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Alcohol hangover impacts learning and reward processing within the medial-frontal cortex

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Abstract

It is common knowledge that alcohol intoxication impairs motor coordination, judgment, and decision making. Indeed, an abundance of literature links intoxication to impaired cognitive control that leads to accidents and injury. A broadening body of research, however, suggests that the impact of alcohol may continue beyond the point of intoxication and into the period of alcohol hangover. Here, we examined differences in the amplitude of reward positivity—a component of the human ERP associated with learning—between control and hangover participants. During performance of a learnable gambling task, we found a reduction in the reward positivity during alcohol hangover. Additionally, participants experiencing alcohol hangover demonstrated reduced performance in the experimental task in comparison to their nonhangover counterparts. Our results suggest that the neural systems that underlie performance monitoring and reward-based learning are impaired during alcohol hangover.

KEYWORDS
alcohol/alcoholism, EEG, ERP, FRN, reward positivity, reward processing

1 | INTRODUCTION

Alcohol intoxication can result in damaging effects for both the individual and the community. Impaired driving results in 10,000 deaths per year in the United States (Centers for Disease Control and Prevention [CDC], 2014; National Highway Traffic Safety Administration, 2013). Despite the well-known risks of alcohol intoxication, more than 1 million people make the choice to drink and drive each year (CDC, 2014). Indeed, evidence suggests the neural systems that underlie human decision making are themselves impaired by alcohol consumption (Bartholow, Henry, Lust, Saults, & Wood, 2012). Acute alcohol use impacts a variety of cognitive processes including planning, memory, and risk assessment (e.g., Starkey & Charlton, 2014). Research has shown alcohol-related impairments to emotional affect, empathy, and interpersonal skills. For example, alcohol intoxication has been linked to increased risk for engaging in violent

behavior (e.g., Giancola et al., 2011), as well as for demonstrating indirect aggression such as social manipulation or bullying (Crane, Licata, Schlauch, Testa, & Easton, 2017). In a study of bystander intervention, Sheehan, Linden-Carmichael, and Lau-Barraco (2016) were able to demonstrate both that alcohol intoxication was linked to an increased risk for participating in sexual violence, and that men who met the criteria for heavy drinking were also less likely to intervene when witnessing a sexually violent act in comparison to their peers who consumed less alcohol.

Undoubtedly, many psychophysiological processes play a role in our decisions to consume alcohol, to drive under the influence, or to intervene when witnessing violence. The ability to assess the impact of our behaviors on the environment is of particular importance to decision making (Holroyd & Coles, 2002) and underlies processes of self-regulation (Botvinick, Braver, Barch, Carter, & Cohen, 2001). A growing body of evidence supports the existence of a neural

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system for error detection within the human medial frontal cortex that allows for human performance monitoring (Holroyd & Coles, 2002; Krigolson, Hassall, & Handy, 2015; Krigolson & Holroyd, 2006, 2007a, 2007b; Krigolson, Pierce, Holroyd, & Tanaka, 2009). For example, research using EEG and ERP techniques has demonstrated that error commission in performance-based tasks can be used to elicit the error-related negativity or ERN, an ERP component that is both reliable and predictable (Gehring, Goss, Coles, Meyer, & Donchin, 1993). Another ERP component, termed the reward positivity, is reliably elicited by tasks that do not allow subjects to monitor their performance at the time of response, but rather provide postresponse feedback as to trial outcome (Miltner, Braun, & Coles, 1997; Proudfit, 2015; Walsh & Anderson, 2012). The reward positivity results from comparisons between neural responses to rewarding and nonrewarding outcomes. Holroyd and Coles (2002) theorize that the medial frontal system responsible for both the ERN and the reward positivity also play an important role in complex and long-term goal attainment. In this case, the system functions to assess rewarding feedback that results from each subset of actions leading up to the end-goal state, and thus drives the decision-making process towards end-goal attainment.

Given that alcohol intoxication results in reduced performance in a variety of tasks (Hendrie, Gao, Hall, Hui, & Unverzagt, 1996; Starkey & Charlton, 2014; Van Dyke & Filmore, 2014) and its potential to influence dopaminergic reward systems (Siciliano et al., 2017), it is not surprising that a number of studies have sought to investigate its effects on the medial frontal system via its impact on the aforementioned ERP components (Henry, 2013; Nelson, Collins, Lang, & Bernat, 2011, Ridderinkhof, Cohen, & Forstmann, 2012). Nelson et al. (2011) found that intoxication impaired the performance of the aforementioned medial-frontal error evaluation system. The authors reported reduced amplitude of the feedback-related negativity component, an ERP component that is functionally identical to the reward positivity (Proudfit, 2015), relative to that observed from nonintoxicated controls. Further, multiple studies have reported a reduction in task performance concomitant with a dampened ERN following alcohol administration (Ridderinkhof et al., 2012; Henry, 2013). Interestingly, Bartholow and colleagues (2012) sought to examine potential mechanisms underlying the impact of alcohol on decision making through investigation of its effects on the ERN component in addition to affect during a cognitive control task. In line with theories of reward processing outlined above (e.g., Holroyd & Coles, 2002), the authors reported that alcohol both impaired cognitive control and blunted the ERN, and that alcohol administration was additionally associated with blunted negative affect. In sum, the available evidence suggests that medial frontal ERPs linked to action and outcome monitoring are impaired during alcohol intoxication.

Importantly, the impact of high levels of alcohol consumption on behavior appears to be more sustained, persisting beyond the state of acute intoxication. Indeed, behavioral studies examining alcohol hangover report performance deficits such as slower response times and an increase in response errors (Ling, Stephens, & Heffernan, 2010; Stephens, Ling, Heffernan, Heather, & Jones, 2008; Verster, 2008). For example, Kim and colleagues (Kim, Yoon, Lee, Choi, & Go, 2003) found that participants who had alcohol hangover exhibited deficits in cognitive functions including visual attention, memory, and information processing, relative to controls. Past literature also suggests motor performance is impacted by alcohol hangover. In a study using animal models, Karadayian and Cutera (2013) found that mice demonstrated an 80% reduction in motor performance during the 16 hr that followed an experimentally induced ethanol hangover. Given that the aforementioned medial-frontal learning system plays a key role in the monitoring of motor activities (Ito, 1970; Krigolson & Holroyd, 2007a,b,c; Krigolson, Holroyd, Van Gyn, & Heath, 2008; Miall, Weir, Wolpert, & Stein, 1993; Miall & Wolpert, 1996; Shadmehr, Smith, & Krakauer, 2012; Wolpert, 1997), an impairment to the medial frontal system due to alcohol hangover may be an underlying factor in the increased risk of accidents and injury that have been associated with alcohol hangover in addition to underlying processes of decision making and cognitive control.

The primary goal of the present study was to measure neural and behavioral correlates of reward-based learning as a function of hangover, to examine the longer-term impact of alcohol misuse on neural systems implicated in reward-based learning, decision making (e.g., Krigolson, Pierce, Holroyd, & Tanaka, 2009, Krigolson, Hassall, & Handy, 2014), and motor control (Krigolson & Holroyd, 2006, 2007b, 2007c; Krigolson et al., 2008). To this end, we recorded EEG data from two groups of participants (hangover, control) while they performed a gambling task to win financial rewards. To assess the impact of alcohol hangover on reward processing, we focused our EEG analysis on the reward positivity—a component of the human ERP associated with reward evaluation/error evaluation (Holroyd & Coles, 2002; Holroyd, Pakzad-Vaezi, & Krigolson, 2008; Krigolson, Hassall, Satel, & Klein, 2015; Miltner et al., 1997). The reward positivity peaks approximately 250–400 ms after reward feedback, has a medial-frontal scalp topography, and has been localized to the anterior-cingulate cortex (Debener et al., 2005; Holroyd et al., 2004). Here, we predicted that the amplitude of the reward positivity would be reduced during alcohol hangover. Yoked to this reduction in performance monitoring, we also predicted that overall gambling task performance would be reduced in hangover participants relative to controls. Finally,

we expected that hangover severity would be negatively correlated with the magnitude of the reward positivity.

2 | METHOD

2.1 | Participants

Sixty-two undergraduate students took part in this study. Due to exclusion criteria (described below), we removed data sets from four of these participants from our analyses. This resulted in a total sample of 58 participants (24 male; mean age 21.5 years, 95% CI: 21.0–22.0). In total, participants consumed alcohol an average of 4.7 (95% CI: 3.8–5.6) occasions per month and 6.5 (95% CI: 5.1–7.9) drinks per occasion (see Table 1). We found no differences in drinking behavior between hangover and control participants other than the night before participation. Participants in the hangover group reported an average of 6.0 (95% CI: 3.9–8.1) drinks consumed on the night previous to the experiment and were selected based on their prior night's drinking behavior and hangover score from participants that came to the laboratory for a regularly scheduled experiment (see below). We obtained informed written consent from all participants and compensated them with three bonus points to use toward grades in psychology courses. This research was approved by the Health Sciences Research Ethics Board at Dalhousie University and followed all ethical standards prescribed in the 1964 Declaration of Helsinki. In accordance with the Center for Open Science, we confirm that we have reported all measures, conditions, and data exclusions. We determined sample size based on the sample size of previous ERP studies and what is proposed by Field (2009).
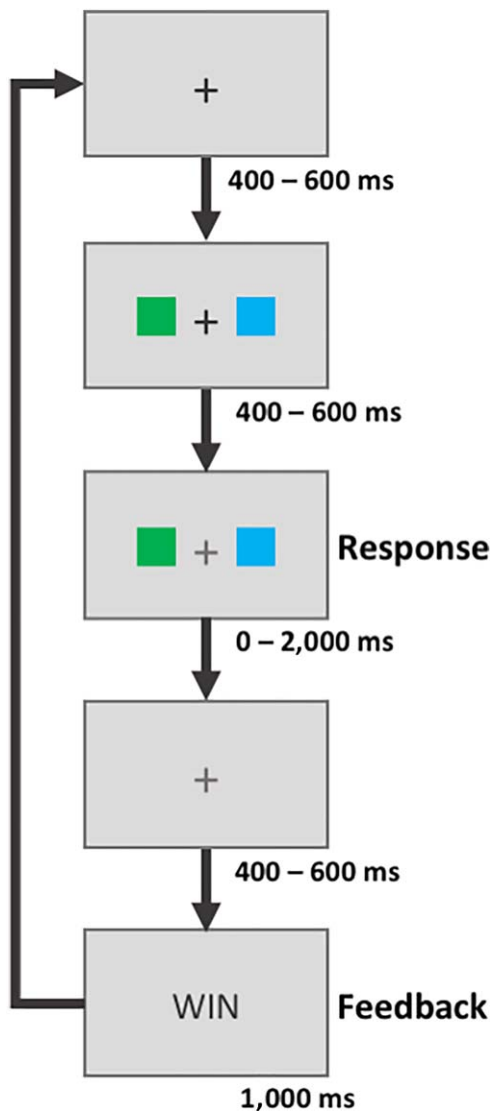
2.2 | Experimental task

Participants completed the experimental task in a sound-attenuated room while seated 75 cm away from a standard computer monitor, on which the stimuli were displayed. The task was based on a two-armed bandit (Sutton & Barto, 1998) gambling paradigm consisting of 25 blocks of 20 trials each and was programmed in MATLAB Version 7.14 (MathWorks, Natick, MA) using the Psychophysics Toolbox Extension (Brainard, 1997). Participants received both verbal and written instructions prior to beginning the task. On each trial, participants gambled by choosing between two differently colored squares using the L1 and R1 buttons on a Logitech USB game controller in an aim to "win" the trial. On each trial, each of the two squares had a different win/loss probability. At the start of each block of trials, one of the squares was assigned a win probability of 0.6 and the other square was assigned a win probability of 0.1. These probabilities were based on past research conducted in our lab, which demonstrated participants could sufficiently determine the "better" square when comparing win probabilities of 0.6 to 0.1. As each block of trials progressed, the win–loss probabilities fluctuated in response to the participants' behavior. Specifically, if the ratio of wins:losses or losses:wins exceeded 3:2, the probability of winning for each square was adjusted to make the task harder (or easier) for the participant. In this case, the probability of winning was increased or decreased by 5% for the square with the higher probability of winning and increased or decreased by 2.5% for the square with the lower probability of winning. Additionally, within each block the probability of winning for each square reversed at Trial 10 of 20. For example, if on Trial 10 the participant had a 65% chance to win when selecting a blue

TABLE 1 Participant demographics and substance behavior

	Control	Hangover	Group difference
Sample size	28	30	
Gender	11 m, 17 f	13 m, 17 f	$p > .05$
Age	21 (20.2–21.8)	21.5 (20.7–22.3)	$p > .05$
Drinks consumed last night	1.5 (0.1–2.9)	6 (3.9–8.1)	$t(56) = 3.4, p < .001$
Hangover score	0.0 (0.0-0.0)	39.3 (34.2-44.5)	$t(56) = 15.2, p < .001$
Hours of sleep last night	6.0 (5.4–6.6)	5.8 (5.2–6.4)	$p > .05$
Number of times alcohol consumed in the past 30 days	4.2 (3.1–5.3)	5.2 (3.0–7.4)	$p > .05$
Average number of drinks consumed when drinking	6.4 (4.3–8.5)	6.8 (4.6–9.0)	$p > .05$
Cannabis use (days)	0.3 (0.0–0.7)	0.8 (0.0–1.9)	$p > .05$
Number of cigarettes smoked	3.4 (0.0–6.8)	3.0 (0.0–6.7)	$p > .05$
Number of caffeinated beverages consumed in the past 30 days	28.8 (16.6–40.0)	25.7 (16.4–36.0)	$p > .05$

Note. All error statistics reflect 95% confidence intervals which appear in parentheses. m = male; f = female.

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FIGURE 1 Overview of a sample experimental trial. Participants were presented with two boxes of varying colors and with differing probabilities of payout (mapped to color). Participants were instructed to select one of the two boxes on each trial and were provided with feedback as to whether they won or lost following each selection

square, and only a 5% chance to win when selecting a red square, they would now have only a 5% chance to win on the blue square on Trial 11, and a 65% chance to win when selecting the red square. These adjustments to outcome frequency were made in order to ensure an even number of win and loss results in each block to avoid frequency contamination of the reward positivity (i.e., N200 effects; see Holroyd & Krigolson, 2007, for the logic behind this manipulation).

At the start of each trial, a white fixation cross appeared (0.84° of visual angle) for 400 to 600 ms (see Figure 1). Next, two colored squares were presented (2.14° of visual angle) on either side of the fixation cross. The colors of the squares were randomly selected at the start of each block but consistent within each block. Shortly after the presentation of the colored squares (again 400–600 ms), the fixation cross

changed to a light gray cueing participants to respond. Trials in which participants either responded before fixation cross color change or in which they were too slow (a response > 2,000 ms) ended the trial with a feedback screen of "too fast" or "too slow," respectively. On valid response trials—a response 0 to 2,000 ms postcolor change—the colored squares disappeared with the light gray fixation cross remaining on screen for a further 400 to 600 ms before a feedback stimulus was presented. The feedback stimulus consisted of either the word "win" or "loss" and was presented for 1,000 ms before the next trial began (see Figure 1 for an overview of a sample experimental trial). Participants completed 25 blocks of the gambling task and viewed their block score and total score at the end of each block of trials.

2.3 | Behavioral and EEG data collection

The MATLAB program recorded response times and gamble selection for each experimental trial. EEG data were recorded from 64 electrode locations at 500 Hz with an ActiChamp amplifier and PyCorder software (Brain Products, GmbH, Munich Germany). Within the PyCorder recording software, an 8 kHz (−3 dB) antialiasing filter and a 60 Hz notch filter were applied at the time of data collection. The 64 electrodes were fitted in an EEG cap with a standard 10–20 layout (http://neuroeconlab.com/electrode-configuration.html). All electrode impedances were kept below 20 kΩ throughout data collection.

2.4 | Survey data collection

Following completion of the experimental task, participants were interviewed using the timeline follow-back method (Sobell & Sobell, 1992, 1995; Sobell et al., 1996) in order to assess recent alcohol and substance use behavior and additionally completed a modified version of the Alcohol Hangover Severity Scale (AHSS; Penning et al., 2013).

2.4.1 | Timeline follow-back

Alcohol and substance use was assessed for a 30-day period. Participants were directly interviewed about their alcohol, cannabis, caffeine, and nicotine use. In addition, they were asked if they had used any other illicit substances such as ecstasy or cocaine, or prescription medications within the previous 30 days. The test-retest reliability of the timeline follow-back interview has a kappa value of 0.77 when used to determine substance use (Carey, 1997). Furthermore, when tested with the Addictions Severity Index, the measure demonstrated acceptable concurrent validity with a bivariate correlation of 0.65 ($p < .0001$; Carey, 1997).

2.4.2 | Modified Alcohol Hangover Severity Scale (M-AHSS)

The AHSS (Penning et al., 2013) consists of 12 items, each measuring a different symptom associated with alcohol hangover. In our study, an additional single-item score of "overall hangover severity" was added to the original 12 criteria of the scale. Participants rated how strongly they were experiencing each symptom on a scale from 0 to 10 (*absent* to *extreme*). An overall hangover score was calculated by summing the responses to the original 12 items and our new 13th item.

2.5 | Data analysis

2.5.1 | Behavioral analysis

After the experiment, participants were assigned to either a hangover or control group. Inclusion in the alcohol hangover group was determined by two criteria: (a) having consumed alcohol within 24 hr of experimental participation (but not within 10 hr of the study start time), and (b) having a score on both the M-AHSS and single-item scale for subjective hangover severity greater than zero. Participants who did not meet both of these criteria were assigned to the control group (see Table 1 for a full summary of all behavioral data about substance use, etc.). Further, we examined prior drinking history (number of times alcohol consumed per month, number of drinks on average consumed) and drug use of each participant. Participants who had used cannabis were included in the study; however, participants who had used other recreational drugs (e.g., cocaine) were not included in the study. We also examined hours of sleep the night before, smoking behavior (number of cigarettes), and caffeine intake. Finally, we computed the mean response time and mean performance (i.e., the percent selection of the optimal response) for each participant.

2.5.2 | EEG analysis

EEG data were processed offline with BrainVision Analyzer 2 software (Version 2.1.1, Brain Products, GmbH) using methods previously employed by our laboratory (see http://www.neuroeconlab.com/data-analysis.html). First, excessively noisy or faulty electrodes were removed. Next, the continuous EEG data were rereferenced to the average of the two mastoid channels (TP9, TP10) and then filtered using a dual-pass Butterworth filter with a pass-band of 0.1 Hz to 30 Hz and a 60 Hz notch filter. Segments encompassing the onset of each event of interest (1,000 ms before to 2,000 ms after) were then extracted from the continuous EEG. Following segmentation, independent component analysis (ICA) was used to correct ocular artifacts (Delorme & Makeig,

2004; Luck, 2014). Data were reconstructed after the ICA, and any channels that were removed initially were interpolated using the method of spherical splines. Shorter epochs were then constructed from 200 ms before to 600 ms after the onset of each event of interest (i.e., the onset of the win and loss feedback stimuli). All segments were then baseline corrected using the 200-ms epoch immediately preceding stimulus onset. Following this, all segments were submitted to an artifact rejection algorithm that marked and removed segments that had gradients of greater than 10 µV/ms and/or a 100 µV absolute within-segment difference (on average 17.62% of the data). Following artifact rejection, win and loss segments were separately averaged to create ERP win and loss waveforms for each participant. Difference waveforms were then computed for each participant by subtracting the average loss from the average win waveform. Finally, grand-averaged waveforms were computed for the win, loss, and difference waveforms for both the hangover and no hangover groups.

In line with previous work (Holroyd & Coles, 2002; Krigolson & Holroyd 2007a; Krigolson et al., 2009), a temporal and topographical examination of the grand-averaged difference waveforms was completed to confirm presence of the reward positivity component. The reward positivity for each participant was quantified as the peak positive deflection of the difference waveform 200 to 400 ms postfeedback onset at electrode FCz. To assess the impact of hangover on the reward positivity and performance, we conducted independent samples t tests to test for differences in these variables between groups. Finally, to investigate the relationship between hangover score and reward positivity magnitude, we conducted a Pearson correlation for these variables within the hangover group.
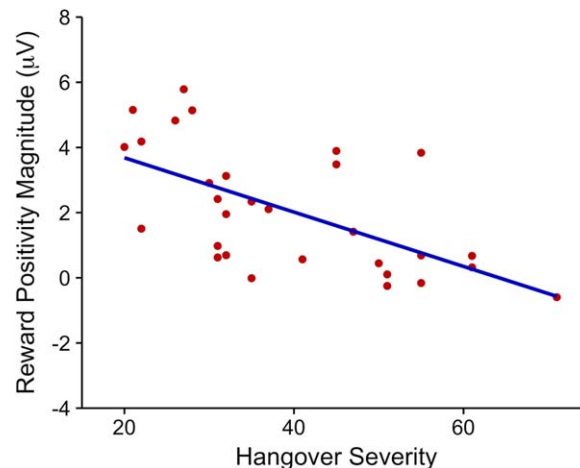
3 | RESULTS

An examination of our substance use and sleep data revealed no differences between participants in the control and hangover groups other than the number of drinks consumed the night before and a score on the M-AHSS (see Table 1 for full details). Behavioral task analysis focused on two primary measures: mean performance and variability of performance with regard to the number of times the optimal response option was selected. Overall, hangover participants demonstrated greater variability in performance than controls (control: 17.8%, hangover: 20.5%; $t(56) = 2.32$, $p = .025$). Further, mean performance was reduced in the hangover group in comparison to the control group (control: 61%, hangover, 58%; $t(56) = 4.29$, $p < .001$). A comparison of performance across trials for the hangover and control groups is presented in Figure 2. We also examined the relationship between hangover severity and variability of performance/
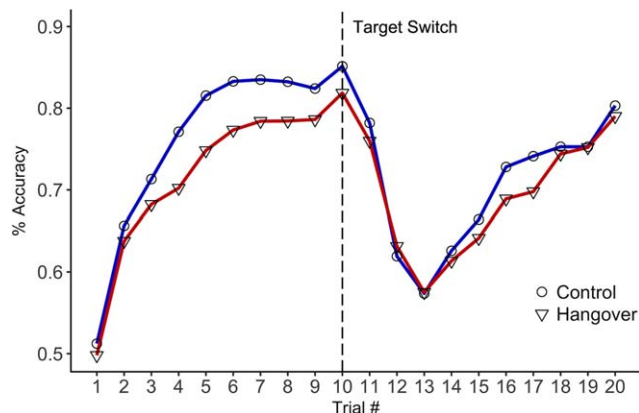
FIGURE 2 Performance across collapsed trials averaged over all blocks and measured in percent of better selections out of all selections. Participants were informed that the target box may switch on some trials. Time of target switch is indicated by a dashed line

FIGURE 4 Peak component magnitudes plotted against hangover severity in the hangover group. Pearson's $r = -.61$

mean performance. We did not observe a relationship between mean performance and hangover severity (Pearson's $r = -.05$, $p = .796$). However, we did observe a small correlation (Cohen, 1988) between variability of performance and hangover severity (Pearson's $r = .18$, $p = .363$).

In terms of the EEG data, in the total sample we observed an ERP component with timing and scalp topography (298 ms, maximal over FCz) consistent with previous accounts of the reward positivity (see Figure 3a; Holroyd & Coles, 2008; Krigolson et al., 2014; Proudfit, 2015). Comparison of the amplitude of the reward positivity between control and hangover participants revealed a reduction in component amplitude for hangover (see Figure 3b) relative to control (see Figure 3a) participants (control: 4.66 uV, hangover: 2.07 uV; $t(56) = 4.26$, $p < .001$). A comparison of grand-averaged difference waves for each group is presented in Figure 3c. Finally, within the hangover group we found that hangover severity was negatively correlated with the magnitude of the reward positivity component (Pearson's

$r = -.61$, $p < .001$, see Figure 4). We also observed a small correlation between the magnitude of the reward positivity and task performance (Pearson's $r = -.28$, $p > .05$) across task performance.

4 | DISCUSSION

In the present experiment, we predicted that reward processing would be impacted by alcohol hangover. Specifically, we predicted that the amplitude of the reward positivity would be reduced for participants experiencing alcohol hangover and that the magnitude of the component would be negatively correlated to hangover severity. Additionally, we hypothesized reduced performance in the cognitive task in the hangover group relative to the control group. Importantly, other than the number of drinks consumed the night prior to the study and the score on the M-AHSS, we found no differences between participants with and without
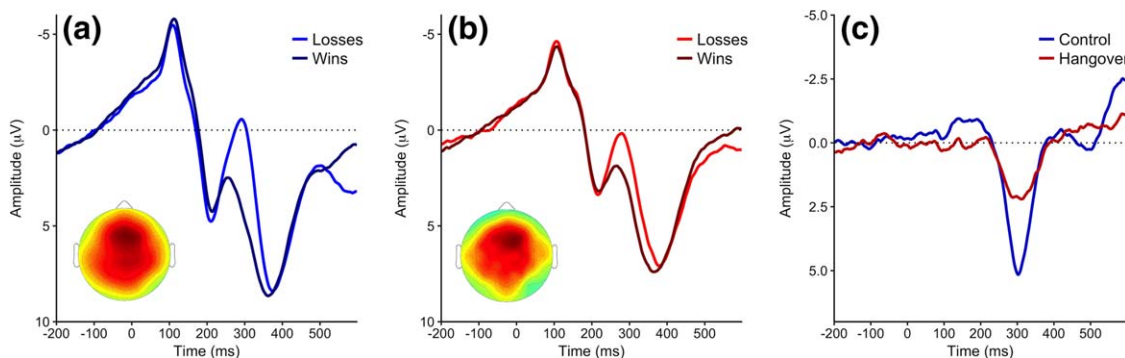
FIGURE 3 Grand-averaged waveforms for wins and losses across all trials and blocks with scalp topographies for (a) control group, and (b) hangover group. Latency and topography of the component is consistent with previous reports of the reward positivity. Scalp topographies are measured at 298 ms postfeedback onset, when the difference between win and loss waveforms is maximal. Topography scale is −10 to 10 µV. (c) Difference waves for each group (control, hangover) as calculated by win-loss

hangovers in terms of hours of sleep, other substance use, and general drinking behavior (see Table 1).

In line with previous behavioral studies (Ling et al., 2010; Stephens et al., 2008; Verster, 2008), task performance was indeed reduced for hangover participants relative to controls. Specifically, hangover participants exhibited increased performance variability and reduced overall performance on the experimental gambling task, as defined by the proportion of trials on which they selected the optimal choice square. Consistent with our primary hypothesis, we also observed a reduction in the amplitude of the reward positivity in hungover participants, suggesting that medial-frontal systems for feedback evaluation are impacted by hangover. These data are consistent with the notion that the impact of higher volumes of alcohol consumption on reward-based learning and reward-related brain activity continue beyond alcohol intoxication into hangover. Indeed, hangover has been associated with increased accidents and motor impairment (Verster, 2007), and the current study suggests a potential mechanism governing the association between hangover and accidents.

It is not difficult to see how impairment in reward processing systems, with a particular focus on the role of these systems in feedback evaluation, could pose potential danger. Specifically, impairments to the medial frontal reward processing system may reduce an individual's ability to successfully perform complex motor tasks, including those necessary for athletic activities and operation of motor vehicles. In both cases, impairment of the medial-frontal system could lead to accidents and injury. As discussed previously, Verster (2007) reported that a large proportion of professional drivers (taxi services, couriers, etc.) admitted to experiencing alcohol hangover while at work. Given the proposed role of the medial-frontal system in the governance of motor control (i.e., Krigolson & Holroyd, 2006, 2007b, 2007c), if alcohol hangover does indeed result in a reduction in the efficacy of the medial-frontal system, then it is important, as suggested by Verster, to educate the public as to the dangers of alcohol hangover.

Previous research has demonstrated that alcohol-related reductions in medial-frontal ERPs are linked to impairments in cognitive control and behavioral regulation that are experienced during intoxication (Nelson et al., 2011), and, further, that this impairment is mediated by negative affect (Bartholow et al., 2012). Insofar as the current results suggest that hangover is associated with attenuated neural response to reward, these findings have broader implication for alcohol misuse. Specifically, blunted neural response to reward has been associated with negative affect (Bartholow et al., 2012) and depression (Bress, Smith, Foti, Klein, & Hacjak, 2012; Foti, Carlson, Sauder, & Proudfit, 2014; Nelson, Perlman, Klein, Kotov, & Hajcak, 2016). If hangover similarly blunts response to reward, individuals with hangover may seek to normalize this attenuated response to reward by consuming alcohol—a possibility that could be tested in future studies. In line with this hypothesis, behavioral research has demonstrated that negative emotional states during hangover can be predicted by a lack of adherence to self-imposed consumption limits, above and beyond the amount of alcohol consumed alone, and that this negative affect is linked to intentions and likelihood of consuming alcohol in the following 24 hr (Muraven & Shmueli, 2006.).

4.1 | Limitations

While the data we acquired from participants seem to clearly show that the participants in both groups were similar other than their drinking behavior the night before and their scores on the M-AHSS, there are some limitations that should be addressed. First, other than a negative response to a general prescreening question of "Do you have any reason for thinking that your data may be impacted by something you have not told us about yourself or your condition today?" we do not know if participants differed in terms of an underlying psychological issue such as psychopathy and/or depression. Second, our knowledge of the prior night's alcohol consumption was entirely based on self-report rather than a urine or saliva test. Third, there are other factors that could potentially explain our results that we did not assess. For instance, while we did ask about the hours of sleep the night before, we did not employ a validated survey to establish quality of sleep. Additionally, there are other potential constructs that we did not measure such as trait inhibition, which could also potentially contribute to the effects reported here. Lastly, while our group sizes are consistent with previous work in the area (e.g., Button et al., 2013), the present study could be considered to have low positive predictive value and thus the effect sizes reported here may be overestimated (post hoc computed power = 0.465 using G power).

4.2 | Conclusions

Coupled with evidence from past behavioral research, the results of this study suggest that individuals may be less able to evaluate important feedback in their environment when experiencing alcohol hangover—feedback that is necessary for cognitive and motor skills including learning. Indeed, the current study provides evidence for hangover-related impairments to neural mechanisms for feedback evaluation. Specifically, our observed attenuation of the reward positivity component in hangover participants suggests reduced activity in the medial-frontal reward system in comparison to controls. Given the hypothesized role of the medial-frontal reward system in motor control (e.g., Krigolson & Holroyd, 2007a), our results identify one mechanism that may explain the increase of performance errors and accidents evident during alcohol hangover.

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