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Effects of alcohol, initial gambling outcomes, impulsivity and gambling cognitions on gambling behavior using a video poker task

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Abstract

Drinking and gambling frequently co-occur, and concurrent gambling and drinking may lead to greater negative consequences than either behavior alone. Building on prior research on the effects of alcohol, initial gambling outcomes, impulsivity, and gambling cognitions on gambling behaviors using a chance-based (non-strategic) slot-machine task, the current study explored the impact of these factors on a skill-based (strategic) video poker task. We anticipated larger average bets and greater gambling persistence under alcohol relative to placebo, and expected alcohol effects to be moderated by initial gambling outcomes, impulsivity, and gambling cognitions. Participants ($N=162$; 25.9% female) were randomly assigned to alcohol (target BrAC = .08 g%) or placebo and were given \$10 to wager on a simulated video poker task, which was programmed to produce 1 of 3 initial outcomes (win, breakeven, or lose) before beginning a progressive loss schedule. Despite evidence for validity of the video poker task and alcohol administration paradigm, primary hypotheses were not supported. Individuals who received alcohol placed smaller wagers than participants in the placebo condition, though this effect was not statistically significant, and the direction of effects was reversed in at-risk gamblers ($n=41$). These findings contradict prior research and suggest that alcohol effects on gambling behavior may differ by gambling type (non-strategic vs. strategic games). Interventions that suggest alcohol is universally disinhibiting may be at odds with young adults' lived experience and thus be less effective than those that recognize the greater complexity of alcohol effects.

Keywords

alcohol; gambling; persistence; betting behavior; impulsivity; cognitions

Many young adults engage in risk behaviors, including alcohol use and gambling. A fair percentage of these individuals engage in heavy episodic consumption of alcohol (up to

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Disclosures

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51.3% of young adults; Naimi et al., 2003), which has been defined in various ways across studies. The National Institute on Alcohol Abuse and Alcoholism (NIAAA) defines heavy episodic drinking, or *binge* drinking, as consumption of five or more drinks for a man, or four or more drinks for a woman, within a 2-hour period, which for the average size man and woman would correspond to an estimated blood alcohol concentration (BAC) of .08g/dL, commonly expressed as .08 g% (NIAAA, 2004). A BAC of .08 g% is associated with impairment of information processing and judgment, sometimes referred to as *alcohol myopia*, as well as impairment in motor coordination (CDCP, 2015) that jointly contribute to increased occurrence of negative consequences, including academic impairment, interpersonal problems, injuries, and unplanned sexual behavior (e.g., Townshend, Kambouropoulos, Griffin, Hunt, & Milani, 2014; Wechsler, Davenport, Dowdall, Moeykens, & Castillo, 1994; Wechsler et al., 2002).

In terms of gambling, approximately 70% of young adults report any gambling in the past year (Barnes, Welte, Hoffman, & Tidwell, 2010; Welte, Barnes, Tidwell & Hoffman, 2008). Gambling includes any behavior that involves wagering something of monetary value on an uncertain outcome, and within this age group commonly involves playing lottery games, casino gambling, playing cards for money, sports betting and internet gambling (Goudriaan, Slutske, Krull, & Sher, 2009; LaBrie, Shaffer, LaPlante, & Wechsler, 2003; Wickwire et al., 2007). Like alcohol use, access to gambling plays a role in how often or what type of gambling someone may engage in (Welte, Barnes, Tidwell & Hoffman, 2009). In recent years, gambling access has increased in the United States, with only two states continuing to ban all forms of gambling (i.e., Hawaii and Utah). Like alcohol use, more frequent or intense gambling is associated with a host of negative consequences, including academic and occupational impairment, financial difficulties, strained social and familial relationships, potential legal involvement, and increased risk of suicide (Currie et al., 2006; Engwall, Hunter & Steinberg, 2004; Larimer et al., 2012). Financial losses can in turn spur further gambling, leading to a cyclical pattern of investing more money to recoup lost money (i.e., *chasing*).

Of course, these behaviors do not occur in a vacuum. Young adults who endorse symptoms of problematic gambling are also more likely to engage in binge drinking (Walker, Clark & Folk, 2010); conversely, those who report binge drinking report greater gambling frequency and severity than those who do not report binge drinking (Bhullar, Simons, Joshi, & Amoroso, 2012). About 26% of young adults indicate that they always or almost always consume alcohol when they gamble (Giacopassi, Stitt, & Vandiver, 1998). The co-occurrence of these behaviors could potentiate their individual effects, leading to greater negative consequences than either behavior alone.

Although findings are mixed (Whitton & Weatherly, 2009), prior laboratory-based alcohol administration studies have demonstrated that alcohol consumption prior to gambling contributes to greater duration of gambling, (Ellery, Stewart, & Loba, 2005), greater persistence in the face of continued losses (Kyngdon & Dickerson, 1999), larger wagers (Cronic & Corbin, 2010) and more rapid loss of funds (Phillips & Ogeil, 2007). Paradigms used across these studies, including BAC target level, inclusion (or lack of inclusion) of a placebo control, and gambling task, have varied widely, with most using games of pure

chance (e.g., simulated slot machines, high-low games). The effect of alcohol on gambling behavior on games of pure chance may differ in important ways from its effect on gambling behavior on games for which skill (e.g., knowledge of odds, past performance) may have a real or perceived impact. Of relevance to the current study, Ellery and Stewart (2014), dosing participants to a breath alcohol concentration (BrAC) of .06 g% and using a video lottery terminal (VLT) that displayed video poker, found that alcohol increased rates of risky betting (“doubling up”) among probable pathological gamblers relative to non-pathological gamblers. Of note, the VLT used by Ellery and Stewart was externally valid, in that it was an actual VLT machine similar to those found in local gambling establishments; however, the authors note that this sacrificed a measure of internal validity, as it was not possible to manipulate outcomes. Moreover, participants in the study by Ellery and Stewart were interrupted during play at 15–30 minute intervals to assess intoxication, which may have created opportunities to evaluate whether to continue gambling, potentially impacting alcohol’s effect on gambling persistence.

Within the alcohol literature, there are two theories that may explain how alcohol promotes risky gambling behavior (Bartholow et al., 2003). The Attention Allocation Model (AAM; Steele & Josephs, 1990) suggests that under conditions where both impelling and inhibiting cues are present, behavior will be determined by the relative strength of the cues. Alternatively, the Behavioral Inhibition Model (BIM; Fillmore & Vogel-Sprott, 1999) suggests that alcohol may slow inhibiting cognitive processes relative to activating cognitive processes, regardless of impelling or inhibiting cues. Thus, the AAM suggests the effect of alcohol on gambling behavior should be moderated by various factors that affect cue salience, including gambling outcomes (wins/losses), impulsivity, and gambling-related cognitions (see Cronic & Corbin, 2010, for further discussion of these individual factors), whereas the BIM suggests that impulsivity should mediate the effect of alcohol on gambling. Additionally, Prospect Theory (Kahneman & Tversky, 1979), suggests there should be a main effect of initial gambling outcomes on subsequent gambling behavior independent of the effect of alcohol, as losses are experienced as greater changes in wealth than gains of the same magnitude, making individuals more risk-seeking when they perceive a loss (Camerer, 1998; Kahneman & Tversky, 1979). Prior research suggests similar main effects on gambling behavior should be evident for impulsivity and gambling-related cognitions (see Cronic & Corbin, 2010, for further discussion).

The current study builds on prior work by using a target BrAC consistent with binge drinking (.08 g%) in comparison to placebo to control; by using a simulated video poker task that controlled outcomes, including initial wins vs. losses and overall trajectory across the gambling session (i.e., progressive loss); and by using theory to guide hypotheses. Based on prior experimental research (Cronic & Corbin, 2010; Ellery et al., 2005; Kyngdon & Dickerson, 1999), it was expected that individuals who received alcohol would persist longer on the simulated video poker task and bet more (per bet) relative to those who received placebo. Based on Prospect Theory (Kahneman & Tversky, 1979), those who initially lost were expected to persist longer and place higher wagers relative to those who broke-even or won; based on prior research, it was expected that individuals higher in impulsivity (e.g., Ginley, Whelan, Meyers, Relyea, & Pearlson, 2014) and those with stronger erroneous gambling beliefs (e.g., Delfabbro & Winefield, 2000) would persist longer and bet more

compared to those lower on these characteristics. Based on the AAM (Steele & Josephs, 1990), alcohol effects were expected to be moderated by each of the aforementioned factors, showing stronger relationships among those who received alcohol versus placebo. Finally, based on the BIM (Fillmore & Vogel-Sprott, 1999), it was hypothesized that the direct effect of alcohol on gambling behavior would be mediated by impairment of behavioral inhibition as assessed by a cued go/no-go task.

Method

Participants and Procedures

Individuals (N = 236) between the ages of 21 and 30 were recruited from two college campuses (one in the northeast and one in the southwest) and their surrounding communities. To qualify for participation, an individual had to: (1) indicate that poker was among their top three most preferred forms of gambling, (2) have played poker at least once in the past year, and (3) have consumed three drinks on at least one occasion per week during the past 3 months. Past studies of alcohol's impact on gambling have almost exclusively examined those who engage in more frequent or problematic patterns of gambling; however, the majority of young adults who gamble engage in lower, non-problematic levels of gambling. Despite this, alcohol use concurrent with gambling may increase risk for harm. Thus, inclusion criteria were set to examine how alcohol use while gambling would impact gambling behavior among the full spectrum of young adults who gamble. To minimize potential harm associated with the protocol, individuals were excluded if they reported contraindications to consuming alcohol including (1) a flushing response, (2) certain current or past medical conditions or taking certain medications, and for women, (3) pregnancy. Individuals who reported current or past participation in abstinence-oriented programs for alcohol or gambling problems were also excluded, but those with alcohol or gambling problems who chose to continue engaging in these behaviors were allowed to participate. These individuals were provided with information about their risk and treatment resources at the conclusion of the study. All study procedures were approved by the Institutional Review Boards at both Universities (Title: Cognitive Effects of Alcohol on Decision Making; Yale University #0712003338 and Arizona State University #0904003873).

Study procedures were completed in a laboratory made to simulate a bar setting. The bar laboratory includes a full bar with bar stools, a mirrored back bar with liquor bottles, a black ceiling, dimmed lighting, and a lounge seating area. Data collection involved two sessions. Female participants were required to self-administer an early detection pregnancy test at the laboratory prior to the first session, and a zero BrAC at baseline was verified using a breathalyzer. The lab session comprised beverage administration, video poker play, and completion of a cued go no-go (CGNG) task. Participants were randomized to beverage condition (alcohol or placebo) in blocks of 2 to 4 people and these individuals completed all procedures (e.g., beverage administration, gambling task) together to enhance external validity (Baer, 2002; Gupta & Derevensky, 1997; Wickwire et al., 2007). Participants were not provided with any explicit instructions to interact; however, research assistants who served as the bartenders (and were blind to condition) observed participants' interactions and

participants typically spontaneously initiated conversation with one another during beverage administration. Efforts were made to avoid having participants with known prior relationships complete data collection on the same evening, though participants were not explicitly asked if they were familiar with other participants on the night of data collection. Prior to beverage administration, participants completed the CGNG to establish baseline behavioral inhibition.

The beverage condition implemented on a given night was predetermined by a coin toss. In the alcohol condition, the volume of alcohol in each drink was adjusted based on each participant's weight and sex, with a target BrAC of .08 g%. Participants in the alcohol condition consumed mixer (diet 7-up, cranberry juice and lime juice) and 80 proof vodka at a 3:1 ratio of mixer to vodka. In the placebo condition, participants consumed the same mixer and flattened tonic water at the same 3:1 ratio; additional details about the placebo controlled beverage administration has been described in detail previously (see Cronic & Corbin, 2010). Briefly, total dose of alcohol (or placebo) was administered across three drinks. Participants had 10 minutes to consume each drink. A 15-minute absorption period followed consumption of the last drink, after which the first measure of BrAC and subjective intoxication was taken. BrAC readings and subjective intoxication assessments occurred every 15–20 minutes thereafter, until all study tasks were completed; timing of the assessments was such that participants were not interrupted during completion of the poker task or CGNG task.

Following beverage consumption, participants completed the beverage manipulation checks and were allowed to play the simulated video poker task. Regardless of beverage condition, participants were randomly assigned to one of the three initial gambling outcomes (win, breakeven, lose; see additional description of gambling outcome manipulation in measures section). As blood alcohol levels were descending, the CGNG task was re-administered to assess the impact of alcohol consumption on inhibitory control. Roughly 1 week after the first session, participants returned to the lab to complete computer-based self-report measures and interview-delivered measures. Computer-based surveys included self-report measures of impulsivity and gambling related cognitions, and interviews including the TLFB and G-TLFB.

Of the 236 participants enrolled in the study, 14 in the alcohol condition did not reach a peak BrAC of .06 g% and were therefore excluded from analyses. Participants in the placebo condition who reported that they did not believe they consumed any alcoholic drinks ($n = 7$) were also excluded, reducing the sample size to 215. Programming of the task was challenging as participants were allowed to vary their bets from 1 to 3 credits per trial and it was not possible for the program to anticipate the next bet. Thus, there were some cases in which the task did not perform as expected, resulting in the removal of additional cases: for 9 participants, the poker task crashed during play yielding unusable data; although the task was programmed to allow a full house as the best possible hand, there were 18 cases in which a participant received four of a kind or a straight flush, preventing the success of the progressive loss schedule. Although the initial gambling manipulation yielded mean values consistent with the loss, break even, and win conditions to which participants were assigned, there was some overlap across conditions. Thus, we removed 14 cases so that there was no

overlap between the three conditions (loss = 13–17 credits remaining; break even = 18–22 credits remaining, win = 23–27 credits remaining). An additional 8 participants were excluded from analyses because they played fewer than 10 trials, and therefore had insufficient play to experience progressive loss. Finally, we examined rate of loss among the remaining participants and removed 4 additional participants with unusually slow or fast rates of loss, yielding a range in the remaining sample of .20 to 1.0 credits lost per trial (Mean = .60, SD = .11). In total, 53 cases were removed based on the poker task data.

The resulting sample size for analyses was 162 (placebo = 86; alcohol = 76). This sample predominantly identified as men (74.1%) and the majority (74.8%) were college students. Typical weekly alcohol consumption was 13.72 drinks ($SD = 11.21$). The majority of participants identified as Caucasian (74.1%), with the remainder identifying as Asian (5.6%), African-American (1.2%), American Indian/Alaskan Native (0.6%), or endorsing “other” (16.0%). An additional 2.5% did not report their racial identity. Unfortunately, due to an error in programming of the computer-based survey, data on Latino/a ethnicity was not available.

Measures

Alcohol consumption—The Timeline Follow-back interview (TLFB; Sobell & Sobell, 1992) provided a retrospective assessment of alcohol use. A research assistant presented each participant with a 30-day calendar and asked for daily drinking estimates, including drinking frequency (number of drinking episodes), drinking quantity (number of standard drinks consumed each episode), and the number of hours over which each drinking episode occurred. A chart was used to facilitate reporting in standard drink units. The TLFB has shown adequate test-retest reliability ($r = .92$) and is positively associated with other indices of drinking frequency/quantity. Total consumption in the past 30-days was used as a covariate in analyses.

Gambling behavior—The Gambling Timeline Follow-back interview (G-TLFB; Weinstock, Whelan, & Meyers, 2004) is a modified version of the TLFB for alcohol, which assesses past 3-month gambling type (game played or activity engaged in), frequency (number of gambling occasions), duration (number of hours gambled per occasion), intent (amount of money an individual intended to bet over the course of one episode), risk (original stake), win-loss (net amount of money won or lost), and amount of alcohol consumed while playing. The G-TLFB has shown excellent test-retest reliability when used with pathological gamblers ($r = .73$ to $.93$) and those who report frequent gambling ($r = .75$ to $.96$). The G-TLFB is positively correlated with self-monitored daily reports of gambling ($r = .59$ to $.87$). Indices derived from the G-TLFB for the current analyses included: days gambled, average amount gambled per occasion, days gambled when drinking, and average amount gambled when drinking.

Gambling problems—The South Oaks Gambling Screen (SOGS; Lesieur & Blume, 1987) was used to measure gambling problem severity experienced within the past 12 months and has been used to differentiate between probable pathological gamblers (score of 5 or greater), sub-threshold problem gamblers (score of 3 or 4), and non-problem gamblers

(score of 2 or lower). The SOGS contains 20 scored items that have demonstrated validity and reliability among college students (Lesieur et al., 1991) and correlate highly with diagnostic criteria from the Diagnostic and Statistical Manual for Mental Disorders (DSM-IV; American Psychiatric Association, 1994). Items use either a likert-type scale (When you gamble, how often do you go back another day to win back money you lost?) or require a yes/no response (e.g., Did you ever gamble more than you intended?); however, each item is scored such that it can be worth 0 or 1 point (see Lesieur & Blume, 1987, for scoring instructions).

Gambling cognitions—The Gambling Expectancies Questionnaire (GEQ; Henderson et al., 2004) is a 50-item measure that assesses how frequently participants expect that gambling will result in positive and negative outcomes, such as “gambling makes me feel helpless” and “gambling makes me more social.” The positive and negative expectancy subscales of the GEQ have demonstrated excellent internal consistency ($\alpha = .97$ and $.98$, respectively) in a sample including college students and community members with gambling problems (Tiell, 2004).

The Gambling Attitudes and Beliefs Scale (GABS; Breen & Zuckerman, 1999) is a 35-item measure that assesses the extent to which individuals agree with various erroneous statements about gambling behaviors, including “Some people can bring bad luck to other people,” and “If I have lost my bets recently, my luck is bound to change.” The GABS has demonstrated validity and reliability ($\alpha = .90$) among college students (Neighbors et al., 2002).

Self-report impulsivity—The impulsive unsocialized sensation seeking subscale of the Zuckerman Kuhlman Personality Questionnaire III – Revised (ZKPQ-IIIIR; Zuckerman, Kuhlman, Joireman, Teta, & Kraft, 1993) consists of 19 true/false items that assess sensation seeking needs and impulsivity by asking participants to respond to items such as “I tend to change interests frequently.” The ZKPQ-IIIIR has shown good convergent validity with other widely used personality scales and produced adequate reliability coefficients (Aluja, García, & García, 2003; Zuckerman et al. 1993).

The Barratt Impulsiveness Scale-11 (BIS-11; Patton, Stanford, & Barratt, 1995) is a 30-item self-report measure used to assess three second-order impulsiveness factors: (1) motor impulsiveness, (2) attentional impulsiveness, and (3) non-planning impulsiveness, and six first-order factors including attention, cognitive instability, motor, perseverance, self-control, and non-planning. The six first-order factors were used as part of a composite variable of impulsivity in the analyses. The BIS-11 has been shown to have acceptable internal consistency within a college student population ($\alpha = .82$).

Behavioral measure of impulsivity—A CGNG task (Fillmore & Weafer, 2004) was used to assess changes in participants’ level of behavioral inhibition from baseline to post-alcohol. Participants completed 250 trials with additional practice trials prior to the test trials to ensure comprehension of the task. The pre-target cue was either a vertical or horizontal black rectangle against a white background. The go and no-go target stimuli were the colors green and blue, which filled the pre-target cue. Participants were instructed to respond as

quickly as possible to “go” targets (green) and withhold response to “no-go” targets (blue). A vertical rectangle preceded the go target on 80% of the trials and the no-go target on 20% of the trials, whereas a horizontal rectangle preceded the go target on 20% of the trials and the no-go target on 80% of the trials. Thus, individuals were cued to expect a green target when a vertical rectangle was presented. Number of inhibition failures (go responses to no-go cues) was used as the index of impulsivity in the current analyses.

Simulated poker task and initial gambling outcome manipulation—A video poker task developed specifically for this study was used to administer the initial gambling outcome manipulation and assess gambling persistence and betting behavior. The program simulated a five-card draw poker game commonly found in casinos (“Jacks or Better”). The payouts were as follows for a 1 credit bet; one pair = 1, two pair = 2, three of a kind = 3, straight = 4, flush = 6, full house = 9, four of a kind = 25, straight flush = 50. Payouts for bets of 2 and 3 credits were simply multiples of the single credit bets. The initial screen displayed five cards dealt face down and a payout schedule at the top of the screen showing the amount each winning combination was worth. Participants were prompted to place a wager to view the cards. After participants placed their wager, the cards were revealed and participants had the opportunity to select cards to “hold.” All cards that were not held were discarded and replaced with new cards. The outcome of any trial was based on the strength of the participant’s final hand.

The first 10 trials served as the initial gambling outcome manipulation. Betting was set at \$0.50 per hand for the first 10 hands to ensure that all individuals in the win, break-even, and loss conditions received approximately 125% (\$12.50; 25 credits), 100% (\$10.00; 20 credits), and 75% (\$7.50; 15 credits) of their original stake, respectively. Following the tenth trial, the game was reset and \$10 in credits was added to participants’ accounts. Participants were able to wager 1, 2 or 3 \$0.25 credits per trial for the remainder of the time they gambled. The net amount won or lost during the first 10 trials (+\$2.50, 0, or -\$2.50) was displayed on the screen throughout the remainder of the task to increase the salience of the initial gambling outcome manipulation. The remaining trials were grouped into blocks of 15 trials to allow for manipulation of the contingencies. All participants were placed on a progressive loss schedule with the amount of money returned decreasing by 20% per block. No time limit was placed on how long participants could play, though the program terminated when all credits had been exhausted. Participants were instructed that they could quit at any time. Persistence was measured as the total number of trials played and betting behavior was measured by the average amount bet per trial played.

Beverage manipulation checks—Participants completed the 2-item Subjective Intoxication (SI) questionnaire, which assesses perceived amount of alcohol consumed (i.e., number of drinks) and perceived BrAC.

Data Analytic Plan and Preliminary Analyses

Prior to conducting the primary analyses, the effectiveness of the placebo manipulation and alcohol dosing protocol were examined. The mean peak BrAC in the alcohol condition was .08 g% ($SD = .01$). Among participants in the placebo condition, the average estimated

number of alcohol drinks consumed following beverage consumption was 2.82 ($SD = 0.98$), compared to 3.30 ($SD = 0.79$) in the alcohol condition. Mean estimated BrAC after placebo consumption was 0.045 g% ($SD = 0.03$) relative to 0.061 g% ($SD = 0.03$) following alcohol consumption.

Examination of the predictor and outcome variables indicated that pre- and post-beverage CGNG failures, TLFB total drinks, the negative expectancy subscale of the GEQ and each of the G-TLFB measures were positively skewed. Although use of alternative distributions for analyses (e.g., negative binomial or Poisson) is typically preferable to transformation of variables, it is not possible to include count variables as predictors in a multi-level model within Mplus; thus, variables were log transformed. To reduce the number of variables in the models, we selected the most relevant gambling history variables with normal distributions after transformation for the outcomes of interest (average bet per typical gambling occasion when drinking for average bet and number of days gambled when drinking for total trials played). We also created composite scores for impulsivity (from subscale scores from the ZKPQ-IIIIR and BIS-11) and gambling cognitions (from subscale scores from the GABS and the positive expectancy subscale of the GEQ). Table 1 presents descriptive statistics for all continuous predictor and outcome variables and Table 2 provides bivariate correlations.

After establishing the measurement model, multilevel models were used given the nested structure of the data (participants assigned to beverage condition by group). We utilized robust maximum likelihood (MLR) estimation and missing data were handled using full information maximum likelihood (FIML) estimation in Mplus 7.31 (Muthen & Muthen, 2012). Maximum likelihood estimation with standard errors based on the first order derivatives (MLF) was used when models failed to converge using MLR. Individual level variables were included at level 1 and group level variables were included at level 2. We initially tested the main effect of beverage condition on the two outcomes of the poker task. Level 1 variables included sex, college student status, days gambled when drinking/typical amount gambled when drinking, and typical weekly drinking. Average bet on the simulated poker task was included as an additional level 1 variable when total trials was the outcome. Total trials played was divided by a constant to reduce the variance and facilitate model convergence.

First, an unconditional level-1 model (no level-1 or level-2 predictors) was tested for each outcome measure. The variance component in these models was used to determine if there was significant variability in the outcome measures related to group membership. If significant variance components were found, random group effects were included, and residual covariances between the outcomes and the random effects were included. Predictor variables were group-mean centered for main effects of level-1 variables, and grand-mean centered for main effects of the level-2 variable of beverage condition (see Enders & Tofighi, 2007). We then proceeded to test main effects of initial gambling outcomes, impulsivity, and gambling cognitions. These variables were added to the level-1 model along with the covariates. Random slopes were included as appropriate based on the level-2 variance components and residual covariances between outcomes and random effects were included.

Next, we examined cross-level interactions between beverage condition and the proposed moderating variables (i.e., initial gambling outcome, impulsivity, gambling cognitions). In these models, random slopes were modeled for the proposed moderating variables regardless of whether there were significant variance components because random effects must be included to characterize the cross-level interactions. Moderator variables were group-mean centered to facilitate interpretation of the interactions (see Enders & Tofighi, 2007). In separate models, we conducted exploratory analyses to see if sex, college student status, and days gambled when drinking/typical amount gambled when drinking moderated the impact of beverage condition.

Finally, we examined the hypothesis that alcohol effects on behavioral inhibition (CGNG performance) would mediate effects of alcohol on gambling outcomes. We first tested a model to determine if alcohol consumption led to significant impairment in behavioral inhibition on the CGNG task (controlling for baseline CGNG inhibition failures and the other covariates). Next, we added CGNG performance after alcohol consumption to the models examining the main effect of beverage condition on gambling outcomes. Tests of indirect effects using procedures outlined by Preacher, Zyphur, and Zhang (2010) were planned if effects in both prior analyses (A and B paths) were statistically significant.

Results

Main effect of beverage condition on average bet

For the outcome of average amount per bet, the variance component was not significant, $p = .96$, and the intraclass correlation (ICC) was small at $.005$. Given the lack of evidence for group-level effects, random effects of group were not included when examining main effects of the level-1 variables. In level 1 of the model, heavier drinkers, men, non-college students, and those with larger average amounts gambled when drinking tended to place larger bets, though these effects were not significant (all p values = $.05$). Though not statistically significant, the direction of effect for the level-2 predictor of beverage condition was opposite hypotheses, with individuals in the alcohol condition placing smaller average bets than participants in the placebo condition ($b = -.09$, $SE = .09$, $p = .31$).

Main effect of beverage condition on total trials played

For total trials played, the variance component was statistically significant, $p < .001$, and the ICC was substantial at $.40$. Thus, random effects of group were included when examining main effects of the level-1 variables. Because models would not converge with multiple random slopes and missing data estimation, we first ran the model with random slopes among participants with complete data ($n = 155$; 7 missing cases) and then replicated the analyses with missing data estimation and no random slopes. When random slopes were included, women, non-college students, heavier drinkers, those who typically gambled more frequently when drinking, and those who placed larger average bets on the poker task tended to persist longer on the poker task, though none of these effects were statistically significant (all p values $> .05$). The same pattern of results emerged when using the full sample without random slopes. Although the direction of effects for the level-2 predictor of beverage condition on total trials played differed across the two sets of analyses ($b = .07$, $SE = .29$, p

= .81; $b = -.02$, $SE = .29$, $p = .96$), in both cases, alcohol (relative to placebo) did not significantly impact the number of trials played.

Main effects and interactions for initial gambling outcomes, impulsivity, and gambling cognitions

Level-1 main effects for average bet—Effects for the covariates reported in prior analyses were comparable when the additional predictors were added. Participants with more inhibition failures on the CGNG task at baseline, lower levels of self-reported impulsivity, and stronger gambling related cognitions tended to place larger average bets, though only the effect of gambling related cognitions was statistically significant ($b = .17$, $SE = .08$, $p = .04$). The effect of beverage condition at level 2 remained non-significant and was virtually identical to the earlier model for average bet ($b = -.09$, $SE = .09$, $p = .35$). Figure 1 provides raw means for average bet by beverage condition and initial gambling manipulation.

Cross-level interactions for average bet—Models failed to converge when we included multiple random effects and estimation of missing data and random effects were required to test cross-level interactions. Thus, we tested cross-level interactions using models with random slopes and no missing data estimation ($n = 147$; 15 missing cases). Contrary to study hypotheses, none of the cross-level interactions between beverage condition and the proposed moderating variables were statistically significant (all p values $> .82$). Exploratory analyses examining interactions between beverage condition and sex, college status, and average amount gambled when drinking also found no evidence for moderation (all p values $> .31$).

Level-1 main effects for total trials played—Given the large ICC and significant variance component, random effects were included when examining effects of the level-1 predictors. Because random effects for the covariates were not significant in the prior model, they were not included in the model. Models with missing data would not converge even without random slopes. Thus, we relied on analyses with random slopes and no missing data estimation ($n = 147$). Effects of the covariates were similar to the prior model and remained non-significant with the inclusion of the new predictors. Participants with more baseline inhibition failures on the CGNG task, lower levels of self-reported impulsivity, and stronger gambling related cognitions tended to play more trials, though only the effect for gambling related cognitions was statistically significant ($b = .57$, $SE = .24$, $p = .02$). As in the earlier model, the level-2 main effect of beverage condition was not significant ($b = -.03$, $SE = .36$, $p = .93$). Figure 3 provides raw means for total trials played by beverage condition and initial gambling manipulation.

Cross-level interactions for total trials played—Examination of cross-level interactions between beverage (level 2) and the four proposed moderators (level 1) again focused on the sample with complete data ($n = 147$). Contrary to study hypotheses, none of the cross-level interactions were statistically significant (all p values $> .33$). Exploratory analyses examining interactions between beverage condition and sex, college status, and days gambled when drinking also found no evidence for moderation (all p values $> .60$).

Alcohol-related impairment of behavioral inhibition as a mediator of alcohol effects on gambling behavior

First, we examined effects of alcohol consumption on inhibition failures on the CGNG task after beverage administration. In the unconditional multilevel model, the ICC was substantial (.17), so we included random effects for the level-1 covariates. Consistent with prior analyses, we first tested a model with random effects among those with complete data ($n = 147$; 15 missing cases) and then replicated the analysis with missing data estimation and no random effects. In the model with random effects, participants in the alcohol condition tended to demonstrate greater impairment in behavioral inhibition (more CGNG inhibition failures) relative to placebo participants, but this effect was not statistically significant ($b = .11$, $SE = .08$, $p = .18$). In the full sample without random effects, alcohol led to greater inhibition failures on the CGNG task relative to placebo ($b = .12$, $SE = .06$, $p = .03$). Figure 3 provides raw means for pre- and post-CGNG performance by beverage condition.

Next, we added CGNG performance after alcohol to the full models for average bet and total trials played. For both outcomes, models with missing data estimation would not converge even without the random slope for the effect of the initial gambling manipulation, so we focused on models with random slopes and no missing data estimation ($n = 142$; 20 missing). Participants who reported greater impairment in CGNG performance after beverage consumption tended to place larger average bets per trial, though this effect was not statistically significant ($b = .58$, $SE = .32$, $p = .08$). Those who showed greater impairment of CGNG performance after alcohol also tended to persist longer on the poker task, but this effect was also not statistically significant ($b = 1.29$, $SE = .86$, $p = .14$). Because the effects of change in CGNG performance on average bet and trials played were not statistically significant, the full mediation model was not tested.

Post-hoc Analyses among Subthreshold and Probable Pathological Gamblers

Given the unexpected findings regarding the lack of alcohol effects, we conducted post-hoc analyses examining main effects of beverage condition on average bet and total trials played among subthreshold and probable pathological gamblers based on SOGS scores greater than or equal to 3. We used the cutoff of 3 rather than 5 (the standard cutoff for probable pathological gamblers) given the small sample of those with scores of 5 or higher ($n = 23$). For average bet ($n = 41$), participants in the alcohol condition tended to place larger average bets than those in the placebo condition, but this effects was not statistically significant ($b = .18$, $SE = .21$, $p = .39$). For total trials played, participants in the alcohol condition tended to play fewer total trials with random slopes and no missing data ($n = 39$; $b = -.16$, $SE = .82$, $p = .85$) and with no random slopes and missing data estimation ($n = 41$; $b = -.32$, $SE = .58$, $p = .58$), though effects were not statistically significant in either case.

Discussion

Although the analyses provided evidence for the validity of the beverage manipulation (i.e., predicted impairments in behavioral inhibition on the CGNG under alcohol), the validity of the simulated poker task (i.e., stronger gambling related cognitions and larger typical bets when drinking associated with higher average wagers on the task), and the validity of the

initial gambling outcome manipulation (i.e., expected differences in remaining credits by condition), they largely did not support the primary hypotheses of the study. First, there was not a significant main effect of beverage condition on either average bet or number of trials played. In fact, although neither main effect was statistically significant, participants in the alcohol condition placed smaller average bets than participants in the placebo condition in direct opposition to study hypotheses. Further, there was no evidence to support hypotheses based on the AAM that initial gambling outcomes, impulsivity, and gambling related cognitions would moderate the impact of beverage condition on average bet and number of trial played. These results directly conflict with prior research indicating that alcohol consumption leads to larger average wagers on a simulated slot-machine task (Crounce & Corbin, 2010), and to greater persistence among those who gamble more frequently (Kynndon & Dickerson, 1999) or problematically (Ellery et al., 2005); however, they are consistent with other research among non-problematic gamblers (Ellery & Stewart, 2014).

Although additional research is needed to determine if there are differential effects of alcohol on specific forms of gambling, the distinction between non-strategic and strategic forms of gambling may be an important one. Because strategic gambling may require more decision-making processes (Odlaug, Marsh, Kim, & Grant, 2011), individuals who engage in these forms of gambling may become more conservative when intoxicated due to concern about their decision-making capacity. Consistent with this possibility, Lorains and colleagues (2014) found that nonstrategic gamblers show greater evidence of deficits in decision-making than strategic gamblers. Whether video poker can objectively be considered strategic (skill-based) or not (c.f., Dixon & Jackson, 2008; Grant, Odlaug, Chamberlain, & Schreiber, 2012; Ledgerwood & Petry, 2006; Odlaug et al., 2011) may be less important than whether the individual gamblers perceive it to be skill-based (Barker & Britz, 2000). For example, using speak-aloud procedures, Walker (1992) showed that individuals were less likely to verbalize irrational thoughts when playing video poker than when playing slot machines. Thus, individuals may adjust their behavior depending on their perceptions of the demands of the game.

Results of the Ellery and Stewart (2014) study also suggest that alcohol effects on strategic gambling may be limited. Although the authors found that alcohol (relative to placebo) was associated with increased risky betting (i.e., doubling up after a winning bet) on a video poker task, they did not find alcohol effects on average bet or time spent gambling, even among pathological gamblers. Post-hoc analyses in subthreshold and probable pathological gamblers in the current sample yielded findings quite similar to those of Ellery and Stewart (2014) with respect to average bet size. Although not significant, those in the alcohol condition placed modestly larger average bets (in contrast to findings in the full sample). Taken together, results of our analyses and those of Ellery and Stewart (2014) provide limited evidence for alcohol effects on strategic forms of gambling, even among problem gamblers. However, it is important to note that power to detect effects within the sample of subthreshold and probable pathological gamblers in the current study was low given the sample size of 41.

The differences in effects for average bet between the full sample and the subsample of pathological gamblers highlight the diversity of gambling experience and problems in our

sample. Although inclusion criteria in this study were intentionally kept broad to increase generalizability of findings, future research may wish to selectively recruit regular video poker players who perceive it to be skill-based and compare the effect of alcohol on gambling behavior within this group to regular video poker players who do not perceive the game to be skill-based. Understanding for whom alcohol may negatively impact behavior leading to consequences is essential to appropriate prevention planning.

Although the effect for average bet was more consistent with hypotheses within the sample of at-risk gamblers, this was not the case for total trials played which showed near zero effects of alcohol in both the full sample and the sample of probable pathological gamblers. In addition, although stronger gambling related cognitions were related to more trials played, neither frequency of gambling behavior or gambling behavior when drinking from the G-TLFB were significantly related to the number of trials played on the video poker task (see Table 2). There were also significant group-level effects suggesting that persistence on the video poker task was based more on the behavior of other participants in the group than the participant's own typical gambling behavior. Thus, it appears that average amount bet on the video poker task provided a better reflection of internal processes governing gambling behavior in this study.

This study had a number of strengths, including use of a simulated poker task that mimicked externally valid conditions while also maintaining internally valid controls related to gambling outcome contingencies; use of a credible placebo to control for the influence of alcohol outcome expectancies; and inclusion of social gambling young adults (versus exclusively focusing on high-risk or disordered gamblers). However, it also had limitations that must be considered. First, study manipulation failures required 74 participants to be excluded. Despite the resultant loss of power, the findings suggest it is unlikely that a larger sample would have yielded results consistent with hypotheses. Second, a target BrAC of .08 g% was used in the alcohol condition, consistent with a BAC that might be obtained during a binge drinking event. Research is mixed on typical BACs individuals achieve when choosing to drink and gamble (c.f., Cronic & Corbin, 2010; Ellery et al., 2005; Focal Research, 1998). The relatively large dose in the current study may have led to high perceived impairment and consequent efforts to engage in a more conservative betting strategy. On the other hand, participants may have been exhibiting acquired tolerance and not have felt, or acted, as impaired as would a sample of lighter drinkers.

Also, aspects of the experimental setting and stimuli may limit generalizability of the findings. Specifically, participants were restricted to the \$10 provided by the experimenters, which may have created a ceiling effect on the variable of total trials played. As suggested elsewhere (Cronic & Corbin, 2010), future research could benefit from using a paradigm in which participants are provided with unlimited funds and the amount of time spent gambling is used as the dependent variable. This approach was not used in this study due to the initial gambling outcome manipulation, which required that participants perceive the \$10 as part of their *wealth*, such that wins and losses would be experienced as deviations from this set point. Another limitation of the current paradigm was displaying the final total of the initial gambling outcome manipulation for all subsequent trials. This was done to enhance the salience of the initial gambling outcome manipulation; another approach that would be more

externally valid would be to use larger dollar amounts, such that the perceived change in wealth would be greater (e.g., losing \$25 instead of \$2.50).

Finally, although potential group effects were controlled for using multilevel modeling, findings may only extend to gambling that occurs in a group context, as opposed to gambling that occurs in isolation. Furthermore, participants' familiarity with one another was not assessed in this study, and could impact an individual's willingness to gamble (e.g., if they typically gamble with a particular friend they may be more likely to gamble if that person is present). Given this, future research that specifically manipulates aspects of the social context in which gambling occurs seems warranted.

Despite these limitations, the current study adds significantly to existing knowledge regarding the effects of alcohol on gambling behavior. The findings suggest that alcohol cannot be assumed to contribute to riskier gambling behavior under all conditions. This has important implications for messages given to college students and other young adults as part of interventions targeting alcohol use and/or gambling behavior. Telling individuals that drinking when gambling will lead to more negative consequences may be directly at odds with their own lived experience if they are a social gambler who engages in video poker play or other forms of gambling perceived as strategic. Given this, future research should focus on determining the boundary conditions under which alcohol contributes to more negative gambling outcomes. This knowledge will be critical to appropriately tailor prevention and intervention content in this population and reduce public health risk associated with concurrent alcohol use and gambling.

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Public Health Statement

The results suggest that alcohol effects on gambling may differ based on the type of game (strategic vs. non-strategic) and the population under study (problem vs. non-problem gamblers). Thus, individual and public health approaches to prevention may need to be tailored to aspects of both the environment and the population of interest.

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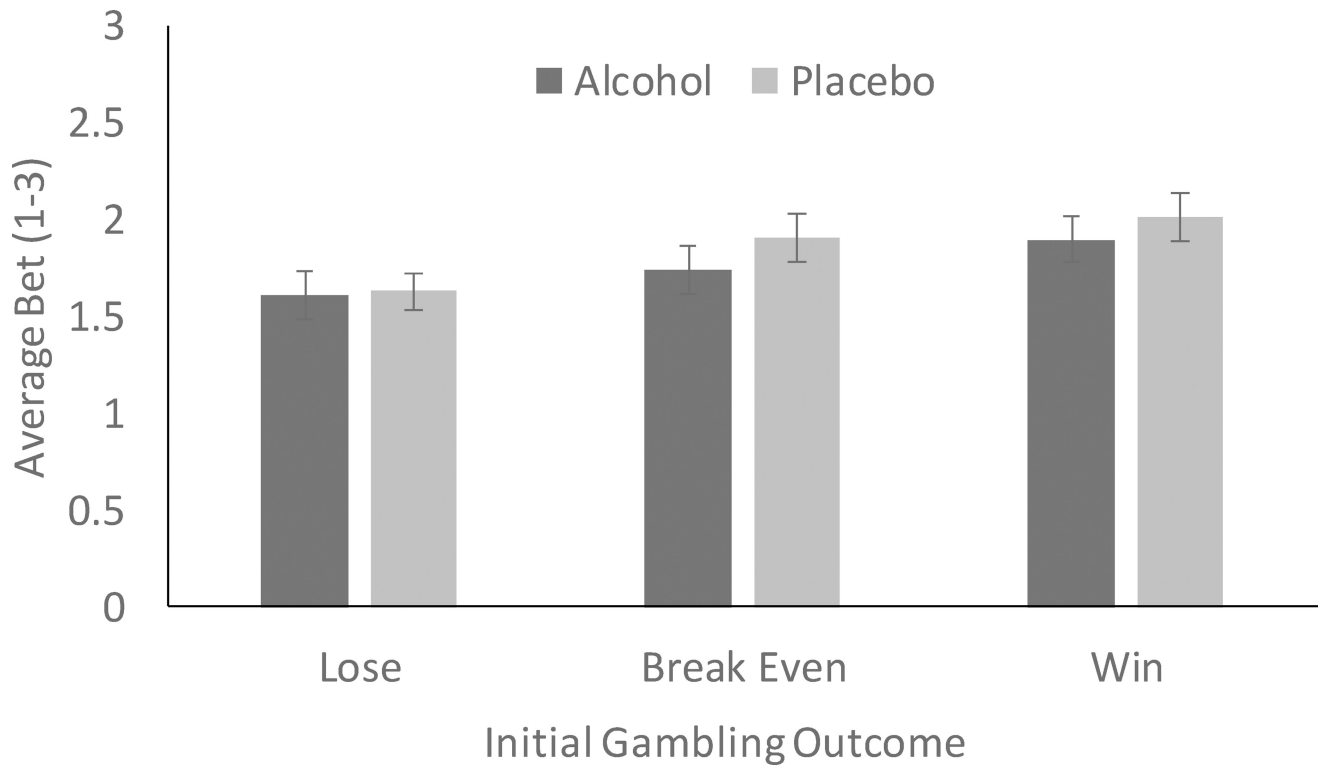


Figure 1. Raw Means (SE) for Average Bet on the Video Poker Task by Beverage Condition and Initial Gambling Manipulation

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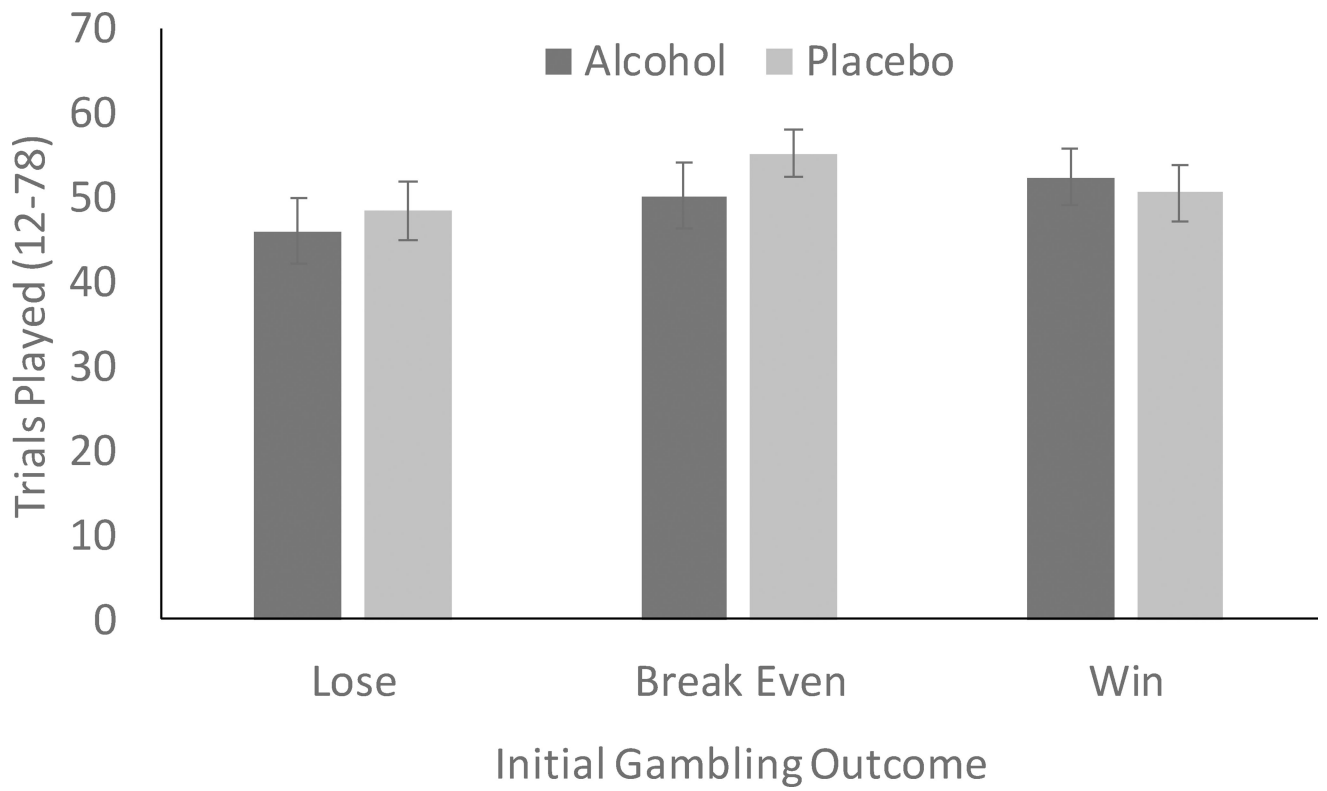


Figure 2. Raw Means (SE) for Total Trials Played by Beverage Condition and Initial Gambling Manipulation

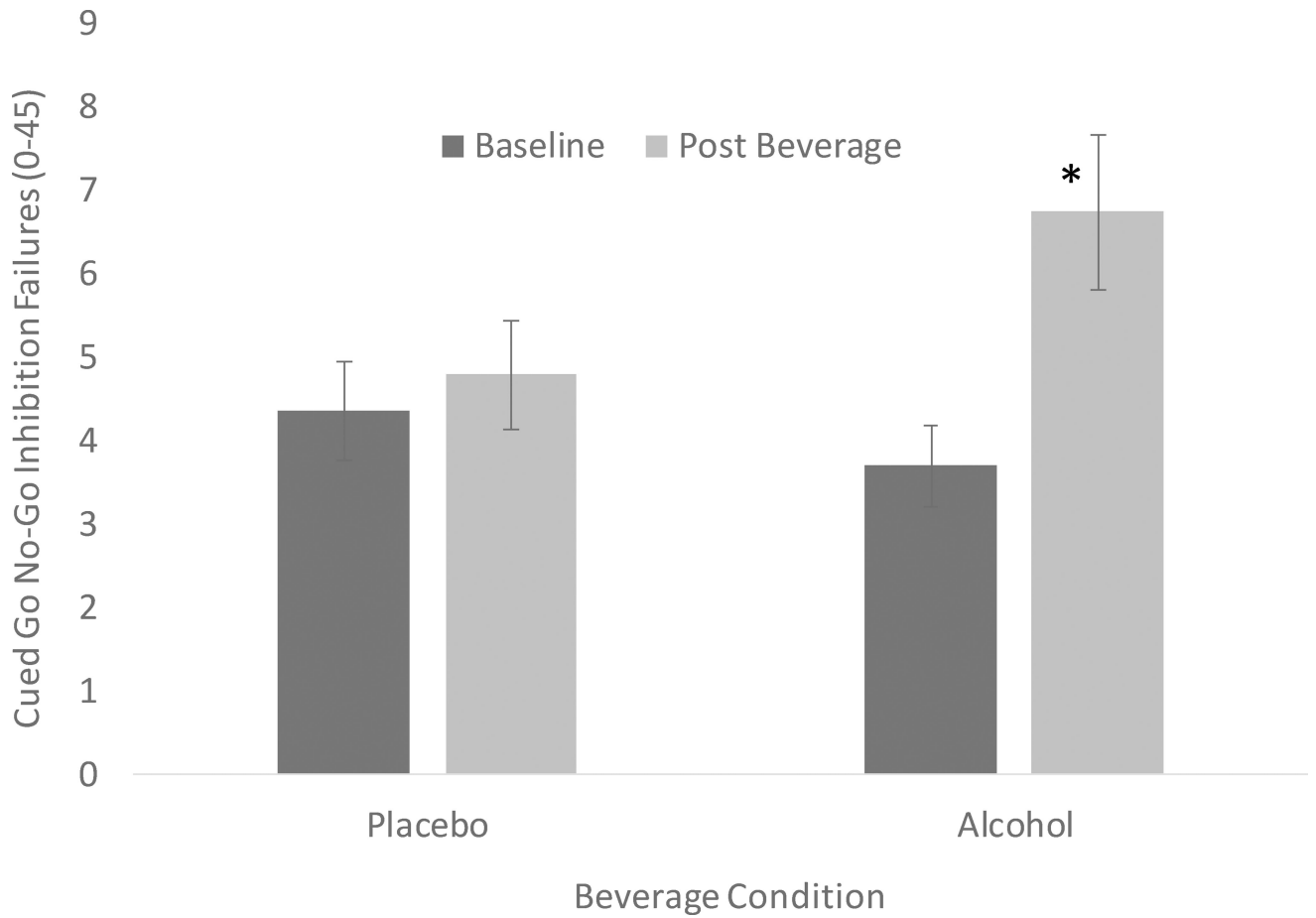


Figure 3. Raw Means for Pre- and Post-Beverage CGNG Inhibition Failures by Beverage Condition. * indicates that the within beverage condition pre-post change was statistically significant at $p < .05$.

Table 1

Descriptive Statistics for Key Study Variables by Beverage Condition

	<u>Placebo</u>		<u>Alcohol</u>	
	Mean	SD	Mean	SD
Age (years)	22.50	2.16	22.75	2.34
ZKPQ Impulsivity	2.76	2.36	3.01	2.36
ZKPQ Sensation Seeking	6.90	2.69	7.27	2.64
BIS Attention	2.12	0.56	2.15	0.58
BIS Cognitive Instability	2.27	0.66	2.21	0.61
BIS Motor	2.23	0.53	2.26	0.46
BIS Perseverance	1.80	0.45	1.87	0.54
BIS Self-Control	2.12	0.50	2.10	0.53
BIS Cognitive Complexity	2.20	0.50	2.20	0.48
CGNG Inhibition Failures	4.35	5.49	3.69	4.13
GABS	78.25	13.97	78.82	13.88
GEQ Positive	75.49	26.05	74.18	28.21
GEQ Negative	41.82	17.65	41.34	15.38
TLFB Total Drinks per Week	14.16	11.70	13.23	10.68
TLFB Days Gambled (Past 90 days)	5.82	7.32	5.33	6.13
TLFB Avg Daily Bet	74.21	161.92	45.78	170.37
TLFB Days Gambled with Alcohol	3.30	4.57	2.91	3.94
TLFB Avg amount Gambled Under Alcohol	47.38	75.00	39.35	124.39
SOGS Score	2.09	2.41	2.31	2.73

Note: ZKPQ = Zuckerman Kuhlman Personality Questionnaire–III Revised; BIS = Barratt Impulsiveness Scale-11; CGNG = Cued Go No Go task; GABS = Gambling Attitudes and Beliefs Scale; GEQ = Gambling Expectancies Questionnaire; TLFB = Timeline Follow-Back Interview; SOGS = South Oaks Gambling Screen.

Table 2

Bivariate Correlations among Predictor and Outcome Variables

	2	3	4	5	6	7	8	9	10	11	12	13
1. Sex (1 = Male)	.155	.301**	.139	.172*	.114	.212**	.222**	.195**	.211**	.137	.302**	-.088
2. College Student (1 = Yes)		.055	.127	-.036	.075	-.037	.128	.061	.167*	-.033	.021	-.049
3. TLFB Weekly Drinks			.059	.277**	-.096	.229**	.223**	.321**	.270**	.112	.208**	.060
4. Impulsivity				.227**	.282**	.092	.008	.104	.139	.331**	-.028	-.088
5. Gambling Cognitions					-.003	.394**	.325**	.379**	.298**	.434**	.195*	.158*
6. CGNG Inhibition Failures						.054	-.017	-.009	-.038	-.024	.165*	.095
7. TLFB Days Gambled							.532**	.821**	.596**	.259**	.068	-.138
8. TLFB Avg Daily Bet								.500**	.735**	.217*	.253**	-.075
9. TLFB Days Gambled/Alc									.713**	.240**	.113	-.050
10. TLFB Avg Gambled/Alc										.234**	.217**	-.056
11. SOGS Score											.162	.003
12. Average Bet												.091
13. Total Trials												

Note:

*
p < 0.05,**
p < 0.01;

Average Bet = Average bet on the video poker task; Total Trials = Total trials played on the video poker task; TLFB = Timeline Follow-Back Interview; Impulsivity = Composite of scores on the Zuckerman Kuhlman Personality Questionnaire-III-R and the Barratt Impulsiveness Scale-11; Gambling Cognitions = Composite of scores on the Gambling Attitudes and Beliefs Scale and the positive subscale of the Gambling Expectancies Questionnaire; CGNG = Cued Go No Go task.