

**The Effect of Acute Alcohol Intoxication on Alcohol Cue Salience: An Event-Related Brain
Potential Study**

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Preparation of this article was supported by Grants R01 AA025969 and F31 AA028990 from the
National Institute on Alcohol Abuse and Alcoholism, and Grant R01 AG026308 from the
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Abstract

Objective: Alcohol cue salience is considered core to the broader understanding of drinking behaviors. In the present research, we sought to build the knowledge of alcohol cue salience by exploring P3 responses to alcohol images among social drinkers within a large-scale alcohol-administration study.

Method: Participants (N=246) were randomly assigned to receive either a moderate dose of alcohol (target BAC=.08%) or a non-alcoholic control beverage. Following beverage administration, participants engaged in image-viewing tasks while EEG was recorded. We examined the impact of alcohol on the amplitude of P3 responses to pictures of alcoholic vs. non-alcoholic beverages, exploring both beverage-manipulation and individual-difference moderators of these effects.

Results: Results revealed a significant effect of acute alcohol intoxication on P3 responses across stimulus types, with the overall amplitude of P3 being significantly smaller among participants consuming alcohol vs. a non-alcoholic beverage. In addition, results revealed a significant main effect of image type, such that P3 amplitude was larger for alcohol images compared to non-alcohol images. No interactions emerged between stimulus type and beverage condition or stimulus type and AUD risk level.

Conclusions: With the aim of better understanding the potential influence of the broader context on responses to individual cues, the current study examined the perceived salience of alcohol cues within a drinking setting. Findings provide evidence for alcohol cue salience that is both robust and also widespread across drinkers. More generally, the current study's findings may offer new directions for understanding neurocognitive processes of alcohol cue salience across contexts.

Key words: cue salience, P3, alcohol administration, ERP

Public Health Significance Statement

The findings of this study offer new directions for studies using neurocognitive measures to explore alcohol cue salience and further highlight the importance of considering contextual factors in basic neurocognitive addiction science.

Introduction

An understanding of alcohol cue salience is considered core to the broader understanding of drinking behaviors (Carter & Tiffany, 1999; Cofresí et al., 2019; MacKillop & Lisman, 2008; Niaura et al., 1988; Robinson & Berridge, 1993). The salience of alcohol cues might vary markedly according to individual and contextual characteristics (Martins et al., 2019; Valyear et al., 2017; Villaruel & Chaudhri, 2016)—depending on the person and the setting, a can of beer or bottle of wine might exert little sway over awareness or, alternatively, seem to fairly leap out at the drinker and dominate attention. Enhanced alcohol cue salience has been shown to be among the more powerful immediate precipitating factors driving alcohol consumption (Kambouropoulos & Staiger, 2009), and, over the longer term, cue salience has been implicated in the development of alcohol use disorders (AUD) (Cox et al., 2002; Robinson & Berridge, 1993; Stormark et al., 1997). Thus, an understanding of alcohol cue salience has emerged as a research priority within addiction science.

Although researchers have long viewed cue salience as being core to the understanding of AUD, capturing processes involved in the automatic evaluation of environmental cues has involved formidable methodological challenges. More specifically, the attentional processes involved in the encoding of environmental cues can often occur at a level beyond conscious thought (Krank & Wall, 2006), difficult to assess via self-report. Thus, the understanding of these processes has required a broad methodological toolkit. Measures with the capability of directly assessing brain activity, such as event-related brain potentials (ERPs), can be especially valuable for capturing such processes, with the P3 component in particular having proven a potent tool for building an understanding of alcohol-related cognition (see Fairbairn et al., in press).

The P3 (here used to more specifically refer to the P3b) is a positive-going waveform feature that peaks after about 300 ms post-stimulus-onset (Donchin, 1981; Polich & Kok, 1995). It is commonly elicited by an “oddball” task, in which participants are asked to monitor a stream of stimuli for infrequent targets. P3 amplitudes are notably affected by probability, such that stimuli that occur less frequently over time elicit larger (more positive) P3 amplitudes compared to those that occur more frequently (Polich, 2007). P3 amplitudes are also modulated by the amount of attentional resources recruited for the evaluation of stimuli (Lang, Bradley, & Cuthbert, 1997; Schupp et al., 2000), including alcohol-related cues (Bartholow et al., 2007; Namkoong, Lee, Lee, Lee, & An, 2004). Prior research suggests that when low risk social drinkers are shown images of alcohol, the amplitude of their P3 response tends to be no larger than that elicited when they view images of non-alcoholic beverages (Bartholow et al., 2010; Bartholow et al., 2007; Herrmann et al., 2001; Martins et al., 2019). However, it has also been found that individuals at risk for developing an alcohol problem—e.g., those with low alcohol sensitivity (Bartholow et al., 2007, 2010)—display larger P3 responses to (equally probable) images of alcoholic beverages versus non-alcoholic beverages, suggesting that these individuals are allocating more attention to alcohol-related stimuli. Such observations have led researchers to suggest that P3 amplitude might represent an individual-level marker for AUD risk.

In addition, alcohol consumption itself has been shown to alter a variety of cognitive processes (Cohen et al., 1997; Field et al., 2010), including working memory and attention (Harvey et al., 2013a, 2013b; Weissenborn & Duka, 2003) as well as motivational states (Koob, 2004; Wang et al., 2015). Acute intoxication has been theorized to impact the cognitive processing of alcohol-related cues (Korucuoglu et al., 2015), thus potentially fueling more alcohol consumption (i.e., drinking begets drinking). Some previous research using behavioral

markers (e.g., eye-tracking, response time) of attentional bias supports this notion that acute intoxication induces alcohol-related cognitive biases (Adams, Ataya, Attwood, & Marcus, 2012; Schoenmakers, Wiers, & Field, 2008). At the same time, other studies suggest that acute administration of alcohol temporarily reduces attentional bias to alcohol cues, possibly because alcohol consumption satiates the motivation to drink (Duka & Townshend, 2004; Roberts and Fillmore, 2015; Monem & Fillmore, 2019). No prior study has used ERP to directly explore how the brain responds to alcohol cues in a drinking setting, nor has prior research examined the impact of alcohol consumption on the amplitude of P3 responses to alcohol cues (see Fairbairn et al., in press). The current study seeks to address this question.

The Current Study

In the present research, we sought to build the understanding of alcohol cue salience by exploring P3 responses to alcohol images among social drinkers within a large-scale alcohol-administration study. In the context of alcohol-administration trials, participants in the experimental condition are assigned to receive a dose of alcohol, whereas participants in both experimental and control/placebo conditions are exposed to a range of cues that might prime attention towards alcohol. In the current research—a study that is, to our knowledge, the largest ERP alcohol-administration study conducted to date (see Fairbairn et al., in press)—we examined the impact of alcohol on the amplitude of P3 responses to pictures of alcoholic vs. non-alcoholic beverages, exploring both beverage-manipulation and individual-difference moderators of these effects. Building on related prior research (e.g., Adams et al., 2012; Bartholow et al., 2007, 2010; Fairbairn et al., in press; Korucuoglu et al., 2015; Schoenmakers et al., 2008), we made three predictions. First, we hypothesized that we would observe a main effect of alcohol on P3 amplitude, such that alcohol consumption would reduce P3 amplitude

across image categories and participants. We further predicted that if environmental signals linked to alcohol consumption indeed increase the salience of alcohol, we would observe an effect of image type on P3 amplitude, such that P3 amplitudes would be larger in response to alcoholic vs. non-alcoholic beverage images. If high levels of environmental cuing are required to obtain this increased salience, then it is possible we would see image type effects only in participants that consumed alcohol. However, if cuing effects are relatively strong, then we should observe a main effect of image type on P3 amplitude across all participants and beverage groups, since all participants were exposed to alcohol-related stimuli. Finally, we hypothesized interactions between individual difference criteria denoting risk (e.g., typical alcohol consumption, alcohol problems, family history of AUD) and P3 responses to alcoholic vs. non-alcoholic beverage images, such that P3 amplitudes in response to alcoholic vs. non-alcoholic beverage images would be larger among individuals at higher risk. In addition to these, we examine interactions between alcohol consumption and individual risk criteria, as well as three-way interactions between alcohol consumption, individual risk, and image type. As relatively little prior research has explored interactions between individual risk and alcohol consumption, we have no firm hypotheses related to these specific interactions, and so our analyses of these questions are primarily exploratory in nature.

Method

Participants

Participants consisted of 246 healthy social drinkers, aged 21 to 30 ($M=22.04$, $SD=1.61$) recruited using the following approaches: (1) posted flyers in super-markets, restaurants, bars, and housing developments in the local community; (2) in-person distribution of flyers outside of

local bars and restaurants; (3) social media advertisements; and (4) the University's mass email announcements. Of the 246 participants, 108 were male and 138 were female. Of participants, 66.7% were White/Caucasian, 7.3% were African American, 23.6% were Asian, 2.4% were multi-racial. In addition, 16.7% were Hispanic (see Table 1). Participants reported drinking on average 8 to 9 times in the past 30 days and consuming 4.15 ($SD=2.06$) drinks per occasion. All participants had normal or corrected-to-normal vision, were free of psychiatric or neurological disorders, did not have prior history of skull fracture, and were right-handed. Participants were required to report drinking alcohol regularly (at least 2 drinks on at least one occasion per two weeks, or at least four drinks on at least one occasion per month, over the past 12 months). Exclusion criteria included: a diagnosis of alcohol use disorder as indexed by the Diagnostic and Statistical Manual of Mental Disorders (5th 16 ed.), having been admitted into a residential treatment for substance use disorder, reported use of a psychoactive drug or depressant/sedative in the past 30 days, pregnancy, or reported discomfort with study drinking requirements. Individuals who reported a body mass index less than 18 or greater than 35 were also excluded, due to alcohol dosing requirements.

Data for this study were derived from a parent trial exploring the effect of social contextual factors on alcohol response, for which recruitment is ongoing (NCT03449095). In line with the goals of the parent study (unrelated to the aims of this report), friend referrals were also required for study participation. This study involves an EEG task completed after participants concluded the beverage-administration phase of the study—the phase during which the main aims of the parent project were explored. A sensitivity power analysis was conducted prior to data analysis, suggesting that the current sample size offered 80% power to detect within-between factors interactions that were small in magnitude (Cohen's $f=.09$), assuming $\alpha=.05$.

Procedures

Pre-Beverage Administration Procedures

The current study's protocol and procedures were approved by the institutional review board of the University of Illinois at Urbana-Champaign. Eligible participants were invited into the laboratory for an experimental session. Upon their arrival in the laboratory, participants' height, weight, and breath alcohol concentration (BrAC) were assessed. After ensuring a 0.00% BrAC, participants signed a consent form and then completed various baseline self-report assessments including those measuring mood, personality, and drinking history.

Beverage Administration Procedures

Upon completion of self-report questionnaires, participants next engaged in beverage administration procedures, during which they were randomly assigned to receive either a moderate dose of alcohol or a control beverage. A placebo condition, in which participants are given a non-alcoholic beverage but informed that they are receiving alcohol (Balodis et al., 2011), was not used in this study due to unanticipated compensatory effects observed with placebo manipulations in this and similar paradigms (Fairbairn et al., 2015; Testa et al., 2006). In line with the aims of the parent study, participants consumed their study beverages in group context. Alcohol participants received a dose of alcohol intended to achieve a peak BrAC approximately equal to the legal driving limit (.08%), with the precise amount of alcohol adjusted for gender, height, age, and weight (for formulas used in calculating total body water as used in this study, see Watson et al., 1981). Control participants received an isovolumic amount of a non-alcoholic beverage. Participants received their study beverages in 3 equal parts over the course of 36 minutes. Immediately upon completion of the beverage administration procedures,

participants' BrACs were collected and they completed self-report measures of mood and social experience.

Alcohol Image Viewing Task

After the beverage administration period, participants were brought into another room in which they completed tasks involving the viewing of images while EEG was recorded.

Participants were assigned to engage in this task at the end of the drinking portion of the study either immediately following beverage administration or, for 44 participants, after an approximate 65-minute absorption period. The main alcohol image viewing task, which was completed immediately following the completion of another brief (10 minute) picture task unrelated to the aims of this study, employed procedures similar to those used in Bartholow, Henry, and Lust (2007). A visual oddball task was used to present relatively infrequent alcohol and non-alcohol beverage target pictures among standards made up of neutral context pictures. The neutral context pictures¹ (60 total) were drawn from the International Affective Picture System (Lang et al., 2008) and included household objects such as a towel, a hairdryer, a cloth hanger, etc. According to Lang et al. (2008), these neutral context pictures had a mean valence rating of 5.06 (SD=0.42) and a mean arousal rating of 3.03 (SD=0.62) (1-9 scales). The alcohol beverage pictures (8 total) included beer bottles, a shot glass, a tequila bottle, a gin bottle, a rum bottle, a glass of wine, a pitcher of beer, and a depiction of several types of alcoholic cocktail. The non-alcohol beverage pictures (8 total) were several types of juice in glasses (e.g., orange, apple, lemonade), a glass of milk, a cup of coffee, a bottle of water, and a sports drink. None of the beverage pictures contained people in order to prevent emotional responses that might

¹ IAPS numbers for the images: 7002, 7004, 7006, 7010, 7020, 7025, 7030, 7031, 7034, 7038, 7044, 7050, 7053, 7054, 7055, 7056, 7058, 7060, 7080, 7090, 7110, 7150, 7170, 7175, 7179, 7190, 7192, 7205, 7211, 7217, 7224, 7233, 7234, 7235, 7236, 5030, 5390, 5395, 5471, 5500, 5510, 5520, 5530, 5533, 5534, 5535, 5731, 7095, 7096, 7100, 7140, 7160, 7161, 7182, 7183, 7184, 7185, 7186, 7187, 7700.

confound with participants' reactivity to beverage cues (Stritzke et al., 2004). All images were presented in the center of a 21" CRT computer monitor at a viewing distance of 100 cm. Images subtended 4.3 degrees of vertical visual angle and 6.5 degrees of horizontal visual angle.

The Alcohol Image Viewing Task consisted of 3 blocks, between which participants were given a short break (1-3 minutes). Each block contained the above-mentioned 76 images presented in random order. In addition, each block used a different random order of the same set of images, and the order of the blocks was counterbalanced. Each trial began with a series of three plus signs (+++) appearing in the center of the screen for 1000 ms to alert the participant that a trial was about to begin. The stimulus was presented in the center of the screen for 1000 ms. At this point, participants were required to respond if they saw a beverage target by pressing a response button in one hand for an alcohol beverage picture and a button in the other hand for a non-alcohol beverage picture. Participants were instructed to do nothing when any other kind of image was presented. Response hands were counterbalanced across participants. There was an interval of 500 ms before the next trial began. Participants were instructed to maintain central fixation throughout the experiment and try to minimize facial muscle movements such as blinking and squinting. To prevent fatigue, the experiment was divided into three equal blocks, each lasting around 4 minutes, with a short break in between each block. The order of the three blocks was counterbalanced.

After the completion of the image viewing tasks, participants engaged in additional study tasks (unrelated to this report), following the completion of which participants assigned to the control condition were paid and allowed to leave. Participants assigned to the alcohol condition remained in the laboratory until their BACs dropped below .025%.

Electrophysiological Recording and Data Processing

Participants' EEG was recorded while they were completing the beverage oddball task. To reduce setup time and thereby maximize the opportunity to record responses before the descending limb of the BrAC curve, we used a small, targeted electrode array consisting of 4 silver/silver-chloride electrodes arranged along the scalp midline at approximately Fz, Cz, Pz, and Oz on the 10-20 system. Electrodes were held in place using an elastic cap (EasyCap, Germany). All scalp electrodes were referenced online to the left mastoid and re-referenced offline to the average of the right and the left mastoids. In addition, one electrode was placed between the left infraorbital ridge and the left outer canthus to monitor for eye movements and blinks. Electrode impedances were kept below 5 k Ω . The continuous EEG was amplified using Sensorium amplifiers through a bandpass filter of 0.02–100 Hz and recorded at a sampling rate of 250 Hz. A baseline acquired over the 100 ms prior to target onset was subtracted before averaging, and the data were subjected to a 30 Hz low-pass digital filter (3rd order Butterworth). A digital bandpass filter of 0.2 to 20 Hz was employed prior to statistical analyses.

Oddball P3 effects are generally most prominent over parietal electrode sites (see Figure 1), so analyses were conducted at the middle parietal electrode site (Pz). Following Bartholow et al. (2007), we measured mean amplitudes between 300-800 ms after stimulus onset – i.e., a time window wide enough to capture effects despite variable peak timing of the P3. For targets (beverage images), only trials followed by a correct button press were included.

To account for the clustering of observations within individuals, we used multilevel models employing restricted maximum likelihood estimation (Bresin & Fairbairn, 2019; Bresin & Verona, 2016; Volpert-Esmond et al., 2018). In the context of these models, within-subject factors were examined at level 1 (e.g., Stimulus type), whereas between-subject factors were examined at level 2 (e.g., Beverage group, Individual-level risk). Stimulus type and Beverage

group were entered into models as dummy variables, and individual-level risk variables were entered into models uncentered. Due to the dyadic nature of clusters at level-1, slopes were estimated as fixed at level-2 and the clustering of observations is captured via the random intercept term (Kenny, Kashy, & Cook, 2020). Main effects of Beverage group (Alcohol, Control) and Stimulus type (Alcohol beverage pictures, Non-alcohol beverage pictures) and their interaction, as well as the 3-way interactions of Beverage group, Stimulus type, and self-reported individual factors (e.g., typical alcohol consumption, binge drinking; see *Drinking Risk Measures* for the full list) were assessed. An unstructured covariance matrix was used to account for correlated responses within subjects, and degrees of freedom were estimated using the between-within method. All models were run in SAS 9.4 using PROC MIXED.

Given the well-known restrictions of classical null hypothesis significance testing (Dienes, 2014; Quintana & Williams, 2018), Bayes factor (BF) analyses were conducted to further test for the degree of support for the above planned analyses. BF analyses were run using the `anovaBF` function of the `BayesFactor` package in R. Bayes factor analysis is a statistical method for assessing the relative evidence for the null vs. alternative hypothesis, with a BF less than 0.1 considered as strong evidence for the null hypothesis, and a BF greater than 10 considered as strong evidence in favor of the hypothesis (Rouder et al., 2009).

Drinking Risk Measures

The following self-report measures were used to examine individual-level risk for AUD. These specific measures were chosen to be in line with those used in prior research on alcohol cue salience (Bartholow et al., 2007, 2010).

Typical Alcohol Consumption. Participants completed a number of self-report items assessing the frequency and quantity of alcohol consumption (Armor et al., 1978; Fairbairn et al.,

2018): a) Drinking Days: Participants were asked to report on how many days out of the past 30 they had consumed any alcohol; b) Drinking Quantity: Participants were asked to report, on the days that they did drink alcohol, how many drinks they consumed on average per drinking occasion.

Binge Drinking. Participants indicated how often they engaged in binge drinking in the past 30 days (4+ standard drinks in a sitting for women, 5+ standard drinks for men).

Alcohol Problems. Participants completed the Short Inventory of Problems (SIP), a 15-item self-report measure that assesses drinking problems across physical, interpersonal, intrapersonal, impulse control, and social responsibility domains. Participants responded to each item on a 4-point (0-3) scale, from which a total score (0-45) was calculated (Miller et al., 1995). Cronbach's alpha indicated an acceptable internal consistency of .77.

Family History of Alcoholism. We assessed familial risk for alcoholism using a modified version of the Children of Alcoholics Screening Test (CAST-6; Hodgins et al., 1993), a 6-item self-report measure for identifying children at risk for developing alcoholism or other emotional and behavioral problems due to their parents. A total score (0-6) was computed based on the number of items endorsed by the participant. Three or more “yes” answers are typically employed as the threshold for considering that the respondent is a child of an alcoholic (Hodgins et al., 1993). Cronbach's alpha indicated good internal consistency ($\alpha=.83$).

Results

Beverage Manipulation Check

Among participants in the alcohol condition ($n=124$), average BrAC immediately prior to the EEG recording was 0.071% ($SD=0.017$). BrAC peaked just after the image viewing task ($M=0.079\%$, $SD=0.015$), indicating that on average the entire task was completed as participants

approached peak BrAC. Participants who completed the alcohol image viewing task after an absorption period reached a BAC level of 0.072% (SD=0.015) at the midpoint of the task, whereas those who did the task right after the group drink procedure reached an average BAC level of 0.075% (SD=0.012) at the midpoint of the task. Of note, the mean BAC levels among those who completed the image viewing task immediately after the drink period vs. after an absorption period did not differ significantly from one another, $t(122)=-1.03$, $p=0.304$. During the control session, all participants registered 0.00% BrAC after the drink period.

Behavioral Results

Nine participants were not able to complete the alcohol image viewing task due to technical issues. Therefore, behavioral results are based on the remaining 237 participants. On average, participants correctly responded on 92.79% of trials.² Response accuracy was somewhat lower for Alcohol participants (91.20%) compared with Control participants (94.32%), although this difference did not reach significance, $t(116)=-1.906$; $p=.058$. In addition, response accuracy did not significantly differ across stimulus types, $t(236)=0.318$; $p=.751$, 93.00% for alcohol beverage images, 92.56% for non-alcohol beverage images.

ERP Analyses

Prior to ERP analyses, trials contaminated by blocking, signal drift, or lateral saccadic eye movements were rejected. Three participants were excluded due to high trial loss (resulting in fewer than 15 items for any critical bin); therefore, analyses are based on the remaining 234 participants, 114 from the Alcohol group and 120 from the Control group. Trials containing blinks were corrected for the 111 participants who had enough blink and non-blink trials to produce a reliable filter (see Dale, 1994, for the procedure); blink trials were removed for the

² Response errors included incorrect button responses and miscategorization (button response for non-target stimulus and no button response for target stimulus).

remaining 123 participants. Average trial loss was 12.02%. Figure 1 shows the grand average ERPs for the Control and Alcohol groups at the four scalp channels. Both groups show a clear oddball effect, with larger P3 responses to target beverage images ($M=5.497$, $SD=5.004$) than to standards ($M=-1.297$, $SD=3.377$), $t(233)=-24.721$; $p<.001$. In addition, P3 was the largest and most distinguishable over posterior electrode sites.

There was a main effect of Beverage group, $b=-2.332$, $SE=0.634$, $t=-3.68$, $p < 0.001$ (Figure 2). Specifically, the amplitude of the P3 component was significantly smaller for participants in the Alcohol group ($M=4.30\mu V$, $SD=4.77$) than in the Control group ($M=6.71\mu V$, $SD=4.98$). This alcohol effect was invariant across participants who completed the alcohol picture viewing task immediately after the beverage administration period vs. after the absorption period, $b=1.033$, $SE=1.464$, $t=0.71$, $p=0.481$. This effect remained robust after controlling for gender, race, age, and ethnicity, $b=-2.276$, $SE=0.636$, $t=-3580$, $p = 0.004$.

Visually, both groups elicit larger P3 responses to alcohol beverage targets than to non-alcohol beverage targets (Figure 1). Analyses focused on the responses to the two beverage target types. These revealed a main effect of Stimulus type, $b=1.109$, $SE=0.227$, $t=4.89$, $p < 0.001$. The amplitude of participants' P3 responses was significantly larger in response to alcohol beverage images ($M=6.05\mu V$, $SD=5.20$) vs. non-alcohol beverage images ($M=4.94\mu V$, $SD=5.39$). This effect remained significant after controlling for gender, race, age, and ethnicity, $b=1.109$, $SE=0.227$, $t=4.89$, $p < 0.001$.³

³ Of the 246 participants who enrolled in this study, 124 completed the beverage administration procedures in an unfamiliar group drinking context, and the other 122 completed the procedures in a familiar group setting (see Methods section for aims of the parent study). Analyses revealed minimal impact of parent study manipulations on response to EEG tasks administered after the completion of group drinking procedures. More specifically, social familiarity did not significantly moderate the main effect of Stimulus type, $b=0.572$, $SE=0.451$, $t=1.27$, $p = 0.206$, or Beverage group, $b=1.194$, $SE=1.267$, $t=0.94$, $p = 0.347$.

The interaction between beverage group and stimulus type was non-significant, $b=-0.416$, $SE=0.455$, $t=-0.91$, $p=0.362$. Results indicate that, whether consuming alcohol or not, participants demonstrated higher neural indicators of cue salience for alcohol vs. non-alcohol beverage images. Specifically, P3 amplitude was larger in response to alcohol beverage pictures vs. non-alcohol beverage pictures both within the Alcohol condition, $b=0.895$, $SE=0.352$, $t=2.55$, $p=0.012$, and also within the Control condition $b=1.311$, $SE=0.289$, $t=4.54$, $p<0.001$. In addition, in cases where results were non-significant, Bayes factor (BF) analyses were conducted to test for the degree of support for the null effect. These analyses returned weak support for the hypothesis that there was no significant interaction effect between beverage group and stimulus type on P3 amplitudes ($BF=0.754 \pm 1.68\%$). Thus, the combined results support the interpretation that the interaction between beverage group and stimulus type on P3 amplitudes is not significant.

Interactions with Individual Difference Factors: In light of prior research indicating associations between individual-level risk factors and P3 alcohol cue salience (e.g., alcohol sensitivity; Bartholow et al., 2007, 2010), we examined interactions between beverage group, stimulus type, and AUD risk levels assessed in this study (see Table 1 for descriptive statistics). First, we conducted 2-way interactions between stimulus type and individual difference factors, exploring whether alcohol vs. non-alcohol beverage picture reactivity differed according to AUD risk levels. Next, we conducted analyses examining 2-way interactions between beverage group and individual difference factors, exploring whether alcohol-related reductions in P3 amplitude varied according to AUD risk levels. Finally, we constructed models integrating 3-way interactions between beverage group, stimulus type, and individual differences, exploring whether the effect of alcohol condition on alcohol-related cue reactivity varied according to

AUD risk levels. There was a significant 2-way interaction between beverage group and quantity of alcohol used, $b=-0.656$, $SE=0.256$, $t=-2.56$, $p=0.011$. To further test for the degree of support for this effect, BF analyses were conducted. The analysis returned weak support for the null hypothesis that there was an interaction effect between beverage group and quantity of alcohol used on P3 amplitudes ($BF=1.846\pm 0.94\%$) (Rouder et al., 2009). Thus, the combined results suggest that the interaction effect between beverage group and quantity of alcohol used on P3 amplitudes may not be significant. No other individual-difference risk factors interactions reached significance. See Table 3 for full results.

Discussion

The current study is the first to examine neurocognitive measures of alcohol cue salience in the context of alcohol administration. Specifically, within a study that is (to our knowledge) the largest alcohol-administration study to examine ERPs, we explored the effect of alcohol administration, beverage picture type, and AUD risk level on P3 amplitude. Results revealed a significant effect of acute alcohol intoxication on P3 responses across stimulus types, with the overall amplitude of P3 being significantly smaller among participants consuming alcohol vs. a non-alcoholic beverage. In addition, results revealed a significant main effect of image type, such that P3 amplitude was larger for alcohol images compared to non-alcohol images. No interactions emerged between stimulus type and beverage condition or stimulus type and AUD risk level—individuals at both high and low risk for AUD, as well as individuals assigned to both alcohol and control conditions, all showed higher P3 amplitude in response to alcohol vs. non-alcohol beverage images.

A notable finding of the present research is that increased P3 amplitude in response to alcohol vs. non-alcohol beverage images was observed across all participants, including heavier

drinkers and lighter drinkers as well as those with and without a family history of AUD. This finding stands in contrast to some prior P3 research, which has indicated increased alcohol cue salience only among heavier drinkers or individuals at high-risk and not among lighter drinkers or those at low-risk (Bartholow et al., 2007, 2010; Herrmann et al., 2001; Martins et al., 2019). Of note, the current study is the first to explore neurocognitive markers of alcohol cue salience within a “drinking setting”—a setting containing multiple independent cues to alcohol consumption. Prior research indicates that alcohol-related stimuli prevalent in the contexts of drinking have the potential to prime an altered attentional state, even if an individual might not be drinking in the moment (Janak & Chaudhri, 2010). In the present study, participants in both alcohol and control conditions were recruited specifically for an experiment advertised as an “alcohol study,” were prompted to provide multiple breathalyzer readings, were asked to report on their level of alcohol intoxication, and engaged in beverage-administration procedures (mixer alone or alcohol+mixer). The specific nature of alcohol cues encountered by participants in this study diverged from those found in some everyday drinking contexts (e.g., breathalyzers vs Budweiser signs). Nonetheless, similar to everyday drinking contexts, alcohol cues were prevalent in our experimental drinking context and had high potential to prime an altered attentional state. One possible interpretation of these findings is that individual differences in neurocognitive responses to alcohol cues emerge selectively in contexts where situational cues are themselves less strong, but tend to disappear in the context of settings containing multiple potent alcohol stimuli. Thus, when considered together with prior research, results of the current study indicate that individual differences in alcohol cue salience might emerge as a function of interacting characteristics of the person and the context.

Beyond the specific nature of cues themselves, researchers have been interested in alcohol's broader impact on the P3 as a way to better understand alcohol's effects on attentional processes (Gray et al., 2004; Pritchard, 1981). Alcohol can affect cognitive and attentional processes in a manner that can lead to serious negative consequences for the drinker. In particular, alcohol-impaired driving fatalities account for 31 percent of overall driving fatalities in the U.S. (National Center for Statistics and Analysis, 2016). Although a number of prior studies have examined alcohol's impact on the P3, sample sizes have been quite small, with most studies recruiting around 30 participants, and the largest study recruiting 148 participants (Fairbairn et al., in press). Perhaps unsurprisingly in light of these smaller samples, results have tended to be mixed, with some studies finding a significant reduction in P3 amplitude with alcohol consumption (e.g., Bartholow et al., 2012; Stock et al., 2014; Wolff et al., 2018), and some finding no significant effect (e.g., Curtin & Fairchild, 2003; Ehlers et al., 1998). In the current study, using a large participant sample, we reveal a significant, moderate reduction in P3 amplitude with alcohol, potentially pointing to one neurocognitive mechanism underlying alcohol-related impairment.

Limitations and future directions should be noted. First, the current study employed only one dose of alcohol—a “moderate” dose. Future studies should examine whether effects generalize to higher or lower doses. Second, due to concerns about potential unanticipated compensatory effects associated with placebo manipulations (Testa et al., 2006), the current study did not employ a placebo beverage condition but rather employed a no-alcohol control comparison. Nonetheless, the absence of a placebo condition means our paradigm is unable to isolate expectancy-related effects on P3 amplitudes. Future research might consider ways to parse pharmacological from expectancy effects. Third, the picture-viewing task used in the

current study may be limited in its sensitivity to detect between-subject differences. Although we have the largest sample size of any ERP alcohol-administration study, studies of between-subject moderators often require extremely large sample sizes to detect even moderate sized effects (Judd et al., 2001, 2017). Although no support was found for alcohol use or family history variables as moderators of P3 responses to alcohol images in the current study, there may be room yet for other individual-level as well as situational-level risk factors to moderate the salience of alcohol cues indexed by the P3. Therefore, future studies may investigate associations between P3 alcohol cue salience and potential risk factors, with even larger samples. Fourth, in the current study we selected a between-subject design due to concerns about potential carryover effects (Martin & Sayette, 1993). A replication of the study in the context of a within-subjects design may provide stronger evidence for the relative importance of individual-level vs. contextual factors in alcohol cue salience. Lastly, as discussed above, it may be important to note that the specific nature of alcohol cues encountered by participants in the current study likely diverged from those they might encounter in everyday drinking contexts. Future studies may consider employing cues more similar to those encountered in bars and other naturalistic drinking settings.

These limitations notwithstanding, the current study advances the literature by integrating a consideration of drinking context into the understanding of neurocognitive markers of alcohol cue salience. In particular, findings provide evidence for alcohol cue salience that is both robust and also pervasive across drinkers. More generally, the current study's findings may offer new directions for understanding alcohol cue salience across contexts.

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Table 1. Descriptive Characteristics of the Sample

	All Participants (n=246)	Alcohol Condition (n=124)	Control Condition (n=122)
Gender			
n(%) Male	108 (43.9 %)	56 (45.2%)	52 (42.6%)
n(%) Female	138 (56.1%)	68 (54.8%)	70 (57.4%)
Race			
n(%) White	164 (66.7%)	81 (65.3%)	83 (68.0%)
n(%) African American	18 (7.3%)	7 (5.6%)	11 (9.0%)
n(%) Asian	58 (23.6%)	30 (24.2)	28 (23.0%)
n(%) Multi-racial	6 (2.4%)	6 (4.8%)	0 (0.00%)
Ethnicity			
n(%) Hispanic	41 (16.7%)	21 (16.9%)	20 (16.4%)
n(%) Not Hispanic	205 (83.3%)	103 (83.1%)	102 (83.6%)
Age			
mean (sd)	22.04 (1.61)	21.97 (1.51)	22.12 (1.71)
median (IQR*)	21 (21.00-22.00)	21 (21.00-22.00)	21 (21.00-22.25)

*IQR: Interquartile Range.

Table 2. Descriptive Statistics for AUD Risk Factors by Beverage Conditions

	All participants (n=246)		Alcohol condition (n=124)		Control condition (n=122)	
	Mean (sd)	Range	Mean (sd)	Range	Mean (sd)	Range
Drinking Days	8.47 (4.94)	1-28	8.84 (5.03)	1-28	8.10 (4.84)	1-26
Drinking Quantity	4.15 (2.06)	1-13	4.10 (1.90)	1-12	4.20 (2.21)	1-13
Binge Drinking	4.13 (3.64)	0-22	3.92 (3.41)	0-15	4.34 (3.87)	0-22
SIP	3.30 (3.06)	0-17	3.14 (2.85)	0-13	3.48 (3.27)	0-17
CAST-6	0.62 (1.05)	0-6	0.51 (0.91)	0-5	0.72 (1.17)	0-6

Table 3. The 2-way and 3-way interactions with Beverage group, Stimulus type, and Individual difference criteria.

2-way interactions (Beverage group x Individual difference criteria)					
Individual difference criteria	<i>b</i>	<i>se</i>	<i>t</i>	<i>p</i>	<i>BF*</i>
Quantity of alcohol used	-0.6564	0.2564	-2.56	0.0111	1.8461
Frequency of alcohol used	0.1946	0.1313	1.48	0.1398	3.9226
Frequency of binge drinking	0.1224	0.1916	0.64	0.5236	5.6386
Alcohol problems	0.0475	0.1903	0.25	0.8031	0.2121
Family history of alcoholism	-1.1137	0.6054	-1.84	0.0671	8.7459
2-way interactions (Stimulus type x Individual difference criteria)					
Individual difference criteria	<i>b</i>	<i>se</i>	<i>t</i>	<i>p</i>	<i>BF</i>
Quantity of alcohol used	0.0525	0.1116	0.47	0.6381	3.8160
Frequency of alcohol used	-0.0261	0.0486	-0.54	0.5920	4.1176
Frequency of binge drinking	0.0132	0.6679	0.20	0.8432	4.7701
Alcohol problems	0.0700	0.0784	0.89	0.3729	0.2446
Family history of alcoholism	-0.3083	0.1908	-1.62	0.1075	6.6607
3-way interactions (Beverage group x Stimulus type x Individual difference criteria)					
Individual difference criteria	<i>b</i>	<i>se</i>	<i>t</i>	<i>p</i>	<i>BF</i>
Quantity of alcohol used	0.4772	0.2464	1.94	0.0540	1.3800
Frequency of alcohol used	-0.0170	0.0950	-0.18	0.8585	2.8300
Frequency of binge drinking	0.1247	0.1422	0.88	0.3816	4.1499
Alcohol problems	0.1526	0.1640	0.93	0.3529	0.1452
Family history of alcoholism	0.1478	0.4020	0.37	0.7135	6.6859

*BF: Bayes Factor. BF less than 0.10 or greater than 10 are taken as strong evidence in favor of the null and alternative hypotheses, respectively.

Figure 1. Event-related brain potential waveforms elicited by alcohol and non-alcohol images. Waveforms elicited by standard (nontarget) images are presented for the 4 midline electrode sites to illustrate the oddball effect in these data. Stimulus onset occurred at 0 ms. Electrodes are arrayed from most anterior to most posterior as they were positioned on the scalp.

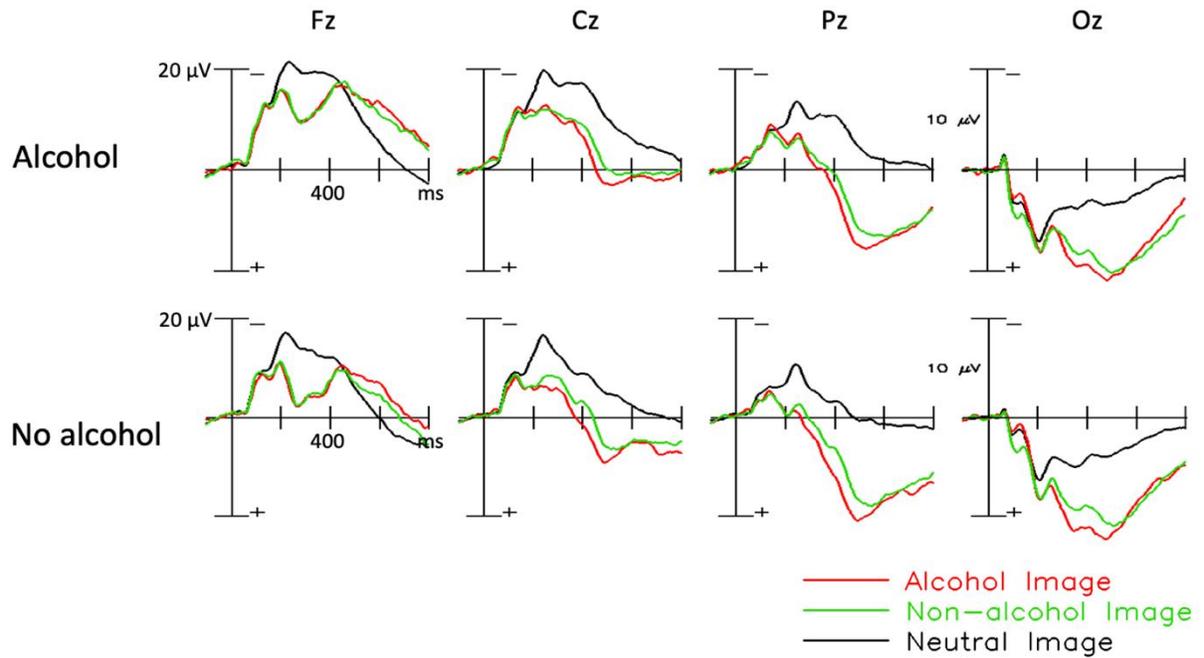


Figure 2. Main Effect of Beverage Condition and Stimulus Type (Pz channel)

