type (right).

Figure 4.
Dose-response functions (top) and drinking risk distributions (bottom) for Models 1 through 5.

Table 1

Model selection for dose-response analysis of total problems

Model #	Reference Model Description	Pseudo-R ²	Model G ²	Model d.f.	Model Comparisons:			
					1	2	3	4
1	Constant model without censoring	0.493	40,986.22	2	ΔG^2 p-value			
2	"Binge" model with censoring, $\kappa = 3^*$	0.559	39,778.60	3		1,207.6 0.000		
3	Linear model without censoring	0.584	39,267.98	3	ΔG^2 p-value	1,718.2 0.000	<i>Models not comparable</i>	
4	Non-linear model without censoring	0.589	39,171.84	4		1,814.4 0.000	96.1 0.000	
5	Non-linear model with censoring, $\kappa = 3^*$	0.590	39,139.02	5	ΔG^2 p-value	1,847.2 0.000	639.6 0.000	32.8 0.000

* Separate analyses were performed with censoring at levels from $\kappa = 2$ to $\kappa = 12$; the best fit was achieved with $\kappa = 3$ as shown.

Table 2

TOBIT Coefficients for Reference Models

Model #		Drinking Risks (f)	"Binge" Risk (g)	Dose-Response Function Slope (h)	Dose-Response Function Exponent (i)
A) Models based on all problems in the full sample:					
1	Constant Model without Censoring (95% Confidence Bounds)	0.786 (0.753, 0.819)	-	-	-
2	Binge Model with Censoring, $\kappa = 3$ (95% Confidence Bounds)	0.153 (0.118, 0.188)	0.921 (0.872, 0.970)	-	-
3	Linear Model without Censoring (95% Confidence Bounds)	0.157 (0.121, 0.192)	-	0.203 (0.193, 0.214)	1.000 (by definition)
4	Nonlinear Model without Censoring (95% Confidence Bounds)	0.034 (-0.007, 0.075)	-	0.407 (0.385, 0.428)	0.644 (0.574, 0.723)
5	Nonlinear Model with Censoring, $\kappa = 3$ (95% Confidence Bounds)	0.183 (0.150, 0.217)	-	0.618 (0.585, 0.650)	0.366 (0.287, 0.436)
B) Best-fitting models for subsets of either population or problems types:					
6	MEN ONLY: Nonlinear Model with Censoring, $\kappa = 3$ (95% Confidence Bounds)	0.179 (0.122, 0.237)	-	0.498 (0.457, 0.539)	0.485 (0.366, 0.614)
7	WOMEN ONLY: Nonlinear Model without Censoring, $\kappa = 2$ (95% Confidence Bounds)	-0.006 (-0.054, 0.042)	-	0.492 (0.459, 0.524)	0.594 (0.515, 0.683)
8	MODERATE PROBLEMS ONLY: Nonlinear Model with Censoring, $\kappa = 3$ (95% Confidence Bounds)	0.035 (0.027, 0.042)	-	0.149 (0.140, 0.158)	0.248 (0.168, 0.317)
9	SERIOUS PROBLEMS ONLY: Nonlinear Model with Censoring, $\kappa = 3$ (95% Confidence Bounds)	-0.004 (-0.018, 0.010)	-	0.154 (0.143, 0.164)	0.456 (0.357, 0.555)
10	LIGHT DRINKERS ONLY: Linear Model without Censoring, $\kappa = 2$ (95% Confidence Bounds)	0.118 (0.050, 0.185)	-	0.504 (0.449, 0.559)	1.000 (0.842, 1.158)
11	MODERATE DRINKERS ONLY: Nonlinear Model with Censoring, $\kappa = 3$ (95% Confidence Bounds)	0.084 (0.023, 0.144)	-	0.295 (0.247, 0.343)	0.753 (0.555, 0.970)

Model #		Drinking Risks (β)	"Binge" Risk (δ)	Dose-Response Function Slope (δ)	Dose-Response Function Exponent (ζ)
12	HEAVIER DRINKERS ONLY: Nonlinear Model with Censoring, $\kappa = 4$ (95% Confidence Bounds)	0.124 (0.041, 0.207)	- -	0.429 (0.363, 0.495)	0.713 (0.528, 0.960)