

## The differential effects of alcohol consumption and dependence on adverse alcohol-related consequences: implications for the workforce

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### Abstract:

Previous literature has supported the hypothesis that high rates of alcohol consumption are associated with adverse social consequences and that dependence on alcohol has an effect on that relationship. The purpose of this paper is to further specify the alcohol consumption-adverse consequences linkage by developing and estimating a latent variable model that incorporates the mediating effects of loss of control over alcohol consumption. This model is applied to measures for three alcohol-related constructs—consumption, loss of control and adverse consequences—incorporated in the 1991 National Household Survey on Drug Abuse, for members of the primary workforce in the US. The research suggests that workplace decision makers attempting to minimize the adverse workplace consequences of alcohol abuse should implement procedures that assess and respond to alcohol dependency rather than relying exclusively on detection of and intervention with alcohol consumption per se.

**Keywords:** alcohol | dependence | consequences | bi-axial | structural equation modeling

### Article:

#### 1. Introduction

A substantial body of research has related alcohol use to its social consequences (Edwards et al., 1977 and Clark and Hilton, 1991). In clinical research and treatment practice, adverse alcohol-related consequences have been routinely linked to the diagnostic process to such a degree that the consequences often verify the alcoholism diagnosis. Large-scale epidemiologic studies have also routinely examined the relationship between self-reported alcohol consumption patterns and problems at home, school and work (Clark and Hilton, 1991). Both clinical and epidemiologic studies confirm the popularly held notion that inappropriate consumption, i.e. excessive episodic or chronic abuse of alcohol, causes problems for abusers and others affected by their behaviors.

As a result, family, educational, military, treatment, criminal justice and other social institutions are expected to engage in preventative and interventionist control activities to mitigate the negative consequences of alcohol abuse. The purpose of this paper is to increase the understanding of the relationship between alcohol consumption and adverse consequences. This was attempted by developing and estimating a latent variable model of the relationship between consumption and consequences that incorporates the mediating effect of loss of control, a dimension of alcohol dependency, over alcohol consumption. This model was then applied to data selected from the 1991 National Household Survey on Drug Abuse (NHSDA). Due to the vast economic and safety costs associated with alcohol abuse (Cahalan and Room, 1974; Polich and Kaelber, 1985; Rice et al., 1990; Clark and Hilton, 1991), there is particular interest in this behavior among members of the workforce in the US, although it was felt that the findings presented here are generalizable in some degree to other populations.

## **2. Literature**

### **2.1. The relationship between consumption and adverse consequences**

As noted by Drummond (1992), the adverse consequences associated with alcohol consumption have been recognized in theories of alcohol addiction, at least since the publication of Benjamin Rush's enquiry into the Effects of Spiritous Liquors on the Human Body in 1785. The Temperance Movement was energized by a growing public awareness of the adverse consequences associated with alcohol abuse (Gusfield, 1963). Some movement members advocated curbing alcohol use and promoting moralistic behaviors in the workforce during the 19th and early 20th centuries (Trice and Schonbrunn, 1981, Levine, 1984 and Staudenmeier, 1987). More recently, the moralistic assumptions of the temperance orientation have been superseded by the disease concept of alcoholism (Jellinek, 1960 and Conrad and Schneider, 1992), which stresses physiological dependence on alcohol as the paramount problem resulting from alcohol abuse. Subsequent empirical research has confirmed the conceptual distinction between abuse and dependence (Grant et al., 1992; Muthen et al., 1993).

With their bi-axial concept, Edwards et al. (1977) have conceptually distinguished between behaviors associated with physiological dependence upon and the adverse consequences resulting from alcohol abuse. They suggest that both dependence and adverse consequences (which they refer to as 'problems') vary on continua of severity and that alcohol users could experience dependence and problems of differing levels of severity at some point in time. For example, one could manifest significant indications of alcohol dependence but not experience adverse consequences, possibly due to situational buffers and supports. On the otherhand, individuals could experience significant problems related to alcohol misuse but not indicate physiological dependence, as in the case of situational abusers experiencing workplace or vehicular accidents.

The bi-axial model, widely adopted in the research literature, adds the construct of dependence to models of the relationship between consumption and adverse consequences. Since the introduction of this perspective, researchers have attempted to discern the relationships between dependence and consumption and between dependence and adverse consequences (Edwards, 1986). In general, dependence has been found to be highly associated with measures of both

consumption and adverse consequences (Orford and Edwards, 1977, Hodgson et al., 1979, Rankin et al., 1982, Vaillant et al., 1982, Edwards, 1986, Jaffe and Ciraulo, 1986, Drummond, 1990 and Hoek Kua, 1995). Since the positive association between consumption and adverse consequences has been accepted for some time, it can be said that these three constructs are all believed to be highly associated with each other.

Further explication of the relationship among these three constructs also involves predicting their causal linkage. While this relationship is difficult to discern outside of controlled longitudinal experimental studies, 'it is intuitively much less likely that problems should directly influence dependence' (Drummond, 1992 and Drummond, 1992: 71). Correlational analyses undertaken by Drummond (1990), Drummond (1992) and Williams and Drummond (1994) suggest that dependence serves as a powerful mediating variable on the relationship between consumption and adverse consequences.

## **2.2. Research on work-related consequences of alcohol abuse**

The research literature indicates that alcohol consumption can produce adverse consequences related to work. High consumption has been linked to absenteeism (Crouch et al., 1989, Normand and Salyards, 1989, Sheridan and Winkler, 1989 and Zwerling et al., 1990); accidents (Fell, 1982, Klein, 1986 and Crouch et al., 1989; Moody et al. 1990); turnover (McDaniel, 1988, Newcomb, 1988, White et al., 1988 and Zwerling et al., 1990); dissatisfaction with the job (Mangione and Quinn, 1975 and Perone et al., 1979); vandalism (Newcomb, 1988); lower scores on technical performance, productivity, self-direction and interpersonal relations (Emrick, 1975, Jones and Vishi, 1979, McClellan, 1982 and Holder, 1987); higher scores on conflict avoidance (Blum et al., 1993); and higher health care costs (Swint and Lairson, 1984 and Drummond, 1992; Holder et al., 1992; Lennox et al., 1995). However, no studies applying the bi-axial model or any other approach to distinguish between consumption and dependence constructs and to determine their effect on adverse work-related consequences have been published.

## **2.3. Methodological characteristics of earlier studies**

Exploration of the effect of alcohol dependency on the consumption-adverse consequences relationship has been conducted in clinical settings using correlational analyses. These settings and analyses create two possible limitations in generating knowledge about the fundamental relationship between these constructs. The first limitation results from the use of clinical populations. For example, Drummond (1990), Drummond (1992) and Hoek Kua (1995) collected usable data from 103 British patients and 72 Chinese patients, respectively with the Severity of Alcohol Dependence Questionnaire (SADQ) and the Alcohol Problems Questionnaire (APQ). The findings from these studies are highly relevant for planning treatment policies for clients with drinking and social histories similar to those of the patients in these clinical settings. However, they may be of less value in understanding the nature and magnitude of the relationships among consumption, dependence and adverse consequences in general non-clinical populations. From an empirical perspective, clinically generated conclusions about the relationships among these constructs could be difficult to generalize to other populations because the range of variation in consumption, dependence and problems measures is limited among research participants who, by definition, manifest these behaviors and characteristics at a severe

enough level to warrant their inclusion in a clinical population. To more broadly understand these relationships, studying a representative sample of a non-clinical population should produce a wider range of variation in measures of consumption, dependence and adverse consequences constructs, since occasional- and non-drinkers are more likely to be represented in such a sample. Therefore, one reason for conducting the current study was to determine earlier clinical research's adequacy in describing the relationship between consumption, dependence and adverse consequences in non-clinical populations.

The second possible limitation of earlier studies is that they have used analytical procedures of limited utility to determine the direction and strength of the relationships between the constructs. As discussed in detail in an earlier paper (Lennox et al., 1996), clinical research using self-reported measures of alcohol consumption, dependence and adverse consequences traditionally relies on correlational techniques that assume perfect measurement or that only tacitly remove measurement error based on classical psychometric theory. While careful administration of standardized and validated instruments in clinical settings can reduce this problem to some degree, this control is much more difficult to maintain in non-clinical research. On the other hand, latent variable modeling allows for formal specification of the measurement models that parcel out measurement error without removing them from the analysis. Further, latent variable models produce measurement models that allow for testable hypotheses concerning the manner in which multiple indicators of a single construct relate. By applying this approach in the current study and comparing the results to earlier correlational analyses, one is able to gain at least a preliminary assessment of the relative value of the latent variable approach in understanding the relationships among consumption, dependence and adverse consequences.

### **3. Method**

#### **3.1. Subjects**

The data used were from the 1991 NHSDA to investigate the relationships among measures of consumption, dependence and adverse consequences for alcohol consumers who were members of the labor force. The labor force is defined as those who are not in school and are likely to be past traditional high-school age (older than 17 years) but before frequent retirement age (younger than 65 years). To represent the population at risk for adverse alcohol-related consequences of current alcohol consumption, all respondents who drank alcohol in the past 30 days were included. The sample excludes all military and institutionalized individuals. The sample included 8755 respondents who are active or potentially active in the labor market<sup>1</sup>. Their demographic characteristics and alcohol consumption patterns are represented in Table 1.

Table 1  
 Characteristics of NHSDA respondents used to model the effects of alcohol consumption and loss of control on adverse alcohol-related consequences

Variable	Mean	S.D.	Min	Max
<b>Demographics</b>				
Age (years)	38.49	11.48	18	64
White (%)	81.74	38.64		
Black (%)	9.07	28.72		
Hispanic (%)	7.15	25.76		
Other race (%)	2.05	14.15		
Married (%)	65.26	47.62		
Female (%)	45.13	49.77		
Less than 12 years of education (%)	14.53	35.24		
12 years of education (%)	35.82	47.95		
13 to 15 years of education (%)	21.71	41.23		
16 or more years of education (%)	27.94	44.87		
Currently employed (%)	79.61	40.29		
<b>Alcohol Consumption</b>				
Number of days drank alcohol in past 30 days	7.61	8.02	1	31
Usual number of drinks per day in past 30 days	3.03	3.06	1	66
Number of days drank 5 or more drinks in past 30 days	1.74	4.50	0	30
Most number of drinks on any one day in past 30 days	4.50	4.48	1	72

### 3.2. Measures

Grounding the investigation in the dependence perspective of Edwards and Gross, 1976, Drummond, 1990, Clark and Hilton, 1991 and Drummond, 1992 and Williams and Drummond (1994), items were selected from the NHSDA that were conceptually consistent with consumption, dependence and adverse consequences constructs. It is noted that terminological distinctions and inconsistencies exist in this literature. Clark and Hilton refer to ‘alcohol abuse’, ‘alcohol dependence’ and ‘adverse alcohol-related consequences’. Drummond and Edwards et al. refer to ‘consumption’, ‘dependence’ and ‘problems.’ For purposes of this study and for reasons discussed later in this paper, the related constructs have been nominally identified as ‘consumption’, ‘loss of control’ and ‘adverse alcohol-related consequences’.

The items selected are represented in Fig. 1. A total of two items were selected indicative of quantity of alcohol ingested (‘Q’ items) and two items indicative of frequency of consumption (‘F’ items) from the NHSDA for the consumption construct<sup>2</sup>.

The items selected for the loss of control construct, indicated as ‘L’ items, measure physical and behavioral symptoms associated with loss of control. These items reflect negative drinking episode behaviors, as well as adverse alcohol-related physiological and cognitive consequences that are borne by the drinker and are more likely to be reoccurring and less transient in nature than distinctive adverse consequence events (Edwards and Gross, 1976; Room, 1977). Others who have attempted to operationalize this construct have used more multidimensional measures of the alcohol dependence syndrome described by Edwards and Gross (1976). For example, the SADQ assesses physical withdrawal symptoms, affective symptoms of withdrawal, craving and relief drinking, daily consumption and reinstatement of symptoms following abstinence. The construct is restricted by the availability of items for analysis in the NHSDA. Therefore, the dependence items are best described as indicators of loss of control, a significant dimension of alcohol dependence. This construct is not meant to mirror DSM criteria or to imply any kind of clinical

diagnosis. Rather, the indicators more closely reflect the chronic loss of control often used in the literature to indicate dependence (Mann, 1950, Jellinek, 1960, Cahalan and Room, 1974 and Fingarette, 1988).

#### **Alcohol Consumption**

##### ***Quantity***

- Q1. About how many drinks did you usually have in a day on the days that you drank during the past 30 days?
- Q2. What is the most you had to drink on any one day during the past 30 days?

##### ***Frequency***

- F1. On about how many different days have you had one or more drinks during the past 30 days?
- F2. On about how many days did you have five or more drinks on the same occasion during the past 30 days?

#### **Loss of Control**

- L1. I stayed drunk for more than one day at a time.
- L2. I have awakened unable to remember some of the things I had done while drinking the day before.
- L3. I had a quick drink or so when no one was looking.
- L4. I often took a drink the first thing when I got up in the morning.
- L5. My hands shook a lot after drinking the day before.

#### **Adverse Alcohol-Related Consequences**

- C1. I felt aggressive or cross while drinking.
- C2. I got into a heated argument while drinking.
- C3. I stayed away from work or school because of a hangover.
- C4. I was high or a little drunk when on the job or at school.
- C5. I lost a job, or nearly lost one, because of drinking.

Fig. 1. Measures of alcohol consumption, loss of control and adverse alcohol-related consequence from the National Household Survey on Drug Abuse.

Finally, items were selected indicating the adverse consequences construct ('C' items) that appeared to be particularly relevant to job performance and work habits, including two measures of belligerence and three measures of adverse job performance and risk outcomes.

### **3.3. Statistical analysis**

#### **3.3.1. Confirmatory factor analysis (CFA)**

The maximum likelihood CFA was used to test the structural validity of the measures of the three latent constructs. The specific measurement design, consisting of three correlated factors with three sets of items, was grouped according to the classification suggested in Fig. 1 and

illustrated in Fig. 2. The model was identified by fixing off-factor loadings to zero and scaling the latent factors by fixing one of the on-factor loadings to one.

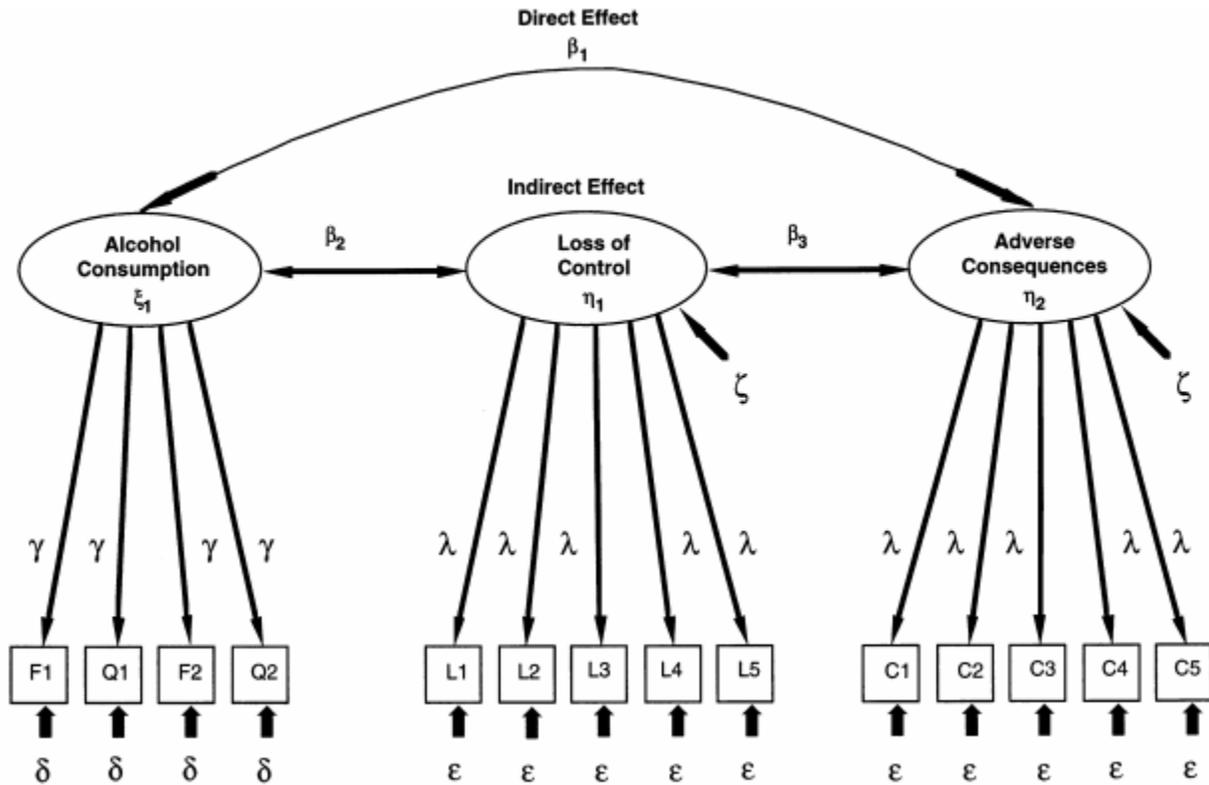


Fig. 2. Hypothesized model of the effects of alcohol consumption and loss of control on adverse alcohol-related consequences.

### 3.3.2. Structural equation model

Simple scales such as those used in the correlational analysis below do not explicitly address the possibility of measurement error in the underlying indicators. Because measurement error may attenuate the correlations between the constructs, a structural equation model was developed and implemented to explicitly model and test the assumptions about measurement error (Lennox et al., 1996 and Drummond, 1992). By using models that systematically control for measurement error, significantly stronger associations between consumption and loss of control and between loss of control and adverse alcohol-related consequences, may be achieved. Using a structural equation framework, the alcohol dependence findings presented by Drummond (1990), Drummond (1992) and Williams and Drummond (1994) imply that the direct relationship between high consumption and adverse alcohol-related consequences will be effectively zero after removing the collinear variation attributed to alcohol dependence.

Fig. 2 illustrates the hypothesized relationship among the three latent constructs labeled: (1) alcohol consumption; (2) loss of control; and (3) adverse alcohol-related consequences. The latent constructs (shown in ellipses in Fig. 2) are measured with multiple indicators (shown in boxes) and modeled as common factors. The relationships among the three latent constructs are the structural coefficients  $\beta_1$ ,  $\beta_2$  and  $\beta_3$  and are shown as arrows connecting the ellipses<sup>3</sup>. The measurement errors ( $\delta$ 's and  $\epsilon$ 's) are indicated by the short arrows under the measured variables

and are estimated as the variance in each indicator not shared with any other indicator or latent construct. Individual reliabilities ( $\gamma$ ' and  $\lambda$ 's) are indicated by the longer arrows between the latent constructs and the measured indicators. Finally, the variance not explained by the latent constructs loss of control and adverse alcohol-related consequences ( $\zeta$ 's) is indicated by the short arrows alongside the ellipses for each endogenous latent variable.

Testing the mediating hypothesis of the alcohol dependence perspective involves estimating the direct and indirect effects of consumption on adverse alcohol-related consequences. As illustrated in Fig. 2, the direct effect is modeled as the structural coefficient connecting consumption with adverse alcohol-related consequences ( $\beta_1$ ). The indirect effects are estimated as one structural coefficient connecting consumption with loss of control ( $\beta_2$ ) and another connecting loss of control with adverse alcohol-related consequences ( $\beta_3$ ).

The alcohol dependence perspective presented by Drummond and Edwards can be described using the structural model shown in Fig. 2. The simplest form of the alcohol dependence model restricts  $\beta_1$  to zero. Estimating this simple model, however, does not test the mediating hypothesis. If the structural model shown in Fig. 2 is estimated without restricting any of the  $\beta$ 's, the alcohol dependence perspective embeds and can test the mediating hypothesis. The mediating hypothesis of the alcohol dependence model implies that  $\beta_1=0$ . Thus, if the full model shown in Fig. 2 is estimated and finds a statistically insignificant or a small estimate of  $\beta_1$ , the alcohol dependence perspective has been confirmed.

### **3.4. Correlational analysis**

We duplicated Williams and Drummond (1994) correlation analysis to compare the relationship between the consumption, loss of control and adverse consequences observed in their respected study of a population of patients in a treatment facility with those in the NHSDA non-clinical population. To do this, simple scales were computed for the consumption, loss of control and consequences constructs by simply summing the items for each construct. Then simple product moment correlation coefficients were computed and partial correlations between these scales.

## **4. Results**

### **4.1. Confirmatory factor analysis**

Detailed results of this analysis are shown in Fig. 3. Results of the CFA indicate that this model produces an adequate fit to the covariance matrix (Normed Bentler-Bonnet Fit index=0.87; Nonnormed Bentler-Bonnet Fit index=0.85; Comparative Fit index=0.88). The  $\chi^2$ -test also showed a significant difference between the observed and model covariance matrices but the large sample size may have made the inferential test overly sensitive to small departures. Although the model does not produce a robust fit to the covariance matrix, the fact that the average standardized residual covariance was only 0.04, indicated that there was very little covariance left to be modeled beyond the three-factor solution. The residual covariance matrix also indicated little evidence of cross-factor loadings. There was some evidence of correlated error between the two frequency items in the NHSDA which apparently resulted from a skip pattern common to both items. However, freeing the restriction of uncorrelated errors for these

two items only slightly improved the model. For the sake of parsimony, the restriction of uncorrelated errors in this analysis was retained. All factor loadings are significantly different from zero at the 0.01 level and all but two loadings are greater than 0.50. The results indicate that item Q14, “What is the most you had to drink in the last 30 days?” is a nearly perfect indicator of the consumption latent construct and is defined by the common factor of high consumption.

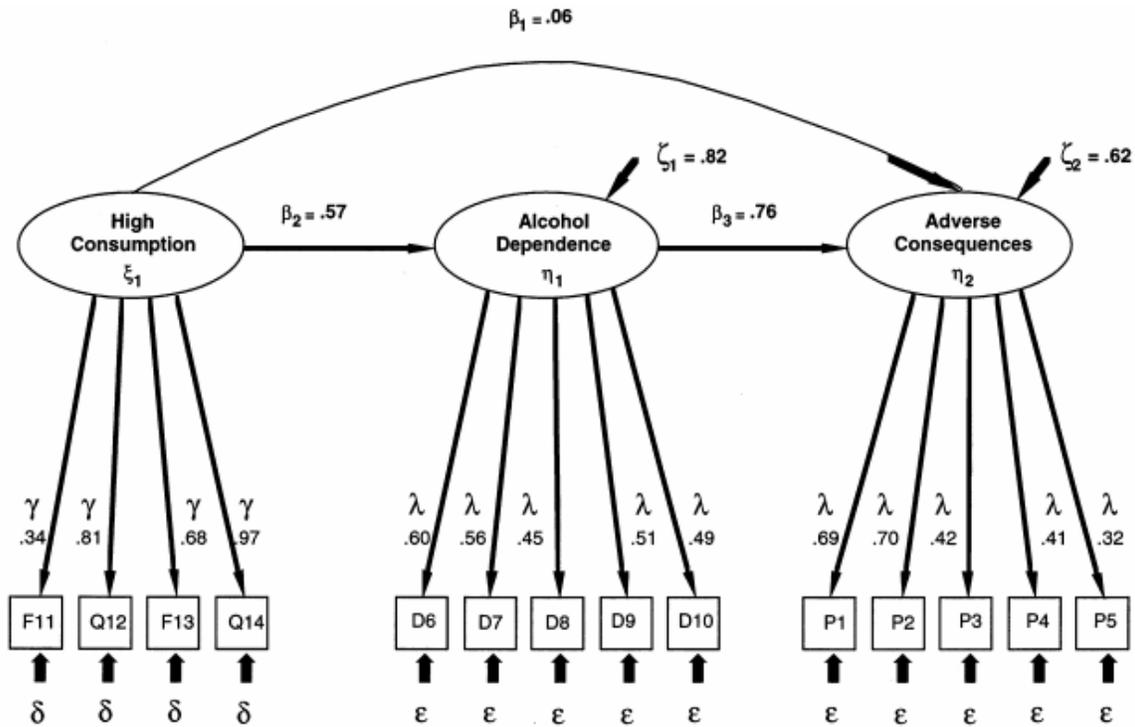


Fig. 3. Mediated structural equation model of the effects of alcohol consumption and loss of control on adverse alcohol-related consequences.

A two-factor model that combined consumption and loss of control into a single factor was also estimated and was found to be statistically inferior to the three-factor model,  $\chi^2$  diff (2)=4,019,  $P < 0.001$ . It was concluded that a three-factor latent construct model adequately operationalizes the measures selected from the NHSDA and is considerably better than a two-factor solution.

#### 4.2. Structural equation model

The test of the loss of control perspective is reflected in the direct and indirect effects of consumption on adverse alcohol-related consequences as shown in Fig. 3. The results show that consumption is strongly associated with loss of control ( $\beta_2 = 0.57, P < 0.001$ ) and that loss of control is strongly associated with adverse consequences ( $\beta_3 = 0.76, P < 0.001$ ). Although the direct effect of high consumption on adverse alcohol-related consequences is statistically significant ( $\beta_1 = 0.06, P < 0.01$ ), its point estimate is essentially zero. To examine the practical significance of the effect of consumption on adverse alcohol-related consequences, a structural model with the restriction that  $\beta_2 = \beta_3 = 0$  was also estimated. Under these conditions, a much larger point estimate of the effect of consumption on adverse alcohol-related consequences was found ( $\beta_1 = 0.46, P < 0.001$ ), suggesting that researchers can easily be misled by naively accepting a direct effect of consumption on adverse consequences.

The results indicate that loss of control is more closely related to adverse alcohol-related consequences than are high rates of alcohol consumption. The mediating model shows that when the effects of loss of control on adverse consequences are removed from the effects of high consumption on consequences, the effects of high consumption decrease substantially.

### 4.3. Correlational analysis

Table 2 presents a comparison of the findings from the NHSDA for the general population with Williams and Drummond's (1994) findings for the clinical population they studied. Using the NHSDA measures, the correlation between consumption and loss of control is 0.46, between consumption and adverse alcohol-related consequences is 0.39 and between loss of control and adverse alcohol-related consequences is 0.57. All of these constructs are significantly related to each other. Also included in this table are partial correlation results using the NHSDA measures. Of particular note, when controlling for loss of control, the relationship between consumption and adverse consequences becomes relatively low (0.18). This suggests a strong mediating effect of loss of control in explaining the relationship between consumption and adverse consequences in a non-clinical sample of members of the labor force.

Table 2  
Simple and partial correlations among alcohol consumption and loss of control on adverse alcohol-related consequences from Williams and Drummond and the NHSDA

Correlations	Simple		Partial	
	Williams and Drummond*	NHSDA	Williams and Drummond*	NHSDA
Loss of control and consequences	0.51 (0.09)	0.57 (0.01)	0.37 (0.09)	0.48 (0.01)
Consumption and consequences	0.41 (0.09)	0.39 (0.01)	0.18 (0.10)	0.18 (0.01)
Consumption and loss of control	0.56 (0.08)	0.46 (0.01)	0.44 (0.09)	0.31 (0.01)

Note: Standard errors in parentheses.

\* Source, Williams and Drummond, 1994. Standard errors not reported in Williams and Drummond but calculated as  $S.E. = [(1 - \rho^2)/(n - 2)]^{0.5}$  where  $n = 101$  as reported in Williams and Drummond.

Table 2 also reproduces the simple and partial correlations from Williams and Drummond's (1994) study of a clinical population. In general, they are quite similar to the current findings, i.e. all simple correlations are significant but controlling for dependency results in a reduced relationship between consumption and problems. Although the differences between the results and those of Williams and Drummond are generally significant, they are not as large as one might expect when comparing a clinical population to the general population. It is noted that both the simple and partial correlations between consumption and loss of control are lower than the analogous relationships in Williams and Drummond's findings. This is attributed to the greater range of responses by the non-clinical sample.

## 5. Discussion

Grounded in the alcohol dependence perspective presented by Clark and Hilton, 1991 and Edwards and Gross, 1976, Williams and Drummond (1994) and others, measures were selected for three alcohol-related constructs—consumption, loss of control and adverse consequences—from measures incorporated in the 1991 NHSDA. A confirmatory factor analysis showed that a three-factor solution for these measures produced a good fit with the responses provided by primary workforce participants.

The analysis was extended to include a latent structural analysis of the three constructs. By comparing the simple and partial correlations from clinical and non-clinical populations represented in Table 2 with the results of the latent variable modeling shown in Fig. 3, it is apparent that measurement error produced substantial bias in the estimates of the relationships among: (1) consumption; (2) loss of control; and (3) adverse alcohol-related consequences constructs. By including measurement models that parcel out measurement error without removing them from the analysis, significantly stronger associations between consumption and loss of control (0.57 compared to 0.46) and between loss of control and adverse alcohol-related consequences (0.76 compared to 0.57) are achieved. At the same time, a very important finding from the structural equation model is that the relationship between consumption and adverse alcohol-related consequences, controlling for the confounding effect of loss of control, becomes effectively zero (0.06 compared to 0.18).

Simple and partial correlations among these constructs produced results that were similar to those reported by Drummond in his study of a clinical population in the UK. That is, the substantial link found between loss of control (dependence) and adverse alcohol-related consequences (problems) shows that, in both clinical and non-clinical populations, dependence is an important mediating variable on consequences and apparently is more important than the direct effect of consumption per se.

The research has some implications for policies and procedures used by decision makers in the workplace as they attempt to prevent and control the impact of alcohol abuse. Workplace prevention and control efforts have been controversial and the source of litigation since their inception (Trice and Schonbrunn, 1981, Steele, 1989 and Denenberg and Denenberg, 1991). From the research, this could be due in large part to the traditional focus on controlling alcohol consumption rather than dependence. The abstinence programs and policies might prevent some instances of alcohol abuse but are not likely to be widely accepted in a national workforce that generally consumes alcohol moderately and responsibly. Consumption-related programs and policies are not likely to succeed because most workers would be unnecessarily constrained in an attempt to control a much smaller target group of dependent alcohol abusers (Staudenmeier, 1987).

In recent years, workplace alcohol testing has become a prevalent and accepted form of alcohol abuse prevention, particularly as a component of drug testing programs and when mandated as in the transportation industries (Hartwell et al., 1996a). However, while alcohol testing might lessen some safety and economic risks among those with low dependence, it is not particularly effective in identifying dependent alcohol abusers because tests measure only current blood alcohol levels and are unable to determine patterns of chronic alcohol misuse (Trice and Steele, 1995). Thus, testing programs alone are unable to prevent the more costly elements of production loss attributed to chronic alcohol abuse (Rice et al., 1990). It is recommended that employers assess those who test positive to alcohol and other substance abuse for their dependency on alcohol as well.

Employee Assistance Programs (EAPs) are the most prevalent mechanisms to intervene with alcohol-related problems encountered by workers on and off the job. These programs now serve

over 1/2 of the US workforce employed in companies with more than 50 workers (Hartwell et al., 1996b). Research has indicated that alcohol problems are reported as the presenting, contributing or related problem among approximately 1/3 of all clients served by EAPs (Blum et al., 1993). From the research, it was suggested that EAP professionals assess the client's dependency on alcohol, rather than relying on information concerning his or her consumption pattern, when determining appropriate treatment services and referrals. Further, monitoring indicators of alcohol dependency after completion of treatment might allow for interventions to prevent relapse and other adverse alcohol-related consequences.

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## **Notes**

1. All analyses was also performed on the sample of respondents age 25–54 who were either employed or had been employed in the past year. Essentially the same results were found using either sample. A full set of results is available from the lead author.
2. Measures of consumption were used that occurred in the 30 days prior to completion of the NHSDA. There were in fact a limited number of consumption indicators available for the year preceding the administration of the survey but none of them were selected because of concerns about recall and their lack of detail. It was confirmed that NHSDA measures of consumption collected for the past month and for the past year were highly correlated with each other, suggesting consistency in adult drinking patterns over time.
3. Figure 2 notes that there may be a reciprocal relationship between the constructs. The literature suggests that drinking affects loss of control, which in turn affects consequences. Alternatively, loss of control may in turn affect consumption and similarly consequences may affect loss of control. However, in cross-section data we cannot identify a reciprocal relationship if it exists. The coefficients  $\beta_1$ ,  $\beta_2$ , and  $\beta_3$  therefore represent the correlation between the constructs with no causality implied.

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