Available online at www.sciencedirect.com

SCIENCE DIRECT®

Addictive Behaviors 30 (2005) 1535–1554

ADDICTIVE BEHAVIORS

Differential effects of past-year stimulant and sedative drug use on alcohol-related aggression

Peter R. Giancola*, Dominic J. Parrott

Department of Psychology, University of Kentucky, 115 Kastle Hall, Lexington, KY 40506-0044, United States

Abstract

The goals of this study were to determine the effects of past-year stimulant and sedative drug use on alcohol-related aggression and to examine whether the relation between stimulant drug use and intoxicated aggression is better accounted for by behavioral disinhibition. Participants were 330 healthy social drinkers (164 men and 166 women) between 21 and 35 years of age. Past-year stimulant and sedative use and behavioral disinhibition were assessed via self-report questionnaires. Following the consumption of either an alcohol or a placebo beverage, participants were tested on a modified version of the *Taylor Aggression Paradigm* [Taylor, S. (1967). Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. *Journal of Personality*, 35, 297–310] in which mild electric shocks were received from, and administered to, a fictitious opponent. Aggressive behavior was operationalized as the shock intensities administered to the fictitious opponent under conditions of low and high provocation. Results indicated that alcohol significantly strengthened the relation between stimulant drug use and aggression, but only among men. Behavioral disinhibition did not account for this effect. Regardless of past-year drug use, alcohol did not facilitate aggression among women. The present findings suggest that stimulant drug use may be a risk factor for intoxicated aggression for men. However, the underlying mechanisms accounting for this effect remain unclear.
© 2005 Elsevier Ltd. All rights reserved.

Keywords: Alcohol; Aggression; Drug Use; Sensation seeking; Impulsivity

* Corresponding author. Tel.: +1 859 257 4502; fax: +1 859 323 1979.
E-mail address: peter@uky.edu (P.R. Giancola).

0306-4603/$ - see front matter © 2005 Elsevier Ltd. All rights reserved.
doi:10.1016/j.addbeh.2005.02.011

Alcohol consumption is related to interpersonal aggression (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997; Ito, Miller, & Pollack, 1996). This association has been documented by both correlational and experimental studies. Correlational research has found alcohol to be present in about 50% of violent crimes (Murdoch, Pihl, & Ross, 1990; Pernanen, 1991). More specifically, a review of this literature indicated that alcohol was present, during the time of the transgression, for 28–86% of homicide offenders, 24–37% of assault offenders, 7–72% of robbery offenders, 13–60% of sexual offenders, 30–70% of suicide attempters, 18–66% of suicide completers, 6–57% of marital violence perpetrators, 13% of child abusers, and 32–54% of child molesters (Roizen, 1993). In addition, correlational studies have also determined that it is the acute effects of alcohol, rather than its chronic effects, that have the largest impact on aggressive behavior (Collins & Schlenger, 1988; Wiley & Weisner, 1995).

Experimental investigations have also demonstrated that alcohol facilitates aggressive behavior. In these studies, aggression has typically been measured using tasks in which participants administer and receive mild electric shocks, tone blasts, or "point subtractions" (redeemable for money) to/from a fictitious opponent under the guise of a competitive interpersonal task (reviewed in Giancola & Chermack, 1998). Results from these studies have indicated that persons who receive alcohol behave more aggressively than those who receive a placebo or a nonalcoholic beverage (reviewed in Bushman & Cooper, 1990; Chermack & Giancola, 1997; Kelly & Cherek, 1993; Taylor & Chermack, 1993). Findings from laboratory studies have also shown that aggression tends to increase in response to rising levels of provocation (Giancola et al., 2002; Hoaken & Pihl, 2000). In fact, provocation has been found to be one of the most powerful elicitors of human aggression both in and out of the laboratory (Anderson & Bushman, 1997; Bettencourt & Miller, 1996; Ito et al., 1996).

An examination of the alcohol and aggression literature clearly indicates that acute alcohol consumption facilitates aggression for some, but not for all persons. In order to clarify why alcohol has this differential effect, theorists have speculated that alcohol is more likely to engender aggression in persons who are already predisposed to behave in such a manner (Collins, 1988; Fishbein, Jaffe, Snyder, Haertzen, & Hickey, 1993; Pernanen, 1991).

It has been suggested that one's drug use preferences might help determine whether one will, or will not, exhibit aggression when intoxicated (Giancola, 2000; Taylor & Chermack, 1993). Most drugs can be categorized into stimulants (e.g., amphetamines) or sedatives (e.g., benzodiazepines). It is well known that many drug users will choose, within one drug-taking session, from both classes of substances in order to produce a "high" or to "come down." However, one can also make the case that individuals with certain dispositional traits will be significantly more likely to choose drugs from one particular class over the other. For example, persons with a reward dominant personality style (i.e., sensation seekers) might be more likely to use stimulants whereas those experiencing chronic anxiety will be more likely to use sedatives. Given the personality traits typically associated with an individual's tendency to use stimulants or sedatives, one's drug of choice may be an especially important marker for predicting intoxicated aggression.

One trait that is strongly associated with regular stimulant use is behavioral disinhibition, which is characterized by a maladaptive pattern of disinhibited behavior that results in recurrent impulsivity and sensation seeking (Baer, 2002). Indeed, research suggests that regular stimulant users report higher levels of sensation seeking (Adams et al., 2003; Carrol & Zuckerman, 1977; Low & Gendaszek, 2002) and impulsivity (Moeller et al., 2002) and are more likely to engage in behaviors that increase arousal (Brecht, O'Brien, von Mayrhauser, & Anglin, 2004) relative to individuals who do not use these drugs. This line of evidence suggests that individuals who use stimulants possess greater levels of behavioral disinhibition and lower levels of fear than non-stimulant users. In accordance with this view, studies indicate that chronic amphetamine use (Kosten & Singha, 1999; Moss, Salloum, & Fischer, 1994) and acute stimulant intoxication (Allen, Safer, & Covi, 1975; Cherek, 1981; Licata, Taylor, Berman, & Cranston, 1993) are strongly associated with heightened aggressive behavior. Moreover, research also suggests that individual differences in these disinhibitory traits, including sensation seeking (Cheong & Nagoshi, 1999), behavioral undercontrol (Parrott & Giancola, 2004), arousal (Giancola & Zeichner, 1997; Graham, Wells, & West, 1997), and reduced fear (Pihl, Peterson, & Lau, 1993), are important risk factors for alcohol-related violence. Therefore, it can be argued that relative to non-stimulant users, stimulant users are more likely to possess higher levels of behavioral disinhibition that may in turn potentiate alcohol-related aggression.

In contrast, regular users of sedative drugs are characterized by higher levels of anxiety (Goodwin & Hasin, 2002; Petrovic et al., 2002) and depression (Goodwin & Hasin, 2002) compared with non-sedative users. In fact, some studies suggest that chronic sedative use may reflect an attempt to self-medicate anxiety and depression (Fava et al., 1997; Patten & Love, 1997). Therefore, given that anxiety and depression are generally negatively related to aggressive behavior, it can be argued that alcohol will most likely not potentiate aggression in these individuals. It is worth noting that while some studies indicate that sedatives might increase aggressive behavior (e.g., Ben-Porath & Taylor, 2002; Berman & Taylor, 1995; Gantner & Taylor, 1988), others have shown that persons low in trait anxiety (i.e., those who are less inhibited and fearful) are most susceptible to this effect (Wilkinson, 1985). Given the theories reviewed above noting that alcohol is more likely to engender aggression for persons who are already predisposed to behave in such a manner (Collins, 1988; Fishbein et al., 1993; Pernanen, 1991), one could argue that, given their characterological make-up, sedative users will be unlikely to become aggressive under alcohol.

Therefore, the purpose of this study was to determine the moderating influence of past-year stimulant and sedative drug use on alcohol-related aggression in men and women and to examine the mechanisms underlying these relations. Three hypotheses were advanced. First, alcohol will be more likely to increase aggression for persons with greater past-year stimulant drug use levels than for those with lower levels. Second, this effect will be accounted for by individual differences in behavioral disinhibition. Third, alcohol will have no effect on the relation between past-year sedative drug use and aggression.

1. Method

1.1. Participants

Participants were 330 (164 men and 166 women) healthy social drinkers between 21 and 35 years of age ($M = 23.04$; $SD = 2.85$). They were recruited through advertisements placed in various newspapers in Lexington, Kentucky. Respondents were initially screened by telephone. Individuals with self-reported past or present drug- or alcohol-related problems or treatment, serious head injuries, learning disabilities, or psychotic or depressive symptomatology were excluded from participation. Individuals who reported abstinence from alcohol use or a condition in which alcohol consumption is medically contraindicated were also excluded. Respondents were screened for alcohol use problems using the *Short Michigan Alcoholism Screening Test* (SMAST; Selzer, Vinokur, & van Rooijen, 1975). Any person scoring an "8" or more on the SMAST was excluded from participation. Three respondents exceeded the SMAST criteria and were not tested. Anyone with a positive breath alcohol concentration (BAC) reading or a positive urine pregnancy or drug test result was also excluded. All pregnancy tests were negative. Two males had a positive BAC and one female had a positive drug test. These individuals were not tested. They were given an opportunity to reschedule another appointment, but they elected to not do so. The sample consisted of 302 Caucasians, 27 African-Americans, and 1 Hispanic-American. Eighty-nine percent of the participants were never married, 31% had a high-school degree and were not pursuing further education, 44% had a high-school degree and were working on a bachelor's or an associate's degree, 22% had a bachelor's or an associate's degree, 2% had a graduate degree, and 1% did not graduate high-school. Forty-six percent of the sample supported themselves financially and earned an average of nearly $18,000 per year; the remainder were supported by a parent or a spouse.

1.2. Pre-laboratory procedures

Following the telephone screening interview, individuals eligible for participation were scheduled for an appointment to come to the laboratory. They were told to refrain from drinking alcohol 24 h prior to testing, to refrain from using recreational drugs from the time of the telephone interview, and to refrain from eating 4 h prior to testing. Due to hormonal variations associated with menstruation which may affect aggressive responding, women were not tested between one week before menstruation and the beginning of menstruation. Participants were told that they would receive $50 at the completion of the study as compensation.

1.3. Assessment of past-year stimulant and sedative drug use

After establishing that participants met all of the inclusion criteria, demographic data were collected. Participants then completed a questionnaire to determine their frequency of stimulant and sedative drug use for the past year (Tarter, 1989). Participants were asked how

often, over the past year, did they use "stimulant" and "sedative" drugs on a scale ranging from "0" (have never tried) to "8" (used every day). We assessed both illicit and licit (over-the-counter) substances. The following terms were used as examples of "stimulants:" cocaine, crack, amphetamines, ice, uppers, speed, crank, crystal meth, bam, diet pills, caffeine pills, etc. The following terms were used as examples of "sedatives:" downers, barbiturates, benzodiazepines, quaaludes, Valium, Xanex, Seconol, Reds, Miltown, sleeping pills and cough syrup (only if used to get "high"), etc.

1.4. Behavioral disinhibition

Behavioral disinhibition was measured using the *Sensation Seeking Scale* (*SSS*; Zuckerman, Eysenck, & Eysenck, 1978) and the *Barratt Impulsivity Scale* (*BIS*; Patton, Stanford, & Barratt, 1995). The SSS is a 40-item 2-choice inventory with 4 subscales (Thrill and Adventure Seeking, Experience Seeking, Disinhibition, and Boredom Susceptibility). It assesses individual differences in the proclivity to take risks motivated by poor inhibitory control as well as the desire to experience high levels of stimulation and arousal. None of the 40 items assess aggression in any way. The scores from all 4 subscales were summed. Higher total scores are indicative of higher levels of sensation seeking. The SSS has been shown to have very good psychometric properties (Zuckerman et al., 1978).

The BIS is a 30-item inventory in which participants rate various behavioral tendencies from "1" (*Rarely/Never*) to "4" (*Almost always/Always*), with higher scores indicative of greater impulsivity. This measure has been shown to correlate significantly with numerous indices of impulsive and risk-taking behavior (Barratt, 1994). It has also been shown to have sound psychometric properties (Patton et al., 1995).

1.5. Experimental design

This study had four independent variables: Beverage, gender, provocation, and past-year drug use. Participants were assigned to one of the following groups: (a) men who received alcohol ($n=82$), (b) men who received a placebo ($n=82$), (c) women who received alcohol ($n=83$), and (d) women who received a placebo ($n=83$). All participants received both levels of provocation thus making it a repeated measure.

1.6. Beverage administration

Men receiving alcohol were administered a dose of 1 g/kg of 100% alcohol USP mixed at a 1 : 5 ratio with Tropicana orange juice. Due to differences in body fat composition, women were given a dose of 0.90 g/kg of alcohol. Beverages were poured into the requisite number of glasses in equal quantities. The dosing procedure was also calculated for the placebo groups, however, they received an isovolemic beverage consisting only of orange juice (i.e., the missing alcohol portion was replaced with orange juice). Three c.c.s of alcohol were added to each placebo beverage and 3 c.c.s were layered onto the juice in each glass. Immediately prior to serving the placebo beverages, the rims of the glasses were sprayed with alcohol.

1.7. Aggression task

A modified version of the *Taylor Aggression Paradigm* (Taylor, 1967) was used to measure aggression. The hardware for the task was developed by Coulbourne Instruments (Allentown, PA) and the computer software was developed by Vibranz Creative Group (Lexington, KY). This task places participants in a situation where electric shocks are received from, and administered to, a fictitious opponent during a supposed competitive reaction-time task. Physical aggression was operationalized as the shock intensities selected by the participants. The Taylor task and other similar laboratory paradigms have repeatedly been shown to be safe and valid measures of aggressive behavior for men and women (Anderson & Bushman, 1997; Giancola & Chermack, 1998; Hoaken & Pihl, 2000; Richardson, Bernstein, & Taylor, 1979; Richardson, Vinsel, & Taylor, 1980).

Participants were seated at a table in a small room. On the table facing the participant was a computer screen and a keyboard. White adhesive labels marked "1" through "10" were attached to the number keys running across the top of the keyboard. The labels "low," "medium," and "high" were placed above keys "1," "5," and "10," respectively, to indicate the subjective levels of shock corresponding to the number keys. The keyboard and monitor were connected to a computer located in an adjacent control room out of the participant's view.

1.8. Measures of aggression

1.8.1. Mean shock intensity

This measure comprises the mean shock intensity selection ("1" through "10") within each provocation condition. As such, two separate dependent variables were calculated: 1) mean shock intensity under low provocation and 2) mean shock intensity under high provocation. These variables represent measures of aggression in response to provocation.

1.8.2. Proportion of "10s" selected ("Extreme Aggression")

This measure constitutes the mean proportion of times that participants selected the highest shock intensity button (i.e., "10") within each provocation condition. Similar to the shock intensity variable, separate extreme aggression means were calculated for the low and high provocation conditions, thus yielding two variables. Although issue can be taken with nomenclature, it has been argued that this measure reflects the tendency to exhibit "extreme" levels of aggression in response to provocation (Chermack & Taylor, 1995).

1.9. Procedure

Upon entering the laboratory, participants were explained the procedures of the study and were asked to sign an informed consent form. The experimenter then assessed their BACs to ensure sobriety. If the BAC test was negative, participants then underwent a urine drug test and women also underwent a urine pregnancy test. BACs were measured using the Alco-Sensor IV breath analyzer (Intoximeters, St-Louis, MO). Demographic data were then collected and participants then completed the drug use questionnaire, SSS, and BIS.

Participants were then escorted into the testing room where they received their beverages. Twenty minutes were allotted for beverage consumption. In order to allow the alcohol to be sufficiently absorbed into the bloodstream, persons receiving alcohol had their pain thresholds tested (described below) 15 min after they finished their drinks. In order to maximize the placebo manipulation, individuals in the placebo group had their pain thresholds tested 2 min after they finished their drinks. It has been shown that placebo manipulations are only effective shortly after beverage consumption (Bradlyn & Young, 1983; Martin & Sayette, 1993; Martin, Earleywine, Finn, & Young, 1990). As such, testing pain thresholds 2 min after beverage consumption ensured that aggression was assessed while the placebo manipulation was most effective (Martin et al., 1990; Martin & Sayette, 1993). BACs were measured following the pain threshold testing. The placebo group began the aggression task immediately after the pain threshold testing.

Given that the aggression-potentiating effects of alcohol are more likely to occur on the ascending limb of the BAC curve (Giancola & Zeichner, 1997) and because a BAC of at least 0.08% is effective in eliciting robust levels of aggression (Giancola & Zeichner, 1997; Gustafson, 1992; Pihl, Smith, & Farrell, 1984), the alcohol group began the task shortly after they reached an ascending BAC of at least 0.09% (two men and one woman never achieved this BAC and were thus removed from the study). This methodology indicates that we decided to standardize BAC on the ascending limb of the BAC curve rather than time/latency following beverage consumption. One could argue that the time duration between the end of beverage consumption and beginning the aggression task should have been standardized for both beverage groups. This was not done because it would have reduced the effectiveness of the placebo manipulation (noted above) and would have produced undesirably large individual differences in BACs during the aggression task. Finally, immediately before beginning the aggression task, participants provided subjective ratings of their level of intoxication. This was done using a specially constructed scale ranging from 0 to 11 on which “0” was labeled “not drunk at all,” “8” was labeled “drunk as I have ever been,” and “11” was labeled “more drunk than I have ever been.”

Participants’ pain thresholds were then assessed to determine the intensity parameters for the shocks they would receive. This was accomplished via the administration of short duration shocks (1 s) that increased in intensity in a stepwise manner from the lowest available shock setting, which was imperceptible, until the shocks reached a subjectively-reported “painful” level. All shocks were administered through two finger electrodes attached to the index and middle fingers of the nondominant hand using Velcro straps. Participants were instructed to inform the experimenter when the shocks were “first detectable” and then when they reached a “painful” level. Later, during the actual testing, participants received shocks that ranged from “1” to “10.” These shocks were respectively set at 55%, 60%, 65%, 70%, 75%, 80%, 85%, 90%, 95%, and 100% of the highest tolerated shock intensity. The threshold determination procedure was conducted while the participant was seated in the testing room and the experimenter was in the adjacent control room. They communicated through an intercom. The experimenter secretly viewed the participant through a hidden video camera.

Following the pain threshold testing, participants were once again explained the aggression task. They were informed that shortly after the words "Get Ready" appeared on the screen, the words "Press the Spacebar" would appear at which time they had to press, and hold down, the spacebar. Following this, the words "Release the Spacebar" would appear at which time they had to lift their fingers off of the spacebar as quickly as possible. A "win" was signaled by the words "You won. You Get to Give a Shock" and a "loss" was signaled by the words "You Lost. You Get a Shock." A winning trial allowed participants to deliver a shock to their opponent and a losing trial resulted in receiving a shock from this individual. Following a winning trial and pressing a shock button, participants could view their shock selection on a specially designed "volt meter" on the computer screen and by the illumination of one of 10 "shock lights" [ranging from 1 (low) to 10 (high)] on the computer screen. Both of these indicators displayed readings that corresponded with the shock level they selected. These images were used to reinforce participants' beliefs that they were actually administering shocks. Upon losing a trial, participants received a shock and were given feedback regarding the level of that shock in the form of a signal on the volt meter and the illumination of one of the 10 "shock lights" on the computer screen.

Participants were told that they had a choice of 10 different shock intensities to administer at the end of each winning trial for a duration of their choosing. Regardless of beverage group assignment, all participants were informed that their opponent was intoxicated. This was done to ensure that the "drinking status" of the opponent would not confound any potential beverage group differences in aggression.

The entire procedure consisted of two successive blocks of trials. During the first block, participants received shock intensities between "1" and "4" (mean intensity = 2.5) after they lost a trial. This denoted the low provocation condition. During the next block, they received shock intensities between "7" and "10" (mean intensity = 8.5) after they lost a trial. This denoted the high provocation condition. Each block consisted of 16 trials (8 wins and 8 loses). There were two "transition trials" between the blocks. Participants lost both of the trials and received respective shock intensities of "5" and "6." These trials were added to give the appearance of a smooth transition between the low and high provocation blocks. Thus, there were a total of 34 trials. Having the high provocation condition always follow the low provocation condition is an intentional aspect of this task. Taylor and Chermack (1993) have argued that using the low–high sequence adds an increased degree of external validity to the task because this ordering best reflects how an escalation in interpersonal provocation leads to increased violence in "real-life" situations.

All shocks delivered to the participants were of a 1 s duration. In actuality, reaction-times were not measured, the competitive task was used to lead participants to believe that they were engaging in an adversarial interaction with another individual. The win/lose sequence was predetermined and controlled by the computer program that executed the task. The sequence was presented in a randomized fashion, however, each participant received the same sequence. The trials were interspersed by 5 s intervals. The initiation of trials, administration of shocks to the participants, and the recording of the participants' responses were controlled by a computer. The experimenters, other electronic equipment, and the computer that

controlled the task were located in an adjacent control room out of the participants' view. The experimenter secretly viewed and heard the participant through a hidden video camera and microphone throughout the procedure.

Immediately following the testing procedure, BACs were measured and participants were again asked to rate their subjective state of intoxication. In addition to this, they were asked whether the alcohol they drank caused them any impairment on a scale ranging from 0 to 10 on which "0" was labeled "no impairment," "5" was labeled "moderate impairment," and "10" was labeled "strong impairment." Participants were then asked a yes/no question regarding whether they believed that they had consumed alcohol. They were also asked a variety of questions to indirectly assess the credibility of the experimental manipulation (see below). Participants were then compensated. All individuals who received alcohol were required to remain in the laboratory until their BAC dropped to 0.04%.

1.10. Deception manipulation

In order to disguise the task as a measure of aggression, participants were given a fictitious cover story. They were informed that the study was aimed at understanding how a person's "thinking-style" and personality influence alcohol's effects on reaction-time in a competitive situation. In order to convince participants that they were actually competing against another person, a confederate was seated in a room adjacent to the testing room. As the experimenter led the participant into the testing room, he/she identified the confederate (same gender as the participant) as the "opponent." No opportunity for an interaction between the participant and the confederate was allowed. Furthermore, immediately prior to testing their pain thresholds, participants were informed that their competitor would undergo the same threshold testing procedure first. They were also informed that they would be able to hear his/her responses over an intercom that ostensibly served the two testing rooms and the control room. In actuality, the confederate acted as the fictitious opponent and answered the experimenter's questions regarding the testing of his/her pain thresholds in accordance with a list of predetermined responses. All participants heard the same experimenter–confederate verbal exchange. Of course, in actuality, there was no real opponent.

2. Results

2.1. Manipulation checks

2.1.1. Aggression task checks

In order to verify the success of the task deception, participants were asked about their subjective perceptions of their opponent, whether their opponent tried hard to win, whether they thought the task was a good measure of reaction-time, and how well they believed they performed on the task. The deception manipulation appeared successful. Typical descriptions from participants about their opponents included profane derogatory remarks as well as statements that the person was "okay," "a good competitor," and "fast." Some participants also

Table 1
Demographic, drinking, and drug use data

Measure	Men		Women	
	M	*SD*	*M*	*SD*
Age	23.38	3.06	22.71	2.60
Years of education	16.00	2.11	16.27	2.00
Salary ($)	18.53*K*	12.58*K*	17.72*K*	9.53*K*
Stimulant drug use	5.63	3.02	5.30	2.57
Sedative drug use	3.31	1.48	3.26	1.78
SMAST	1.87	2.77	0.36	1.44*
Age at first drink	15.28	2.65	15.72	2.46
Age when first drunk	15.96	2.44	16.58	2.51
Age when regular drinking began	18.56	2.38	19.12	2.20
Drinks per week	15.44	12.75	6.67	6.29*
Cigarettes per day	5.38	8.09	4.73	8.44
Sensation seeking	24.22	5.22	20.20	6.28*
Impulsivity	69.48	10.16	70.11	10.75

SMAST = Short Michigan Alcoholism Screening Test, *K* = $1000.
* $p < 0.05$.

indicated that they had "no feelings for this person either way." The majority of participants stated that they did well on the task and thought that their opponent tried hard to win. All felt that the task was a good measure of reaction-time. Five individuals (2 women and 3 men) reported that they did not believe that they were competing against another person and were thus removed from the analyses. Otherwise, no participant raised any suspicion about the credibility of the task.

2.1.2. Placebo checks

All participants in the placebo group indicated that they believed that they drank alcohol. In response to the question regarding how drunk they felt, persons in the alcohol group reported average pre- and post-task ratings of 4.5 and 5.0 (scale range: 0–11). The placebo group reported average ratings of 1.6 and 2.0, respectively, [pre-task ratings: $t(328) = -16.1$, $p < 0.01$; post-task ratings: $t(328) = -14.6$, $p < 0.01$]. In response to the question about whether the alcohol they drank caused any impairment, persons in the alcohol group reported an average rating of 5.8 and those in the placebo group reported an average rating of 2.0, $t(328) = -16.9$, $p < 0.01$, (scale range: 0–10). There were no significant gender or gender × beverage group effects for any of the placebo check measures.

2.1.3. BAC levels

All participants tested in this study had BACs of 0% upon entering the laboratory. Individuals in the alcohol group had a mean BAC of 0.098% ($SD = 0.01$) just before beginning the aggression task and a mean BAC of 0.104% ($SD = 0.01$) immediately after the task. Persons given the placebo had a mean BAC of 0.01% ($SD = 0.01$) just before the task and a mean BAC of 0.01% ($SD = 0.01$) immediately after the task. There were no significant gender or gender × beverage group effects for any of the BAC measures.

2.2. Demographic data

In order to test for unexpected group differences, all demographic variables were analyzed using 2 (beverage) × 2 (gender) between-groups design analyses of variance (ANOVA). As expected, there were no such differences. However, significant gender main effects indicated that, compared with women, men consumed more alcoholic drinks per week, $F(1, 329) = 62.43$, $p < 0.01$, had higher SMAST scores, $F(1, 329) = 37.76$, $p < 0.01$, and reported higher levels of sensation seeking, $F(1, 329) = 40.12$, $p < 0.01$. However, men and women did not differ with regard to age, years of education, salary, past-year stimulant drug use, past-year sedative drug use, impulsivity, age at first drink, age when regular drinking began, age when first drunk, and cigarettes per day. These data are presented in Table 1.

2.3. Regression analyses

As was noted earlier, the principal aim of this study was to determine whether past-year stimulant and sedative drug use would moderate the alcohol–aggression relation in men and women. Furthermore, we also sought to examine whether deviations in behavioral disinhibition (sensation seeking and impulsivity) would better account for the relation between past-year stimulant drug use and alcohol-related aggression. Separate equations were calculated for each drug use variable (i.e., stimulant use and sedative use). Given that the drug use scores were continuous in nature, regression analyses were indicated. Drug use scores were first converted into *z*-scores. Beverage and gender groups were coded as follows: alcohol = 1; placebo = −1 and male = 1; female = −1. Interaction terms were calculated by obtaining the cross-products of pertinent first-order variables. It is important to create interaction terms using *z*-scores rather than raw scores inasmuch as standardizing cross-products after they have already been created does not yield the same regression coefficients as multiplying standardized values (Aiken & West, 1991; Friedrich, 1982). Standardizing the first-order variables also automatically centers the values (i.e., deviation scores with a mean of zero), which reduces multicollinearity between interaction terms and their constituent lower-order terms (Aiken & West, 1991). When using this procedure, it is important to interpret the unstandardized, and not the standardized, regression solution. Traditional standardized solutions should not be interpreted because they are not scale invariant for multiplicative terms and will thus yield incorrect regression coefficients for these effects. As such, readers should be aware that the parameter estimates for the regression equations are reported as unstandardized *b*s.

Analyses were conducted using two-step hierarchical regression equations. The first step involved the entry of all main effects as well as all 2- and 3-way interactions. In analyses of past-year stimulant use, a second step was performed that involved the entry of sensation seeking and impulsivity (behavioral disinhibition) to determine whether these variables had any diminishing effect on the impact of stimulant drug use. This resulted in a full model comprised of nine variables. Due to the fact that the dependent variable in this study was a repeated-measure (Low Provocation and High Provocation), the use of standard regression techniques was not possible unless separate models were computed for each level of

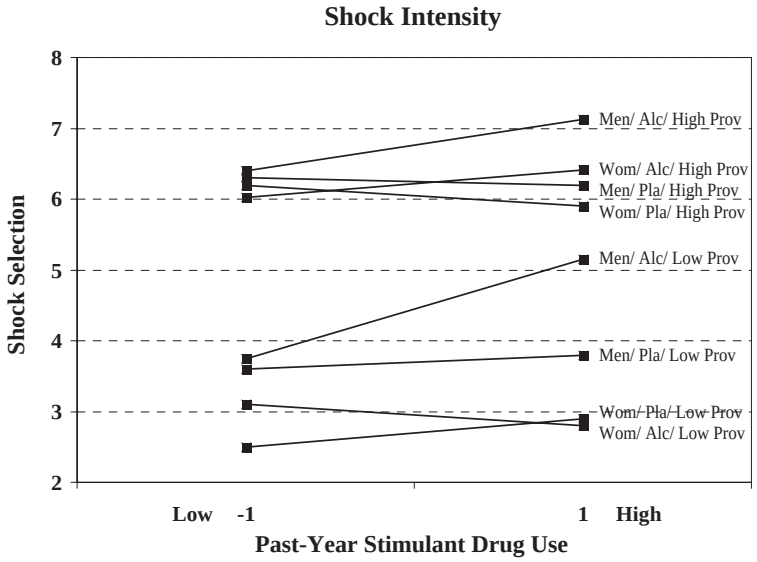

Fig. 1. Effects of alcohol, gender, provocation, and past-year stimulant drug use on aggression (shock intensity).

provocation. This would not have been optimal because it would not allow the testing of any interaction terms involving provocation. Given this, the *Sum/Difference* regression method was chosen because it affords the ability to examine interaction terms involving repeated-measure variables (Hope, 1975; Judd, Kenny, & McClelland, 2001). This method is the "regression equivalent" of a between–within, or mixed model, analysis in ANOVA (S. West, personal communication, 2002).

Using this method, two new dependent variables were created. The first, (DV1), constitutes the sum of the low and high provocation responses (DV1 = Low Provocation + High Provocation) and the second, (DV2), constitutes the difference between the two responses (DV2 = Low Provocation − High Provocation). The regression model is then computed twice; once for DV1 and then for DV2. The coefficients for the DV1 model represent all of the "between" effects and those for the DV2 model represent all of the "within" effects. The coefficient for the intercept for the DV2 model represents the test for the difference between the two provocation conditions. Again these effects are equivalent to those produced by a between–within, or mixed model, analysis in ANOVA.

Finally, according to the procedures put forth in Aiken and West (1991), significant interaction terms were interpreted by plotting the effect and testing to determine whether the slopes of the simple regression lines differed significantly from zero.

2.4. Past-year stimulant drug use

2.4.1. Mean shock intensity

The first step of the model for the between effects (DV1) was significant, $F(7, 322) = 4.40$, $p < 0.001$; $R^2 = 0.09$. Gender ($b = 0.69$, $p < 0.001$) and Stimulant Drug Use

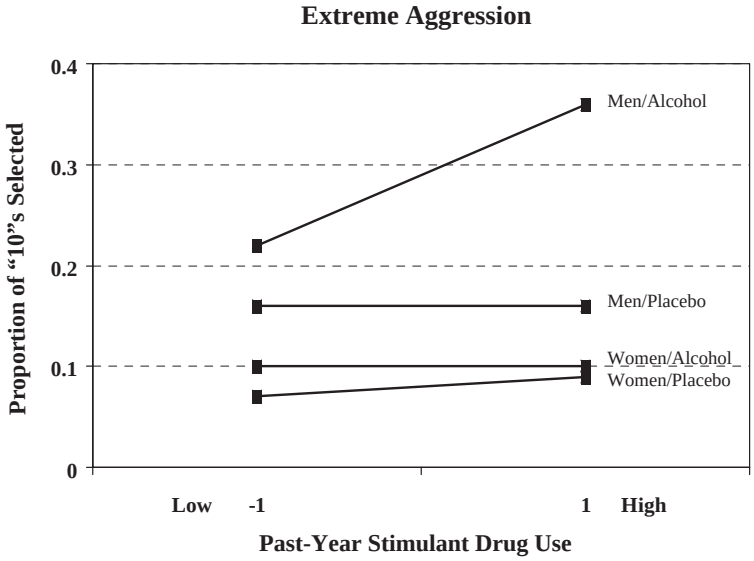

Fig. 2. Effects of alcohol, gender, and past-year stimulant drug use on extreme aggression (proportions of "10s" set).

($b=0.55$, $p<0.01$) were the only significant variables. In the next step, sensation seeking and impulsivity were entered simultaneously. This step was also significant, $F(9, 320)=3.58$, $p<0.001$; $R^2=0.09$. However, the addition of these variables did not significantly increase the explained variance in the model. Gender ($b=0.71$, $p<0.01$) and Stimulant Drug Use ($b=0.48$, $p<0.01$) remained the only significant variables in the final model, indicating that men were more aggressive than women and that stimulant drug use was positively related to aggression.

The first step of the model for the within effects (DV2) was also significant, $F(7, 322)=4.43$, $p<0.001$; $R^2=0.09$. Provocation ($b=-2.91$, $p<0.001$), Gender × Provocation ($b=0.40$, $p<0.001$), and Beverage × Gender × Stimulant Drug Use × Provocation ($b=0.28$, $p<0.01$) were all significant. As in the analyses of the between-subject effects, sensation seeking and impulsivity were entered simultaneously in the second step of the within-subjects model. This step was significant, $F(9, 320)=3.63$, $p<0.01$; $R^2=0.09$. The addition of these variables did not significantly increase the explained variance in the model. Provocation ($b=-2.68$, $p<0.001$), Gender × Provocation ($b=0.34$, $p<0.001$), and Beverage × Gender × Stimulant Drug Use × Provocation ($b=0.28$, $p<0.01$) all remained significant in the final model. Taken together, these findings indicate that across beverage type, gender, and provocation, sensation seeking and impulsivity did not account for the relation between stimulant drug use and aggression.

A plot of the 4-way Beverage × Gender × Stimulant Drug Use × Provocation interaction revealed that the relation between stimulant drug use and aggression was only significant for men given alcohol, under low provocation, ($b=0.79$, $p<0.01$). These data are displayed in Fig. 1. Out of all the variables, provocation had the strongest effect on aggression. In order

to not duplicate previously published data, readers interested in significant gender effects not involving drug use (i.e., Gender × Provocation; Gender × Beverage) for this, and all remaining analyses, are referred to Giancola et al. (2002).

2.4.2. Extreme aggression

The first step of the model for the between effects (DV1) was significant, $F(7, 322) = 13.81$, $p < 0.001$; $R^2 = 0.23$. Beverage ($b = 0.07$, $p < 0.001$), Gender ($b = 0.12$, $p < 0.001$), Stimulant Drug Use ($b = 0.06$, $p < 0.01$), Beverage × Gender ($b = 0.05$, $p < 0.01$), and Beverage × Gender × Stimulant Drug Use ($b = 0.05$, $p < 0.05$), were all significant. The next step of the model was also significant, $F(9, 320) = 10.96$, $p < 0.001$; $R^2 = 0.24$. However, the addition of sensation seeking and impulsivity did not significantly increase the explained variance. Beverage ($b = 0.06$, $p < 0.001$), Gender ($b = 0.12$, $p < 0.001$), Stimulant Drug Use ($b = 0.05$, $p < 0.01$), Beverage × Gender ($b = 0.05$, $p < 0.01$), and Beverage × Gender × Stimu- Stimulant Drug Use ($b = 0.05$, $p < 0.05$), all remained significant in the final model. A plot of the 3-way Beverage × Gender × Stimulant Drug Use term indicated that the relation between stimulant drug use and extreme aggression was only significant for men given alcohol, ($b = 0.09$, $p < 0.01$). These data are presented in Fig. 2.

The first step of the model for the within effects (DV2) approached significance, $F(7, 322) = 1.92$, $p < 0.08$; $R^2 = 0.04$. Nevertheless, Provocation ($b = -0.19$ $p < 0.001$), Gender × Provocation ($b = -0.02$, $p < 0.05$), and Gender × Stimulant Drug Use × Provocation ($b = 0.02$, $p < 0.05$) were significant. The second step of the within effects model was not significant. Collectively, the findings for extreme aggression indicate that across beverage type, gender, and provocation, sensation seeking and impulsivity did not account for the relation between stimulant drug use and extreme aggression. Again, out of all of the variables, provocation had the strongest effect on extreme aggression.

2.5. Past-year sedative drug use

2.5.1. Mean shock intensity

The between effects (DV1) model was significant, $F(7, 322) = 2.88$, $p < 0.01$; $R^2 = 0.06$. Beverage ($b = 0.43$, $p < 0.05$) and Gender ($b = 0.73$, $p < 0.001$) were the only significant variables. The model for the within effects (DV2) was also significant, $F(7, 322) = 4.29$, $p < 0.001$; $R^2 = 0.09$. Provocation ($b = -2.91$, $p < 0.001$) and Gender × Provocation ($b = 0.42$, $p < 0.001$), were the only significant variables. Out of all the variables, provocation had the strongest effect on aggression. Sedative drug use was not related to aggression.

2.5.2. Extreme aggression

The between effects (DV1) model was significant, $F(7, 322) = 9.30$, $p < 0.001$; $R^2 = 0.17$. Beverage ($b = 0.07$, $p < 0.001$), and Gender ($b = 0.13$, $p < 0.001$), were the only significant variables. The within effects (DV2) model was not significant. Nevertheless, Provocation ($b = -0.19$, $p < 0.001$), and Gender × Provocation ($b = -0.03$, $p < 0.05$), were significant. Again, out of all of the variables, provocation had the strongest effect on aggression. Sedative drug use was not related to aggression.

3. Discussion

This is the first investigation to examine the moderating effects of past-year drug use on the alcohol–aggression relation in men and women. The finding of paramount interest in this study was that, compared with placebo, alcohol significantly strengthened the relation between past-year stimulant drug use and aggression, but only for men. In other words, alcohol was more likely to increase aggression for men with greater past-year stimulant drug use than for those with less stimulant drug use levels under both provocation conditions. However, for the shock intensity variable, alcohol's effect on aggression was stronger under low provocation (see Fig. 1). This finding is noteworthy given that a meta-analysis indicated that alcohol has a stronger effect on aggression under conditions of low, compared with high, provocation (Ito et al., 1996). Ito et al. (1996) explained that because high provocation has such a strong impact on aggression, the additional effect produced by alcohol is relatively weak. However, inasmuch as low provocation has a smaller effect on aggression, alcohol has "more room" to make a large impact.

As expected, alcohol did not influence the relation between past-year sedative drug use and aggression for men or women. This finding suggests that regular sedative users, who are characterized by high levels of anxiety and increased inhibition, are not especially susceptible to the aggression-promoting effects of alcohol. Results for women also showed that neither past-year stimulant drug use nor alcohol intoxication had any effect on aggression. This finding deserves some discussion. It should be made clear that our findings are consistent with those of five other laboratory studies, that used shock-aggression tasks, which showed that alcohol does not appear to increase aggression in women (Giancola & Zeichner, 1995; Giancola et al., 2002; Gustafson, 1991; Hoaken, Campbell, Stewart, & Pihl, 2003; Hoaken & Pihl, 2000). One possible explanation for the negative findings for women could be that certain factors related to societal gender-role expectations inhibited their responses. Specifically, despite being given alcohol and having high past-year stimulant drug use levels, some possible reasons for why women in the present study did not exhibit increased aggression include a) the greater societal constraints placed on the expression of female, versus male, aggression; b) societal expectations for women to not express aggression; and c) the fact that women may have developed other coping mechanisms/styles other than aggression that are more socially accepted. The only factor that significantly increased aggression for women in this study was provocation.Our findings indicate that elevated past-year stimulant drug use is related to increased levels of intoxicated aggression for men. It was posited that behavioral disinhibition (i.e., higher levels of sensation seeking and impulsivity) would account for this effect. This prediction was based upon research literature suggesting that regular stimulant users report high levels of sensation seeking (Adams et al., 2003; Low & Gendaszek, 2002) and impulsivity (Moeller et al., 2002) and are likely to engage in "high arousal" behaviors (Brecht et al., 2004). Due to a reduced ability to regulate their behavior, these individuals are believed to be less capable of inhibiting aggressive impulses when intoxicated and provoked. In contrast, regular sedative users appear to be less impulsive as evidenced by higher levels of trait anxiety and a tendency to avoid

arousal- and fear-inducing stimuli (Goodwin & Hasin, 2002). Consequently, sedative users may be more capable of inhibiting/regulating aggressive impulses when intoxicated and provoked. The present results are not consistent with this theorizing, as sensation seeking and impulsivity did not account for the relation between stimulant drug use and intoxicated aggression. As a result, the mechanisms underlying this relation remain unclear. Additional research is needed to determine whether behavioral disinhibition adequately explains the higher levels of intoxicated aggression observed among regular stimulant users.

Because it remains to be seen whether premorbid personality traits account for the present findings, it is reasonable to contend that regular stimulant use contributes to biological or psychological changes that may increase one's susceptibility to the aggression-promoting effects of alcohol. If true, the present findings may have immediate relevance to health professionals who work in applied settings, especially those who prescribe stimulant medications. For example, health professionals who prescribe stimulant medication to individuals diagnosed with Attention Deficit-Hyperactivity Disorder may wish to assess their patients for a history of alcohol use/abuse. In doing so, the likelihood of increasing patients' risk for intoxicated aggression during long-term pharmacotherapy (i.e., the prescription of stimulant medication) may be avoided. Despite this possibility, it must be stressed that future research is still needed to elucidate the mechanism underlying our findings before conclusive clinical recommendations can be made.

Several limitations of the present study deserve mention. First, only two self-report measures of behavioral disinhibition were employed. It is possible that other instruments, including behavioral tasks, might more effectively assess this construct and may yield more promising findings. Future research is needed to examine this possibility. Second, due to the nature of drug use in our sample, the present investigation was unable to determine the relative personality differences between regular stimulant (e.g., more impulsive, less anxious) and sedative users (e.g., less impulsive, more anxious). As such, it is unclear whether the traits hypothesized to facilitate intoxicated aggression in stimulant users (e.g., behavioral disinhibition), and inhibit intoxicated aggression among sedative users (e.g., increased anxiety and fearfulness), actually distinguish these two groups of drug users. This limitation speaks to the complex nature of drug abuse, in that regular drug users tend to use multiple drugs and rarely do individuals use drugs exclusively from one drug class. Therefore, in the present study, it was difficult to group participants into "exclusive" stimulant and sedative drug use groups that could be easily comparable on various personality measures. Despite this limitation, however, individuals who reported greater stimulant drug use during the past year, regardless of their use of other drugs, were more susceptible to the aggression-promoting effects of alcohol. This finding highlights the importance of a recent history of heavy stimulant use as a risk factor for alcohol-related aggression.

In conclusion, this is the first investigation to examine the moderating effects of past-year stimulant and sedative drug use on the alcohol–aggression relation in men and women. The results support the hypothesis that alcohol is more likely to increase aggression in men with higher, as opposed to lower, levels of past-year stimulant drug use.

Given that this is the first study to demonstrate this effect, it is important to replicate this finding both in and out of the laboratory. For example, field studies could use retrospective assessment methods, such as a modified version of the Time-Line Follow Back interview (Sobell & Sobell, 1992), to assess the moderating effect of past stimulant use on the relation between episodes of acute alcohol intoxication and aggression. In addition, survey-based research is especially well suited for the assessment of "at risk" populations where alcohol administration may be contraindicated, such as individuals incarcerated for alcohol- or drug-related offenses. Unfortunately, it remains unclear which personality traits best account for this effect. This fact underscores the importance of taking a multivariate approach to the study of alcohol and aggression. In doing so, scientists can better predict in whom and in what circumstances alcohol will facilitate aggression. Moreover, such an approach will enhance the ability to elucidate the mechanisms that underlie the alcohol–aggression relation.

Acknowledgements

This research was supported by grant R01-AA-11691 from the *National Institute on Alcohol Abuse and Alcoholism.*

References

Adams, J., Heath, A., Young, S., Hewitt, J., Corely, R., & Stallings, M. (2003). Relationships between personality and preferred substance and motivations for use among adolescent substance abusers. *American Journal of Drug and Alcohol Abuse*, *29*, 691–712.

Aiken, L., & West, S. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage Publications.

Allen, R. P., Safer, D., & Covi, L. (1975). Effects of psychostimulants on aggression. *The Journal of Nervous and Mental Disease*, *160*, 138–145.

Anderson, C., & Bushman, B. (1997). External validity of "trivial" experiments: The case of laboratory aggression. *Review of General Psychology*, *1*, 19–41.

Baer, J. S. (2002). Student factors: Understanding individual variation in college drinking. *Journal of Studies on Alcohol*, *14*, 40–53.

Barratt, E. S. (1994). Impulsiveness and aggression. In J. Monahan, & H. Steadman (Eds.), *Violence and mental disorder* (pp. 61–79). Chicago, IL: University of Chicago Press.

Ben-Porath, D., & Taylor, S. (2002). The effects of diazepam (Valium) and aggressive disposition on human aggression: An experimental investigation. *Addictive Behaviors*, *27*, 167–177.

Berman, M., & Taylor, S. (1995). The effects of triazolam on aggression in men. *Experimental and Clinical Psychopharmacology*, *3*, 411–416.

Bettencourt, B., & Miller, N. (1996). Gender differences in aggression as a function of provocation: A meta-analysis. *Psychological Bulletin*, *119*, 422–447.

Bradlyn, A., & Young, L. (1983). Parameters influencing the effectiveness of the balanced placebo design in alcohol research. In L. Pohorecky, & J. Brick (Eds.), *Stress and alcohol use* (pp. 87–103). New York, NY: Elsevier.

Brecht, M., O'Brien, A., von Mayrhauser, C., & Anglin, M. (2004). Methamphetamine use behaviors and gender differences. *Personality and Individual Differences*, *29*, 89–106.

Bushman, B., & Cooper, H. (1990). Effects of alcohol on human aggression: An integrative research review. *Psychological Bulletin*, *107*, 341–354.

Carrol, E., & Zuckerman, M. (1977). Psychopathology and sensation seeking in "downers," "speeders," and "trippers": A study of the relationship between personality and drug choice. *International Journal of the Addictions*, *12*, 591–601.

Cheong, J., & Nagoshi, C. (1999). Effects of sensation seeking, instruction set and alcohol/placebo administration on aggressive behavior. *Alcohol*, *17*, 81–86.

Cherek, D. (1981). Effects of smoking different doses of nicotine on human aggressive behavior. *Psychopharmacology*, *75*, 339–345.

Chermack, S., & Giancola, P. (1997). The relationship between alcohol and aggression: An integrated biopsychosocial approach. *Clinical Psychology Review*, *6*, 621–649.

Chermack, S., & Taylor, S. (1995). Alcohol and human physical aggression: Pharmacological versus expectancy effects. *Journal of Studies on Alcohol*, *56*, 449–456.

Collins, J. (1988). Suggested explanatory frameworks to clarify the alcohol use/violence relationship. *Contemporary Drug Problems* (pp. 107–121). Spring

Collins, J., & Schlenger, W. (1988). Acute and chronic effects of alcohol use. *Journal of Studies on Alcohol*, *49*, 516–521.

Fava, M., Abraham, M., Clancy-Colecchi, K., Pava, J., Matthews, J., & Rosenbaum, J. (1997). Eating disorder symptomatology in major depression. *Journal of Nervous and Mental Disease*, *185*, 140–144.

Fishbein, D., Jaffe, J., Snyder, F., Haertzen, C., & Hickey, J. (1993). Drug users' self-reports of behaviors and affective states under the influence of alcohol. *International Journal of the Addictions*, *28*, 1565–1585.

Friedrich, R. (1982). In defense of multiplicative terms in multiple regression equations. *American Journal of Political Science*, *26*, 797–833.

Gantner, A., & Taylor, S. (1988). Human physical aggression as a function of diazepam. *Personality and Social Psychology Bulletin*, *14*, 479–484.

Giancola, P. (2000). Executive functioning: A conceptual framework for alcohol-related aggression. *Experimental and Clinical Psychopharmacology*, *8*, 576–597.

Giancola, P., & Chermack, S. (1998). Construct validity of laboratory aggression paradigms: A response to Tedeschi and Quigley 1996. *Aggression and Violent Behavior*, *3*, 237–253.

Giancola, P., Helton, E., Osborne, A., Terry, M., Fuss, A. M., & Westerfield, J. (2002). The effects of alcohol and provocation on aggressive behavior in men and women. *Journal of Studies on Alcohol*, *63*, 64–73.

Giancola, P., & Zeichner, A. (1995). An investigation of gender differences in alcohol-related aggression. *Journal of Studies on Alcohol*, *56*, 573–579.

Giancola, P., & Zeichner, A. (1997). The biphasic effects of alcohol on human aggression. *Journal of Abnormal Psychology*, *106*, 598–607.

Goodwin, R. D., & Hasin, D. (2002). Sedative use and misuse in the United States. *Addiction*, *97*, 555–562.

Graham, K., Wells, S., & West, P. (1997). A framework for applying explanations of alcohol-related aggression to naturally occurring aggressive behavior. *Contemporary Drug Problems*, *24*, 625–666.

Gustafson, R. (1991). Aggressive and nonaggressive behavior as a function of alcohol intoxication and frustration in women. *Alcoholism: Clinical and Experimental Research*, *15*, 886–892.

Gustafson, R. (1992). Alcohol and aggression: A replication study controlling for potential confounding variables. *Aggressive Behavior*, *18*, 21–28.

Hoaken, P., Campbell, T., Stewart, S., & Pihl, R. (2003). Effects of alcohol on cardiovascular reactivity and the mediation of aggressive behaviour in adult men and women. *Alcohol and Alcoholism*, *38*, 84–92.

Hoaken, P., & Pihl, R. (2000). The effects of alcohol intoxication on aggressive responses in men and women. *Alcohol and Alcoholism*, *35*, 471–477.

Hope, K. (1975). Models of status inconsistency and social mobility effects. *American Sociological Review*, *40*, 322–343.

Ito, T., Miller, N., & Pollock, V. (1996). Alcohol and aggression: A meta-analysis of the moderating effects of inhibitory cues, triggering events, and self-focused attention. *Psychological Bulletin*, *120*, 60–82.

Judd, C., Kenny, D., & McClelland, H. (2001). Estimating and testing mediation and moderation in within-subject designs. *Psychological Methods*, *6*, 115–134.

Kelly, T., & Cherek, D. (1993). The effects of alcohol on free-operant aggressive behavior. *Journal of Studies on Alcohol*, *Suppl. 11*, 40–52.

Kosten, T. R., & Singha, A. K. (1999). Stimulants. In M. Galanter, & H. D. Kleber (Eds.), *Textbook of substance abuse treatment*, (2nd ed.). Washington, DC: American Psychiatric Press.

Licata, A., Taylor, S., Berman, M., & Cranston, J. (1993). Effects of cocaine on human aggression. *Pharmacology, Biochemistry and Behavior*, *45*, 549–552.

Low, K. G., & Gendaszek, A. E. (2002). Illicit use of psychostimulants among college students: A preliminary study. *Psychology, Health and Medicine*, *7*, 283–287.

Martin, C., Earleywine, M., Finn, P., & Young, R. (1990). Some boundary conditions for effective use of alcohol placebos. *Journal of Studies on Alcohol*, *51*, 500–505.

Martin, C., & Sayette, M. (1993). Experimental design in alcohol administration research: Limitations and alternatives in the manipulation of dosage-set. *Journal of Studies on Alcohol*, *54*, 750–761.

Moeller, F., Dougherty, D., Barratt, E., Oderinde, V., Mathias, C., Harper, A., et al. (2002). Increased impulsivity in cocaine dependent subjects independent of antisocial personality disorder and aggression. *Drug and Alcohol Dependence*, *68*, 105–111.

Moss, H. B., Salloum, I. M., & Fischer, B. (1994). Psychoactive substance abuse. In M. Hershen, R. T. Ammerman, & L. A. Sisson (Eds.), *Handbook of aggressive and destructive behavior in psychiatric patients* (pp. 175–201). New York, NY: Plenum Press.

Murdoch, D., Pihl, R., & Ross, D. (1990). Alcohol and crimes of violence: Present issues. *International Journal of the Addictions*, *25*, 1065–1081.

Parrott, D. J., & Giancola, P. R. (2004). The effect of past year heavy drinking on alcohol-related aggression. under review.

Patten, S., & Love, E. (1997). Drug-induced depression. *Psychotherapy and Psychosomatics*, *66*, 63–73.

Patton, J. H., Stanford, M. S., & Barratt, E. S. (1995). Factor structure of the Barratt impulsiveness scale. *Journal of Clinical Psychology*, *51*, 768–774.

Pernanen, K. (1991). *Alcohol in human violence*. New York, NY: Guilford Press.

Petrovic, M., Vandierendonck, A., Mariman, A., van Maele, G., Afschrift, M., & Pevernagie, D. (2002). Personality traits and socio-epidemiological status of hospitalized elderly benzodiazepine users. *International Journal of Geriatric Psychiatry*, *17*, 733–738.

Pihl, R., Peterson, J., & Lau, M. (1993). A biosocial model of the alcohol–aggression relationship. *Journal of Studies on Alcohol*, *Suppl 11*, 128–139.

Pihl, R., Smith, M., & Farrell, B. (1984). Alcohol and aggression in men: A comparison of brewed and distilled beverages. *Journal of Studies on Alcohol*, *45*, 278–282.

Richardson, D., Bernstein, S., & Taylor, S. (1979). The effect of situational contingencies on female retaliative behavior. *Journal of Personality and Social Psychology*, *11*, 2044–2048.

Richardson, D., Vinsel, A., & Taylor, S. (1980). Female aggression as a function of attitudes toward women. *Sex Roles*, *6*, 265–271.

Roizen, J. (1993). Issues in the epidemiology of alcohol and violence. In S. E. Martin (Ed.), *Alcohol and interpersonal violence: fostering multidisciplinary perspectives* (pp. 3–36). Rockville, MD: National Institutes of Health.

Selzer, M., Vinokur, A., & van Rooijen, L. (1975). A self-administered short Michigan alcoholism screening test (SMAST). *Journal of Studies on Alcohol*, *36*, 117–126.

Sobell, L., & Sobell, M. (1992). Timeline follow-back: A technique for assessing self-reported alcohol consumption. In R. Litten, & J. Allen (Eds.), *Measuring alcohol consumption*. New York, NY: Humana Press.

Tarter, R. *Drug use chart.* Unpublished instrument. University of Pittsburgh.

Taylor, S. (1967). Aggressive behavior and physiological arousal as a function of provocation and the tendency to inhibit aggression. *Journal of Personality*, *35*, 297–310.

Taylor, S., & Chermack, S. (1993). Alcohol, drugs and human physical aggression. *Journal of Studies on Alcohol*, *Suppl. 11*, 78–88.

Wiley, J., & Weisner, C. (1995). Drinking in violent and nonviolent events leading to arrest: Evidence from a survey of arrestees. *Journal of Criminal Justice*, *23*, 461–476.

Wilkinson, C. (1985). Effects of diazepam (Valium) and trait anxiety on human physical aggression and emotional state. *Journal of Behavioral Medicine*, *8*, 101–114.

Zuckerman, M., Eysenck, S. B., & Eysenck, H. J. (1978). Sensation seeking in England and America: Cross-cultural, age, and sex comparisons. *Journal of Consulting and Clinical Psychology*, *46*, 139–149.