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ORIGINAL INVESTIGATION

Acute effects of alcohol on error-elicited negative affect during a cognitive control task

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Abstract

Rationale Alcohol intoxication can dampen negative affective reactions to stressors. Recently, it has been proposed that these acute anxiolytic effects of alcohol may extend to dampening of negative affective reactions to error commission during cognitive control tasks. Nonetheless, empirical verification of this claim is lacking.

Objectives Test the acute effect of alcohol on negative affective reactions to errors during an effort-demanding cognitive control task.

Methods Healthy, young adult social drinkers ($N = 96$ [49 women], 21–36 years old) were randomly assigned to consume alcohol (0.80 g/kg; $n = 33$ [15 female]), active placebo (0.04 g/kg; $n = 33$ [18 women]), or a non-alcoholic control beverage ($n = 30$ [16 women]) before completing the Eriksen flanker task. Corrugator supercilii (Corr) activation, a psychophysiological index of negative affect, was tracked across the task. Two neurophysiological reactions to errors, the error-related negativity (ERN) and the error positivity (Pe), were also measured.

Results Erroneous actions increased Corr activation in the control and (to a lesser extent) placebo groups, but not in the alcohol group. Error-induced Corr activation was coupled to ERN and Pe in the control, but not in the alcohol and placebo groups. Error-induced Corr activation was not coupled to post-error performance adjustments in any group.

Conclusions The ability of alcohol to dampen error-related negative affect was verified. It was also shown that placebo alone can disrupt coupling of affective and (neuro)cognitive reactions to errors. Although its behavioral relevance remains to be demonstrated, more attention should be paid to the role of affect in action monitoring and cognitive control processes.

Keywords Action monitoring · Alcohol · Anterior cingulate cortex · Blame attribution · Cognitive control · Corrugator supercilii · ERP · Errors · Negative affect · Performance adjustments

Cognitive control refers to a set of cognitive processes that allow individuals to guide their behavior in accordance with internal goals (Alexander and Brown 2010; Braver 2012; Gratton et al. 2018). A core component of cognitive control is the ability to adjust behavior in response to varying situational demands (Botvinick et al. 2001). Considerable research points to a neural circuit centered on the dorsal anterior cingulate cortex (ACC) as critical to cognitive control, particularly for signaling when adjustments in control are needed, i.e., following errors (Carter et al. 1998; Davis et al. 2005; Hall et al. 2007; Smith et al. 2019; Van Veen et al. 2001; Wang et al. 2005; Yeung et al. 2004). ACC responses to errors can be observed in the amplitude of the scalp-recorded error-related negativity (ERN), a response-locked event-related potential (ERP) component that is much larger during incorrect versus correct responses (Gehring et al. 1993; Holroyd and Coles 2002; Van Veen and Carter 2002) and that has been localized to ACC and neighboring structures in the medial prefrontal cortex (Debener et al. 2005; Dehaene et al. 1994; Herrmann et al. 2004).

Impaired cognitive control is one of the acute effects of alcohol (e.g., Casbon et al. 2003; Guillot et al. 2010). Supporting evidence comes in part from studies showing that alcohol reduces or eliminates adjustments in performance that typically occur following errors (Bailey et al. 2014; Bartholow

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et al. 2012; Ridderinkhof et al. 2002). Additional evidence comes from studies showing that alcohol reduces error-related neurophysiological responses (Anderson et al. 2011; Marinkovic et al. 2012), including the ERN (Bailey et al. 2014; Bartholow et al. 2012; Easdon et al. 2005; Nelson et al. 2011; Ridderinkhof et al. 2002).

At least four explanations for alcohol-induced cognitive control impairment have been offered. The first is that impairment reflects alcohol-induced *global* deficits in executive functions (Giancola 2000; Pihl et al. 2003). The second is that impairment reflects alcohol-induced deficits in visual stimulus processing (Yeung et al. 2007; Yeung and Cohen 2006). Both of these explanations were challenged by Bailey et al. (2014), who showed no effect of alcohol on conflict adaptation in behavioral performance or neurophysiological correlates of conflict monitoring (reactive control) processes during sequences of correct response trials. Acute effects of alcohol on performance and neurophysiology were observed only after error commission. A third explanation is that impaired cognitive control reflects deficits in error detection/recognition under alcohol as evidenced by reduced ERN (Ridderinkhof et al. 2002; also suggested in Yeung et al. 2007). This explanation was challenged by Bartholow et al. (2012) and Bailey et al. (2014), who replicated the finding that the ERN is reduced under alcohol, but also showed that participants given alcohol were just as accurate in detecting/recognizing their mistakes as participants given control and placebo beverages. A fourth explanation, proposed by Bartholow et al. (2012), is that impaired cognitive control might reflect alcohol-induced reduction of affective reactions to failures of control.

Negative affective reactions to failures of control, such as error commission, are theorized to motivate post-error increases in attention and improvements in performance (K. Aarts et al. 2013; Dignath et al. 2020; Inzlicht et al. 2015; Proudfit et al. 2013; Saunders et al. 2015). The idea that negative affective reactions may be especially important for cognitive control is rooted in a long history of research showing that the ACC is critically involved in the evaluation of distress and pain (e.g., Ballantine et al. 1967; Rainville et al. 1997; Talbot et al. 1991), that failures of control, such as error commission, are aversive (Hajcak et al. 2004; Hajcak and Foti 2008), and that error-elicited activity in the ACC indexed by ERN amplitude covaries with the motivational significance of errors (Gehring et al. 1993; Gehring and Taylor 2004; Hajcak et al. 2005). Viewed from this perspective, acute effects of alcohol on error processing in cognitive control tasks could reflect the drug's well-known anxiolytic properties.

Acute alcohol has well-documented anxiolytic effects in humans (for review, see Greeley and Oei 1999; Sayette 1999, 2017). Specifically, alcohol dampens subjective negative affective reactions (Bradford et al. 2013; Bujarski and Ray 2014; Levenson et al. 1980; Ray et al. 2009, 2013; Sher et al. 2007) as well as facial expressions of negative affect (Kushner et al. 1997; Sayette et al. 1992, 2012) in a dose-dependent manner. It similarly dampens negative affective reactions at the level of central nervous system activity (e.g., Curtin et al. 2001; Franken et al. 2007; Gorka et al. 2013) and autonomic nervous system regulation (e.g., Bradford et al. 2013; Donohue et al. 2007; Sher et al. 1994, 2007; Udo et al. 2009; Vaschillo et al. 2008).

Yet, the degree to which negative affect elicited by erroneous actions is reduced by alcohol remains unclear. Indirect support for this idea comes from studies showing that both alcohol-induced ERN reduction and impairments of post-error performance adjustment can be mediated by alcohol-induced decreases in subjective negative affect (Bartholow et al. 2012), and from studies showing that anxiolytic medication (e.g., lorazepam) also reduces the ERN (De Bruijn et al. 2004). Direct support for this idea, however, requires an approach in which alcohol's effects on negative affective reactions can be precisely time-locked to error commission.

Decades of research point to reactivity in the corrugator supercilii, the facial muscles that furrow the brow, as an excellent measure for this purpose (Cacioppo et al. 1984, 1986, 1988; Cacioppo and Petty 1981; Larsen et al. 2003; Tan et al. 2012; Tassinary et al. 1989; Vrana 1993). Corrugator supercilii reactivity, measured via electromyography (EMG) (Fridlund and Cacioppo 1986), has been advanced as an indicator of automatic (involuntary) expression of negative affect (Dimberg et al. 1998, 2000, 2002; Dimberg and Thunberg 1998). Corrugator supercilii EMG (cEMG) activity increases during exposure to different kinds of aversive stimuli (e.g., electric shock, loud noise, disgusting odors, upsetting pictures and videoclips, angry and fearful faces, negative emotion words) (Cacioppo et al. 1986; Dimberg et al. 1998, 2000; Dimberg and Thunberg 1998; Hermann et al. 2000; Larsen et al. 2003; Neumann et al. 2005; Rymarczyk et al. 2011; Sestito et al. 2013). Additionally, aversive stimulus-elicited cEMG activity may reflect neural activity in the ACC because the latter has projections to the brainstem facial nucleus, which innervates the corrugator (Cattaneo and Pavesi 2014; Shackman et al. 2011). Consequently, error-elicited cEMG activity may reflect error-elicited activity in ACC. In keeping with this idea, response-locked cEMG waveforms show amplified activity shortly after error commission (Elkins-Brown et al. 2016; Lindström et al. 2013), and this activity appears related to the magnitude of the error positivity (Pe), an ERP component that follows the ERN and has been linked to distinct aspects of error processing, such as conscious error recognition (Overbeek et al. 2005). Like the ERN, the Pe is thought to emanate from the ACC (Falkenstein et al. 2000; Herrmann et al. 2004).

Here, we revisited the experiment reported by Bailey et al. (2014), which aimed to characterize performance (accuracy, response time, confidence) as well as stimulus- and response-

locked ERPs (N2, FSW, ERN) during a classic, effort-demanding cognitive control task, the Eriksen flanker task (B. A. Eriksen and Eriksen 1974), among participants who had consumed alcohol, a placebo, or a control beverage. The breath alcohol concentrations and placebo manipulation check for this experiment were also published in the Bailey et al. report. For the current report, we examined never-before-reported cEMG activity elicited by errors and correct responses during the flanker task. We predicted (1) that response-locked cEMG activity would be enhanced on erroneous versus correct response trials; (2) that cEMG activity elicited by errors would be dampened following alcohol consumption; (3) that cEMG activity elicited by errors would correlate with ACC activation, as reflected in the amplitude of the ERN and/or Pe components of the response-locked ERP; and (4) that cEMG activity elicited by errors would predict post-error performance adjustments.

Method

Participants

A total of 96 healthy young adult social drinkers (46% female; $M_{age} = 23.19$ yra; $N = 96$) from Columbia, MO, completed the single-session experimental study. Recruitment strategy and study eligibility criteria were previously reported (Bailey et al. 2014). Characteristics of the final analytic sample for this report ($N = 74$) are presented in Supplemental Table 1.

Materials

Beverage administration

Details were previously published in Bailey et al. (2014). Briefly, participants were randomly assigned to consume one of three beverages during the experiment: an alcohol beverage (dose = 0.80 g/kg), an active alcohol placebo beverage (dose = 0.04 g/kg), or a control beverage. The alcohol beverage was 5:1 tonic to vodka (50% ABV). The placebo beverage was 5:1 tonic to diluted vodka (9:1 flattened tonic to vodka). Doses were calculated based on estimated total body water and the duration of the drinking period (15 min) using published formulae (Curtin and Fairchild 2003). The control beverage was tonic. Beverages were divided into three drinks, each consumed over 5 min. After the third drink, participants sat idle for another 5 min to permit complete absorption. Alcohol and placebo groups were told the beverage contained "a moderate amount of alcohol." The control group was told that the beverage contained no alcohol.

Breath alcohol concentration

The concentration of alcohol in exhaled breath was measured using a breathalyzer (Alco-Sensor IV; Intoximeters, Inc., St. Louis, MO, USA). Breath alcohol concentration (BrAC) was never shown to participants.

Cognitive control task

As previously reported in detail by Bailey et al. (2014), participants completed an arrows version of the Eriksen flanker task (B. A. Eriksen and Eriksen 1974; C. W. Eriksen and Hoffman 1973; Gratton et al. 1992) adapted from Ridderinkhof et al. (2002). Stimulus arrays were presented for 100 ms and contained a left- or right-facing central arrow plus two similarly or oppositely facing flanker arrows on each side. Participants identified the direction of the central arrow by pressing a left-hand or right-hand button on an ms-accurate button box. Left- and right-hand responses were equally frequent. On correct response-compatible trials, flanker arrows faced the same direction as the central arrow. On correct response-incompatible trials, flankers faced the direction opposite to the target. Correct response-compatible and response-incompatible arrays were presented pseudo-randomly and occurred with equal probability. Following a button press on each trial, participants rated their confidence in the correctness of their response (see Hester et al. 2005; Nieuwenhuis et al. 2001; Payne et al. 2005). Three seconds later, an inter-trial interval (randomly varying between 1100 and 1500 ms) occurred, after which the next trial began. Over the course of seven practice blocks (28 trials/block), participants were titrated to a speed-accuracy balance that produced approximately 10% errors. Participants making fewer errors were instructed to speed up; those making more errors were instructed to slow down. No feedback was given during the subsequent experimental trials (10 blocks total, 80 trials/block).

Electrophysiological recording

The scalp electroencephalogram (EEG) was recorded continuously throughout the flanker task from 32 tin electrodes embedded in a stretch-lycra cap (ElectroCap, Eaton, OH). Electrode sites were prepared such that measured impedance of the skin was ≤ 5 kΩ. The EEG was referenced to the right mastoid during recording. The surface electromyogram (EMG) was recorded throughout the task from 0.25 cm Ag-AgCl electrodes in a bipolar recording configuration placed about 1 cm apart over the left corrugator supercilii, following EMG recording guidelines (Fridlund and Cacioppo 1986). The EMG signal was grounded to the middle of the forehead near the hairline. Both EEG and EMG signals were amplified using a Synamps2 amplifier (Compumedics Neuroscan,

Charlotte, NC) and sampled at 1000 Hz. The EEG signal was band-pass filtered online (0.05 to 40 Hz). A 20–500-Hz band-pass filter was applied offline to the EMG to attenuate all slow non-muscle potentials (De Luca et al. 2010; Fridlund and Cacioppo 1986; Van Boxtel 2001). After recording, the EEG was re-referenced to an average of the two mastoids. Ocular artifacts (blinks) were removed from EEG and EMG using regression-based procedures (Semlitsch et al. 1986).

EEG data were segmented into epochs of − 200 to 1200 ms of post-response activity. Epochs containing artefactual deflections (exceeding ± 75 μV; e.g., due to major muscle movement) were rejected (28.36% of trials/subject). Average voltage from 200 to 100 ms before response onset was subtracted from the rest of the waveform. Inspection of the grand average ERP waveforms indicated a negative-going voltage deflection peaking between 25 and 75 ms post-response that was maximal over the fronto-central scalp, corresponding to the ERN and its correct response analog, the correct-related negativity (CRN), as shown in Fig. 3a. There was also a later, broader, positive-going deflection peaking between 150 and 450 ms that was maximal over the centro-parietal scalp, corresponding to the Pe and its correct response analog, the correct positivity (Pc), as shown in Fig. 4a. Consequently, the ERN/CRN in each retained epoch was quantified as the average voltage in the 25–75-ms post-response window on the frontal and central electrode sides (Fz, F3, F4, FCz, FC3, FC4, Cz, C3, C4). The Pe/Pc was quantified in each retained epoch as the average voltage in the 150–450-ms post-response window on the centro-parietal and parietal electrode sides (CPz, CP3, CP4, Pz, P3, P4).

cEMG data were segmented into two sets of epochs: 200 ms of pre-stimulus activity and − 200 to 1200 ms post-response activity. The root mean square voltage was computed for each 100-ms bin. Epochs containing artefactual deflections (i.e., exceeding ± 3.5 standard deviations from the average activity in the epoch or from the average activity across all epochs for the subject) were rejected (1.98% of trials/subject for pre-stimulus epochs and 1.91% of trials/subject for response-locked epochs). Retained pre-stimulus epochs were averaged together (collapsing the two bins) to produce each subject's overall average pre-trial cEMG (presented in Online Supplemental Information). Retained response-locked cEMG epochs were baseline corrected by subtracting the average root mean square voltage in bin "-2" (which corresponded to the − 200 to − 100-ms post-response window) from the other bins in the epoch.

Procedure

Figure 1 summarizes the experimental procedure and shows the timing of different within-session events. Procedural details were previously published in Bailey et al. (2014).

Results

Of 96 participants, three (all control group) were excluded due to equipment malfunction (they also were excluded in Bailey et al. 2014). Stored data were lost for two participants (1 alcohol group, 1 placebo group). Data from four participants (1 alcohol group, 2 control group, 1 placebo group) were excluded due to unstable baseline cEMG.[1] Data from 11 participants (2 alcohol group, 3 control group, 6 placebo group) were excluded because fewer than six error trials with artifact-free cEMG and ERP data were available for analysis.[2] Thus, our final analytic sample consisted of 74 participants (21 control group, 28 alcohol group, 25 placebo group). Given that this sample differs substantially from that used by Bailey et al. (2014), it was necessary to re-analyze certain measures critical for understanding the current report. Re-analyses of BrAC, flanker task performance, and ERN data are presented in the main text. Re-analyses of placebo manipulation check and subjective intoxication data are presented in Online Supplemental Information. Readers also are referred to Online Supplemental Information for analyses of pre-trial cEMG and subjective affect data. Briefly, pre-trial cEMG levels were reduced in the alcohol relative placebo and control groups (but note that response-locked cEMG data were baseline corrected). There were no beverage effects on subjective affect data. Manipulation check and subjective intoxication scores indicated a convincing placebo.

BrAC

Repeated-measures ANOVA indicated a significant main effect of the repeated measure (5 levels of "assessment": #2–6), $F(4, 104) = 5.207$, $p < 0.001$, $\eta^2 = 0.003$. As shown in Fig. 2, pairwise comparisons indicated an increase in BrAC from assessment 3 to 4 capturing the final ascent to peak BrAC, $t(27) = 5.340$, $p < 0.001$, $d = 2.094$, and successive decreases in BrAC from assessment 4 through 6 capturing the initial descent from peak BrAC, $t(27) \geq 1.995$, $p \leq 0.057$, $d \geq 0.782$.

[1] As indicated by mean pre-trial cEMG ± 2.5 SD from the grand mean. Such extreme values are most likely due to poor cEMG recording (e.g., poor skin preparation, poor electrode placement, sweating).

[2] Prior psychometric work on ERP indices of performance monitoring (Olvet and Hajcak 2009; Rietdijk et al. 2014) indicates that ideally six or more artifact-free error trials are needed to measure error-related neurophysiological signals reliably within individuals. Initial psychometric work on response-locked cEMG has focused on within-person standardized activity, and suggests that ideally 14 or more artifact-free error trials are needed for reliable estimates of error-induced activity (Elkins-Brown et al. 2016, 2017). Since it is unclear that the latter guideline can be applied to non-standardized cEMG (given that standardization distorts the EMG waveform), we applied the "six or more artifact-free error trials" guideline from the performance monitoring ERP psychometrics literature.

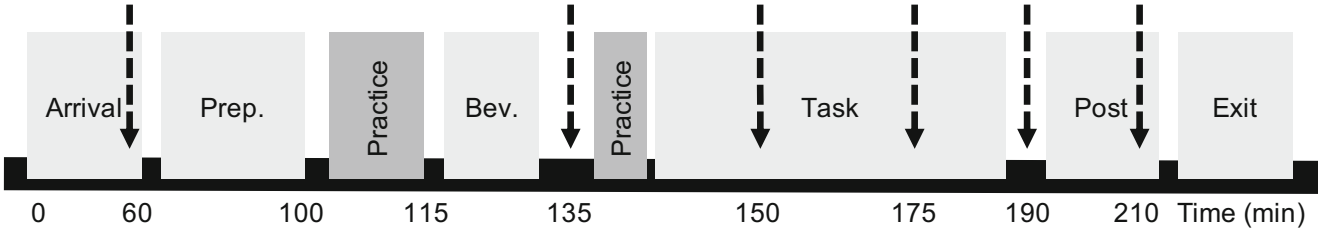


Fig. 1 *Timeline of within-session events*. Numbers below the line indicate planned timing. *Arrival*: Participants provided informed consent, indicated compliance with pre-study protocols, and were randomly assigned to a beverage condition (alcohol, placebo, control). Participants then completed a questionnaire battery unrelated to the current report. *Dashed arrows*: At several times across the session, all participants completed brief questionnaires to assess subjective affect (see Online Supplemental Information). At these times, BrAC also was measured in alcohol and placebo group participants. At arrival, however, BrAC was measured in all participants to confirm sobriety (0.000 g%). *Preparation (Prep.)*: Experimenters escorted the participant to a recording chamber, measured height and weight, and then placed and tested electrodes for electrophysiological recording. *Practice*: Participants completed 5 blocks of practice trials for the cognitive control task before beverage administration, and 2 blocks of practice trials afterward. *Beverage administration (Bev.)*: Experimenters prepared beverages in front of participants, and participants then consumed the beverage (three drinks, 5 min/drink). *Task*: Participants completed 10 blocks of experimental trials of the cognitive control task with breaks occurring after blocks 3 and 7. *Post*: Experimenters detached electrodes and escorted participants to the restroom where the latter could wash recording gel from their face and hair. All groups then completed a post-experiment questionnaire. For the alcohol and placebo groups, the latter contained a placebo manipulation check (see Online Supplemental Information). All participants were then debriefed. *Exit*: Participants in the control and placebo groups were dismissed. Participants in the alcohol group were retained until BrAC ≤ 0.02 g%

Response-locked ERPs

ERN/CRN

ANOVA[3] on ERN/CRN mean amplitudes considered the between-subject factor of the beverage group (3 levels: alcohol, placebo, control), the within-subject factor of the current trial response accuracy (2 levels: correct, incorrect), and their interaction, controlling for the within-subject factor of the electrode (9 levels: Fz, F3, F4, FCz, FC3, FC4, Cz, C3, C4). ANOVA detected a significant beverage group × response accuracy interaction, $F(2, 1315) = 5.520$, $p = .004$, $\eta^2 = 0.016$, evident in Fig. 3b. We followed up on this interaction by testing a planned directional prediction based on what was found in the larger sample (Bailey et al. 2014). Specifically, we tested whether the ERN was significantly *less negative* for the alcohol group compared to the control and placebo groups using a 1-sided, independent-samples *t* test. This prediction was confirmed: $t(72) = 2.026$, $p = 0.023$, $d = 0.478$. In contrast, the ERN was similar between the control and placebo groups, $t(44) \leq 0.082$, $p \geq 0.247$, $d \leq 0.025$.

Pe/Pc

This response-locked ERP component was not considered by Bailey et al. (2014), but we were interested in it as a potential predictor of response-locked cEMG activity and, thus, quantified and analyzed it here. ANOVA[3] on Pe/Pc mean amplitudes considered the between-subject factor of the beverage group (3 levels: alcohol, placebo, control), the within-subject factor of the current trial response accuracy (2 levels: correct, incorrect), and their interaction, controlling for the within-subject factor of the electrode (6 levels: CPz, CP3, CP4, Pz, P3, P4). Neither the main effect of the beverage nor its interaction with response accuracy was significant, $F(2, 876) \leq 2.180$, $p \geq 0.114$, $\eta^2 = 0.007$, in keeping with Fig. 4a. ANOVA detected only a significant main effect of response accuracy, $F(1, 876) = 611.320$, $p < 0.001$, $\eta^2 = 0.408$. This within-subject effect was due to a larger (*more*

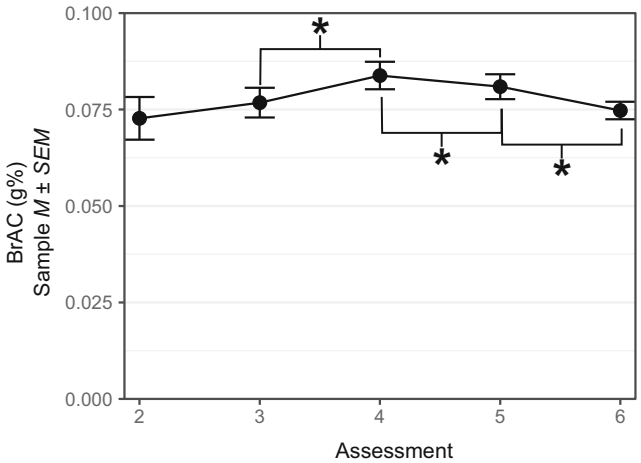


Fig. 2 *Breath alcohol concentration (BrAC) across post-drinking assessments*. Sample *M* and *SEM* are shown ($n = 28$). BrAC at assessment 1, which took place before drinking, was 0.000 g%. Assessment 2 took place 5 min after drinking and before starting the cognitive control task experimental trials. Assessments 3, 4, and 5 took place after cognitive control task experimental trial blocks 3, 7, and 10, respectively. Assessment 6 took place after electrodes were detached but before debriefing. Asterisk indicates $p < 0.05$

[3] Repeated-measures ANOVA was used for response-locked ERP analyses (instead of linear mixed modeling [LMM], which was used for response-locked cEMG analyses) because the effect of beverage on ERN/CRN and Pe/Pc mean amplitudes is not of central interest in this report. Furthermore, similar results were obtained when we re-analyzed the ERN/CRN and Pe/Pc mean amplitudes at the trial level using LMM, following Volpert-Esmond et al. (2018). For simplicity, only ANOVA results are presented.

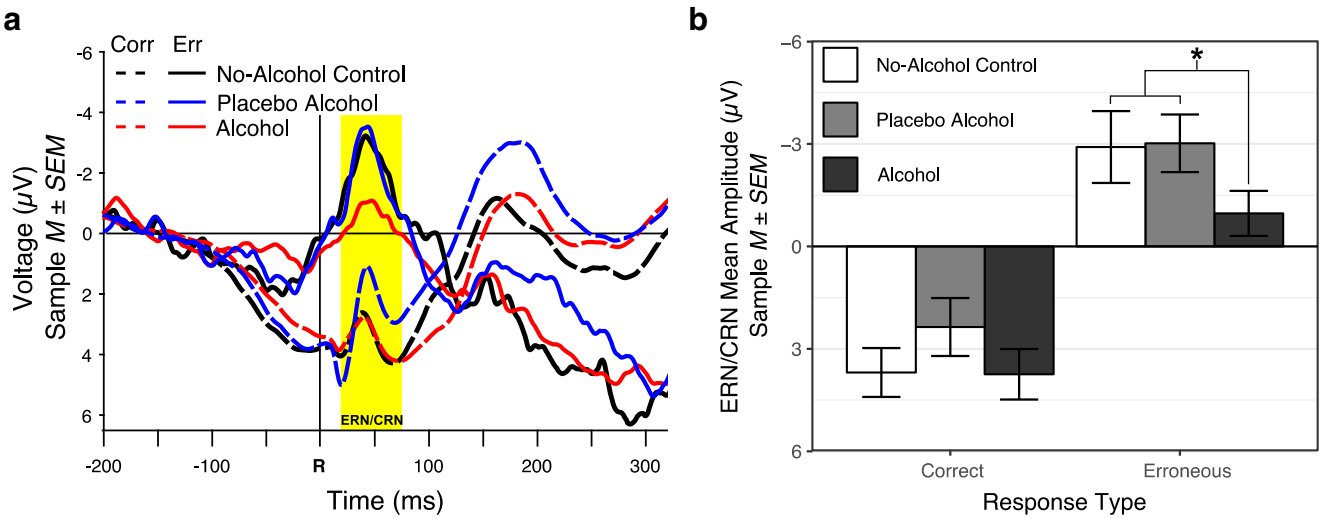


Fig. 3 *ERN/CRN as a function of beverage group and response accuracy*. **a** Waveforms shown represent the average across frontal, fronto-central, and central electrodes elicited by correct ("Corr") and erroneous ("Err") responses. "R" on the *x*-axis denotes time of button press. Note that the *y*-axis is reversed flowing convention for ERPs. Window (25–75 ms) for component mean amplitude quantification denoted by the yellow rectangle. **b** Mean amplitudes shown represent average across frontal, fronto-central, and central electrodes. Note that the *y*-axis is reversed because ERN/CRN are negative-going ERP components. Asterisk indicates $p < 0.05$. **a**, **b** Sample $M \pm$ SEM shown for the no-alcohol control group ($n = 21$), placebo alcohol group ($n = 25$), and alcohol group ($n = 28$)

positive) mean amplitude following erroneous compared to correct responses, $t(73) = 12.732$, 2-sided $p < 0.001$, $d = 2.980$.

Response-locked cEMG activity

Trial-by-trial response-locked corrugator EMG waveforms were analyzed using linear mixed models (LMMs) in R version 3.6.0 using packages lme4 (Bates et al. 2015), lmerTest (Kuznetsova et al. 2017), and emmeans (Lenth 2019) in order to best account for the nested structure of repeated psychophysiological measurements (E. Aarts et al. 2014; Page-Gould 2019). Technical details are presented in Online Supplemental Information.

Hypothesis 1 To test the prediction that response-locked cEMG activity would be enhanced on erroneous versus correct response trials, we considered the simple slopes of bin and bin^2 as a function of response accuracy. In keeping with our prediction, the simple slope of bin was more positive for erroneous compared to correct response trials, $\Delta\, b \pm \mathrm{SE} = 0.028 \pm 0.006$ μV/bin, $z = 5.022$, $p < 0.001$, and the simple slope of bin^2 was more negative for erroneous compared to correct response, $\Delta\ b \pm \mathrm{SE} = 0.0019 \pm 0.0005$ μV/bin^2, $z = 4.146$, $p < 0.001$.

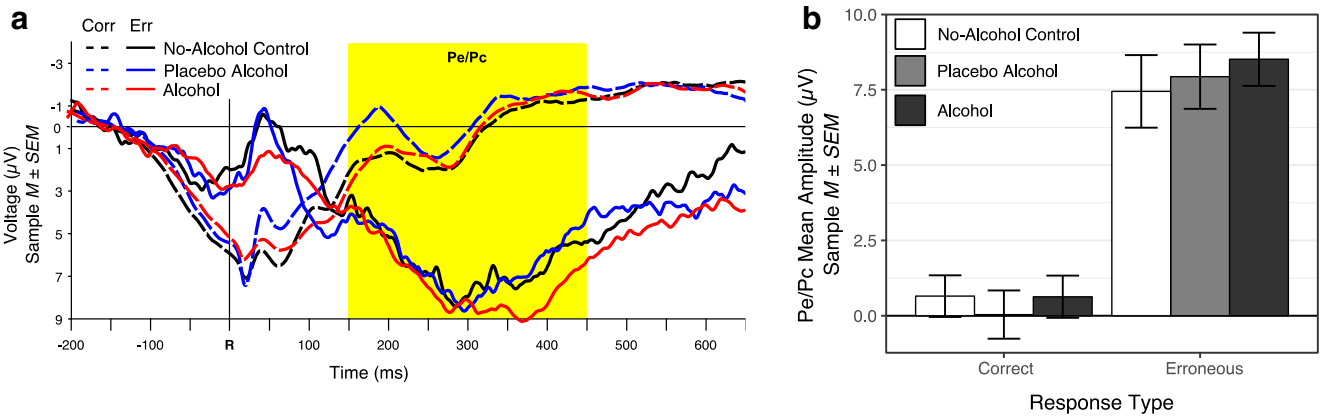


Fig. 4 *Pe/Pc as a function of beverage group and response accuracy*. **a** Waveforms shown represent the average across centro-parietal and parietal electrodes elicited by correct ("Corr") and erroneous ("Err") responses. "R" on the *x*-axis denotes time of button press. Note that the *y*-axis is reversed following convention for ERPs. Window (150–450 ms) for component mean amplitude quantification denoted by the yellow rectangle. **b** Mean amplitudes shown represent average across centro-parietal and parietal electrodes. **a**, **b** Sample $M \pm$ SEM shown for the no-alcohol control group ($n = 21$), placebo alcohol group ($n = 25$), and alcohol group ($n = 28$)

Hypothesis 2 To test the prediction that error-elicited cEMG activity would be dampened in the alcohol relative to control and placebo groups, we first considered the simple slopes of bin and bin^2 as a function of response accuracy within groups. We then considered whether the simple slopes of bin and bin^2 for erroneous response trials differed among groups. In keeping with our prediction, the simple slope of bin was more positive for erroneous compared to correct response trials in the control group, $\Delta\, b \pm \mathrm{SE} = 0.052 \pm 0.011$ μV/bin, $z = 4.839$, $p < 0.001$, and in the placebo group, $\Delta\, b \pm \mathrm{SE} = 0.029 \pm 0.010$ μV/bin, $z = 2.910$, $p = 0.004$, but not in the alcohol group, $\Delta\, b \pm \mathrm{SE} = 0.004 \pm 0.008$ μV/bin, $z = 0.494$, $p = 0.6211$. Similarly, the simple slope of bin^2 was more negative for erroneous compared to correct response trials in the control group, $\Delta\, b \pm \mathrm{SE} = 0.0037 \pm 0.0009$ μV/bin^2, $z = 4.101$, $p < 0.001$, and the placebo group, $\Delta\, b \pm \mathrm{SE} = 0.0027 \pm 0.0008$ μV/bin^2, $z = 3.389$, $p < 0.001$, but not the alcohol group, $\Delta\, b \pm \mathrm{SE} = -0.0006 \pm 0.0007$ μV/bin^2, $z = 0.863$, $p = 0.388$. Furthermore, the simple slopes of bin for erroneous response trials were significantly more positive for the control and placebo groups compared to the alcohol group, $\Delta\, b \pm \mathrm{SE} \geq 0.042 \pm 0.013$ μV/bin, $z \geq 3.350$, $p < 0.001$, and the corresponding simple slopes of bin^2 were significantly more negative, $b \pm \mathrm{SE} \leq -0.004 \pm 0.001$ μV/bin^2, $z \geq 3.398$, $p < 0.001$. The simple slopes of bin and bin^2 for erroneous response trials were not significantly different between the control and placebo groups, $z \leq 0.423$, $p \geq 0.672$.[4] Group differences are evident in the average and LMM-estimated response-locked cEMG waveforms (Fig. 5a, b).

Hypothesis 3 To test the prediction that error-elicited cEMG activity would correlate with error-elicited neural activity in the ACC, as reflected in the amplitude of the ERN and/or Pe components of the response-locked ERP, we added new effects to the best LMM of the trial-by-trial cEMG waveform. Since this hypothesis concerns within-trial associations within-persons, we first isolated within-person trial-by-trial changes in ERN and Pe following previous work from our laboratory (Von Gunten et al. 2018). Technical details are presented in Online Supplemental Information.

In keeping with our prediction, in the control group, erroneous response-elicited cEMG activity tended to be greater overall when the same-trial ERN was large (i.e., more negative), $b \pm \mathrm{SE} = -0.005 \pm 0.002$ μV cEMG per μV ERN, $z = -2.965$, $p = 0.003$. As shown in Fig. 6, this association was reversed in the placebo group, $b \pm \mathrm{SE} = 0.007 \pm 0.001$ μV cEMG per μV ERN, $z = 4.866$, $p < 0.001$, and nullified in the alcohol group, $b \pm \mathrm{SE} = 0.0005 \pm 0.0011$ μV cEMG per μV ERN, $z = 0.471$, $p = 0.638$. Also, in keeping with our prediction, for the control group, the simple slope of bin in erroneous response trials became significantly more positive with increasing (i.e., more positive) Pe, $b \pm \mathrm{SE} = 0.523 \pm 0.110$ μV cEMG per bin per μV Pe, $z = 4.746$, $p < 0.001$, and the corresponding simple slope of bin^2 became significantly more negative, $b \pm \mathrm{SE} = -0.0585 \pm 0.0092$ μV cEMG per bin^2 per μV Pe, $z = -6.385$, $p < 0.001$, effects evident in Fig. 7. In contrast, for the placebo group, the simple slopes of bin and bin^2 in erroneous response trials were not significantly affected associated with Pe, $z \leq 1.174$, $p \geq 0.241$. For the alcohol group, the simple slope of bin in erroneous response trials became significantly more negative with increasing (i.e., more positive) Pe, $b \pm \mathrm{SE} = -0.305 \pm 0.091$ μV cEMG per bin per μV Pe, $z = -3.352$, $p < 0.001$, and the corresponding simple slope of bin^2 became significantly more positive, $b \pm \mathrm{SE} = 0.025 \pm 0.008$ μV cEMG per bin^2 per μV Pe, $z = 3.332$, $p < 0.001$.

Hypothesis 4 To test the prediction that post-error cognitive control adjustments would be informed by affective reactivity to error commission, we first evaluated evidence for post-error reduction of interference (PERI) in the flanker task, a post-error behavioral effect linked to cognitive control processes (Burle et al. 2002; Danielmeier and Ullsperger 2011; King et al. 2010; Ridderinkhof et al. 2002). The flanker task interference effect manifests as lower accuracy (probability of correct response), and larger (slower) correct response times (RTs), on trials with correct response-incompatible relative to response-compatible flanker stimuli. Consequently, we fit LMMs of trial-by-trial accuracy and RT as a function of beverage group, current trial flanker type (correct response-compatible vs. response-incompatible), and previous trial response accuracy (correct vs. erroneous response) accounting for time on task. After determining the best LMMs of accuracy and RT, we added new effects corresponding to isolated within-person, trial-by-trial changes in previous trial response-locked cEMG activity, following Von Gunten et al. (2018). Technical details are presented in Online Supplemental Information. As shown in Fig. 8, PERI was evident in accuracy, but not in RT, and did not differ by beverage group. Other effects evident in the best LMMs (e.g., beverage effects on the flanker task interference effect, beverage effects on other post-error adjustments) are presented in Online Supplemental Information. Critically, contrary to our prediction, there was no evidence for associations between error-elicited cEMG on one trial and performance (accuracy or RT) on the next trial.

[4] For completeness, we also considered whether the simple slopes of bin and bin^2 for correct response trials differed among groups. Ordering of the simple slope of bin was as follows: placebo > alcohol > control, pairwise comparisons: $z \geq 2.337$, $p \leq 0.019$. Ordering of the simple slope of bin^2 was as follows: control ≥ alcohol ($z = 1.793$, $p = 0.073$) > placebo, pairwise comparisons: $z \geq 3.020$, $p \leq 0.005$. These patterns are consistent with highly specific cEMG reactivity to errors in the control group, and some specificity loss in the alcohol or placebo group, though most evident in the latter.

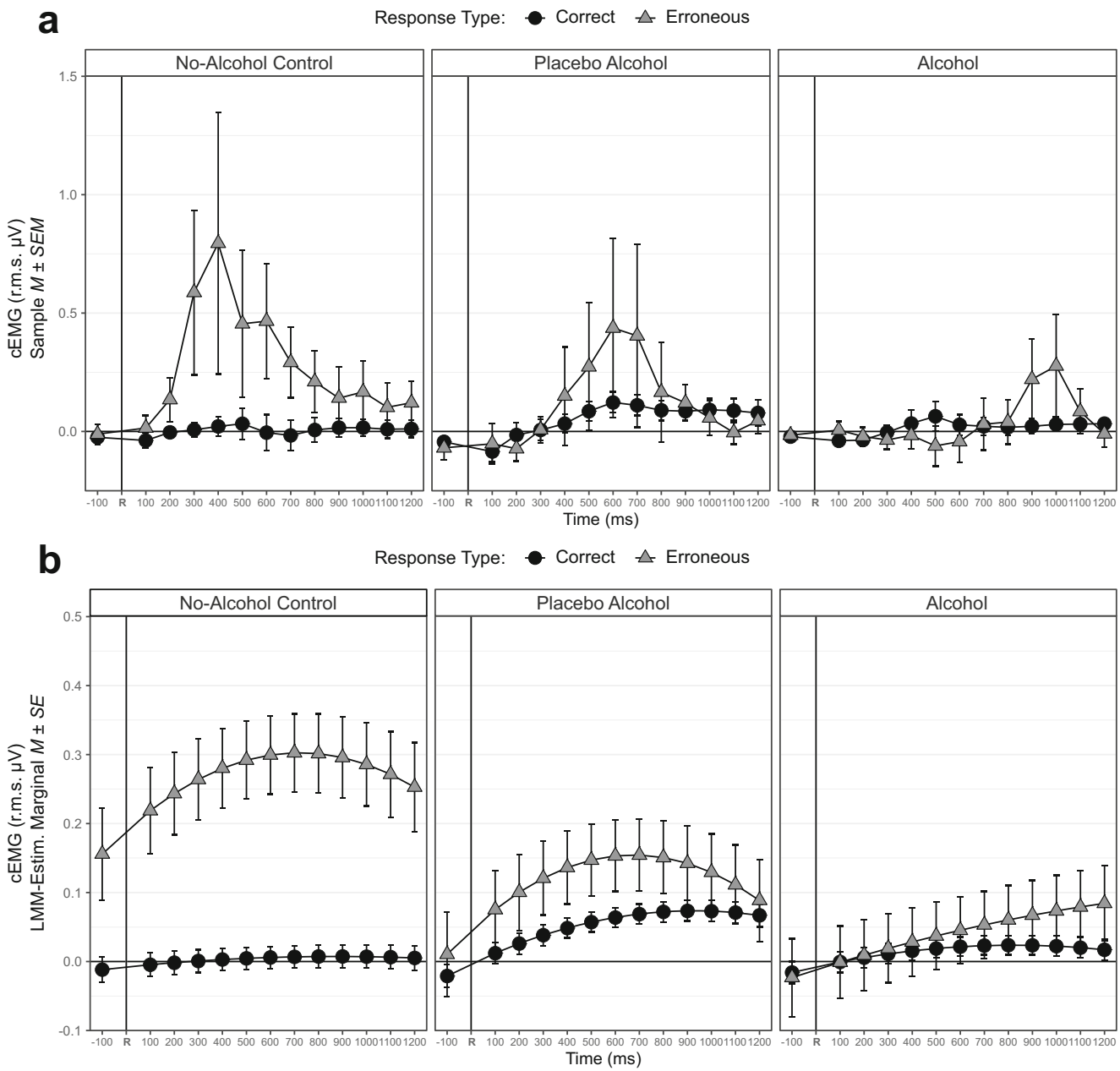


Fig. 5 *Response-locked cEMG activity as a function of beverage group and response accuracy*. **a** Sample *M* and SEM are shown. **b** Estimated marginal population *M* and SE from the best conditional LMM are shown. **a**, **b** Data represent $n = 21$ participants who consumed the no-alcohol control beverage, $n = 25$ who consumed the placebo alcohol beverage, and $n = 28$ who consumed the alcohol beverage. On the *x*-axes, "R" indicates button press. *Y*-axes differ between **a** and **b**. Some error bars are hidden underneath the point symbols

Discussion

The current study examined negative affective reactions to error commission in the flanker task following consumption of an alcohol, placebo, or control beverage by measuring response-locked cEMG activity. Four hypotheses were tested: (1) cEMG activity would be enhanced following erroneous versus correct responses; (2) error-elicited cEMG activity would be dampened in the alcohol group; (3) error-elicited cEMG activity and error-elicited neural activity in the ACC, as indexed by the ERN and/or Pe components of the response-locked ERP, would be correlated; and (4) error-elicited cEMG activity would be correlated with post-error adjustments in cognitive control task performance.

Hypothesis 1 was supported, replicating previous work on error-elicited cEMG activity (Berger et al. 2020; Dignath et al. 2019; Elkins-Brown et al. 2016, 2017; Lindström et al. 2013). Hypothesis 2 was also supported, providing empirical weight to the idea that alcohol consumption might decrease negative affective reactions to erroneous actions in effort-demanding

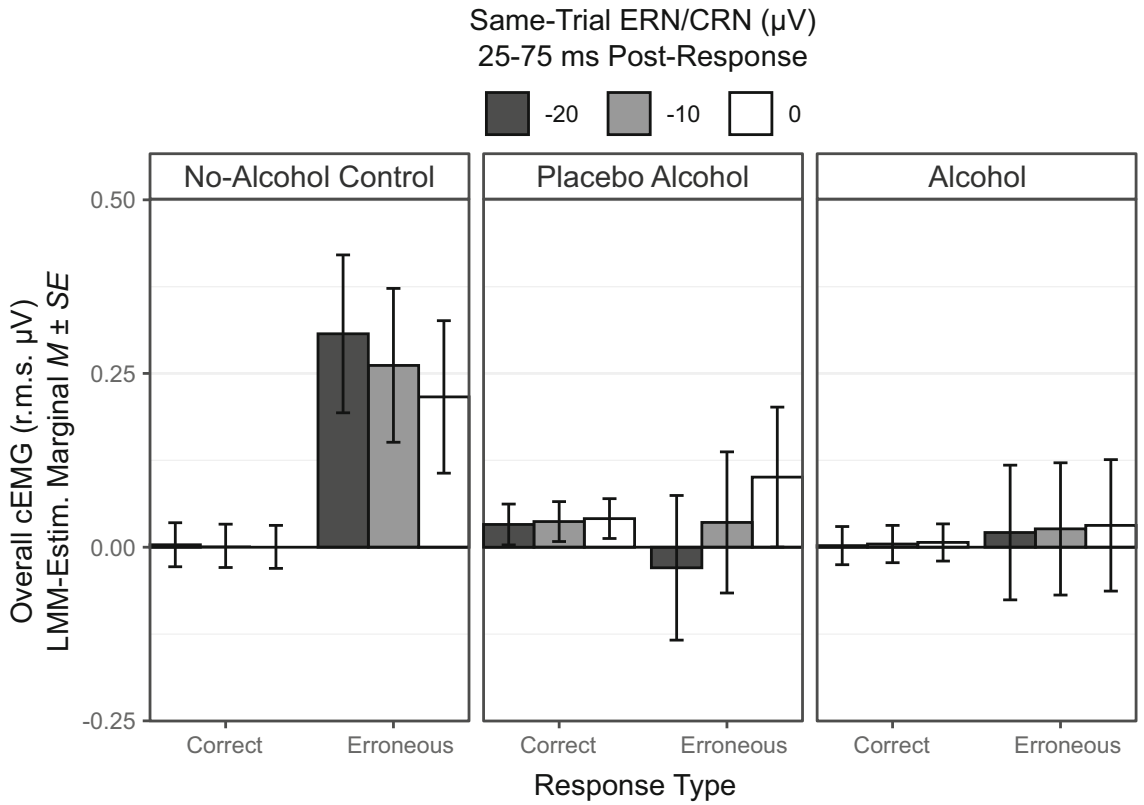


Fig. 6 *Overall response-locked cEMG activity as a function of beverage group, response accuracy, and ERN/CRN*. Marginal population *M* and SE estimated from best conditional LMM are shown. Data represent $n = 21$ participants who consumed the no-alcohol control beverage, $n = 25$ who consumed the placebo alcohol beverage, and $n = 28$ who consumed the alcohol beverage

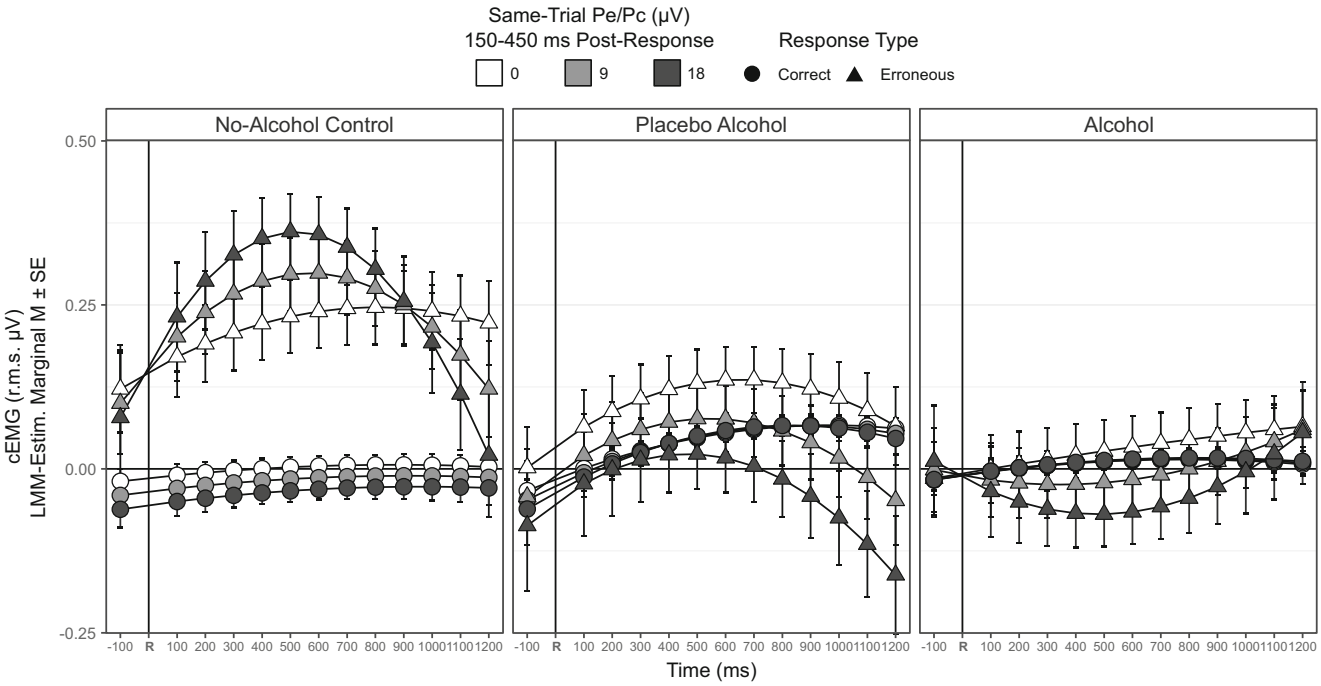
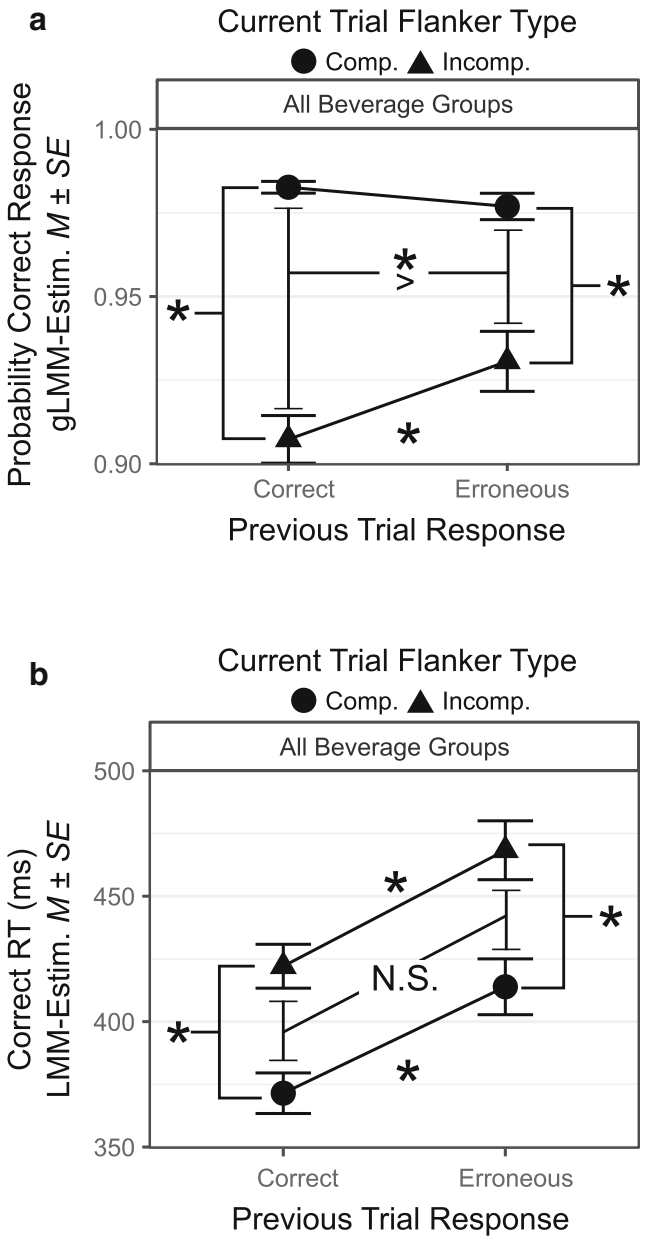


Fig. 7 *Response-locked cEMG activity as a function of beverage group, response accuracy, and Pe/Pc*. Marginal population *M* and SE estimated from best conditional LMM are shown. Data represent $n = 21$ participants who consumed the no-alcohol control beverage, $n = 25$ who consumed the placebo alcohol beverage, and $n = 28$ who consumed the alcohol beverage



◀ **Fig. 8** *Correct response probability and response time (RT) as a function of current trial flanker type and previous trial response accuracy*. **a** Back-transformed marginal population *M* and SE estimated from the best conditional generalized LMM (binomial, logit) of accuracy are shown. Post-error reduction of interference (PERI) was detected: difference in predicted probability of correct response when the current trial contains correct response-compatible vs. response-incompatible flankers was significantly diminished when previous trial response was incorrect relative to correct. PERI effect was driven by a significant increase in the probability of correct response on correct response-incompatible trials after an incorrect relative to correct response. **b** Marginal population *M* and SE estimated from best conditional LMM of RT are shown. PERI was not detected: difference in RT due to flanker type was similar post-error vs. -correct trial. **a**, **b** Data represent $n = 21$ participants who consumed the no-alcohol control beverage, $n = 25$ who consumed the placebo alcohol beverage, and $n = 28$ who consumed the alcohol beverage. Nevertheless, LMMs found no support for beverage × current trial type × previous trial response accuracy effects (these LMM-estimated marginal *M*s and SEs and corresponding sample *M*s and SEMs are presented in Supplemental Fig. 5). Consequently, only the current trial type × previous trial response accuracy effects are shown. Note that the current trial type × previous trial response accuracy effect was not significant for RT, and was ultimately dropped from the best LMM of RT used to test effects of previous trial cEMG. Note also that all LMM-estimated *M*s and SEs shown control for other effects evident in the LMMs (e.g., beverage × current trial type, beverage × previous trial response accuracy). Asterisk indicates $p < 0.05$

cognitive control tasks, as first proposed by Bartholow et al. (2012). Although not hypothesized, we also found that pre-trial cEMG activity in the flanker task was selectively dampened in the alcohol group (see Online Supplemental information). Given that response-locked cEMG activity was corrected for pre-response activity, this effect suggests that there may be acute effects of alcohol on both tonic affective state and phasic error reactivity. Previous studies using between-subject designs and similar participants have failed to find acute effects of alcohol on stimulus-locked cEMG in the context of a passive picture-viewing task (Curtin et al. 1998; Glautier et al. 2001; Stritzke et al. 1995), which is consistent with the idea that certain effects of alcohol are restricted to active and self-relevant contexts (for review, see Sayette 2017).

Hypothesis 3 was supported in the control group, providing *in principle* replication of findings reported by Elkins-Brown et al. (2016) and confirmation of ideas proposed by Lindström et al. (2013). This finding suggests that negative affective reactions to errors, at least as indexed by cEMG activity, are shaped by both early (ERN) and late (Pe) error processing-related neural activity in the ACC. Specifically, under normal circumstances, the magnitude of negative affect elicited by error commission may be set primarily by early ACC activity captured in the ERN, whereas how quickly that negative affective response is emitted relative to error commission and how quickly it dissipates appear to be tuned by later ACC activity captured in the Pe.

Hypothesis 3 was not supported in the alcohol or placebo group. Within-trial associations between ERN or Pe and error-elicited cEMG activity in these groups were inconsistent. ERN failed to predict overall error-elicited cEMG activity in the alcohol group, perhaps in keeping with selective dampening of the ERN in this group. In contrast, Pe predicted a dip below pre-response baseline for error-elicited cEMG activity in the alcohol group, although the Pe did not differ across groups. Nonetheless, error-elicited cEMG activity was largely abolished in the alcohol group. Consequently, it is possible that within-trial ERP-cEMG associations in the alcohol group are statistical artifacts. It is difficult to apply the same logic to explain within-trial ERP-cEMG associations in the placebo group. Erroneous actions still elicited cEMG activity in the

placebo group, even if less robustly than in the control group. Yet, in the placebo group, ERN predicted dips below pre-response baseline in overall error-elicited cEMG activity, and Pe failed to predict error-elicited cEMG activity. There were no differences in ERN or Pe between the placebo and control groups.

We are left with one substantive factor to explain differential ERP-cEMG coupling in the control versus placebo groups, namely, activation of alcohol use-outcome expectancies in the latter group. To the extent that affective reactions to error commission are shaped by attributional processes, then the expectation of sub-optimal performance due to alcohol (i.e., attribution to an external cause) could explain why error-elicited cEMG was uncoupled from ERN and Pe in placebo-consuming participants (see Testa et al. 2006). It also could explain dampened error-elicited cEMG in placebo- vs. control beverage-consuming participants despite equivalent ERN and Pe. Consequently, one reason why errors failed to elicit cEMG in alcohol-consuming participants may have been that they too were liable to externalize blame for their mistakes. There is some precedent for such an account (e.g., Critchlow 1987; Isleib et al. 1988). Future studies could test this idea by manipulating participants' beliefs about the locus of performance deficits prior to task completion (e.g., participants could be convinced that alcohol does *not* diminish performance on the task while covertly titrating task difficulty to promote error commission). Alternatively, participants' biases toward explicitly externalizing vs. internalizing success and failure could be measured (e.g., using false feedback) while sober vs. intoxicated.

Hypothesis 4 was not supported. Performance data provided no support for the idea that error-elicited negative affect is coupled to post-error adjustments in performance, or our contention that alcohol-induced diminution of error-elicited negative affect has implications for adjustment of cognitive control. In hindsight, we propose two reasons for this apparent failure. First, the experiment was not optimally designed to test questions about the association between affective reactivity to errors and subsequent control adjustment per se. Rather, it was designed specifically to test alcohol's effect on the first part of this premise, i.e., that alcohol reduces negative affective reactions to errors, as indicated by ERN and error-elicited cEMG activity. This was our primary interest. The experiment was designed to provide an alternative explanation for alcohol-induced reduction of the ERN, the first demonstration of which was reported by Ridderinkhof et al. (2002), who argued that alcohol reduced the ERN by impairing participants' ability to recognize when they made mistakes. Our alternative hypothesis has been that alcohol *does not* impair the ability to recognize when errors are made—indeed, data from this experiment (as reported in Bailey et al. 2014) and a separate experiment (Bartholow et al. 2012) strongly support this idea—but rather that alcohol reduces the ERN because it mollifies negative affective reactions to control failures—a prediction confirmed by the response-locked cEMG data reported here. The desire to test this alternative hypothesis led us to modify Ridderinkhof et al.'s original paradigm by introducing response accuracy confidence judgments following each trial. This design feature had the unfortunate side effect of introducing long inter-trial intervals (≈5 s) into the task, which make it difficult to test whether error-elicited negative affect on a given trial influences performance on the subsequent trial.

A second possible reason why we failed to find associations between error-elicited cEMG activity and subsequent-trial performance is that participants likely had little motivation to adjust their behavioral performance. There was no penalty for errors or slow responses (and no performance feedback during the experimental trials) and no real benefit for accurate or fast responses. Over the course of a nearly hour-long task, participants likely lost motivation for engaging control in a consistent manner. Thus, although the cEMG and ERN data support the idea that errors in the task elicited some negative affect, and that alcohol reduced affective reactions to errors, there likely was little motivation on the part of participants to translate affective reactions into control adjustments (e.g., see Boksem et al. 2006).

Findings from the current study should be considered in light of its strengths and weaknesses. The current study improved upon previous work in two important ways. First, it joins two studies (Dignath et al. 2019; Elkins-Brown et al. 2017) in showing that error-elicited cEMG activity can be observed in the absence of explicit feedback about response accuracy as well as in the absence of a threat (risk) of erroneous response-contingent punishment, which has been shown to amplify overall (Curtin et al. 1998) and error-elicited cEMG activity (Lindström et al. 2013). Second, the current study used statistical methods (i.e., LMM) that are arguably more statistically powerful yet conservative, if not at least more appropriate, for modeling psychophysiological data (E. Aarts et al. 2014; Page-Gould 2019). The use of this methodology allowed us to model the response-locked cEMG waveform, its sensitivity to within- and between-person factors, and change in the waveform across the task.

Despite these strengths, the current study was not without limitations. First, we tested a single alcohol dose in a between-subject design, yet effects of alcohol on affective state and reactivity can vary in extent, nature, and specificity with dose (Donohue et al. 2007; for review, see Sayette 2017). Second, participants were predominantly non-Hispanic White young adults. Third, we found no acute effect of alcohol on subjective affect, despite repeated assessment, including during the flanker task (see Online Supplemental Information).

However, subjective affect assessment during the task was not time-locked to errors, unlike in Spunt et al. (Spunt et al. 2012). Fourth, the probability of errors across the task was low, limiting the number of error trials available for analysis. Accuracy was titrated to be 90% correct, by design, to ensure that beverage effects on cognitive control task-related ERPs were not confounded by major differences in the frequency of correct versus incorrect responses. The relatively low frequency of errors in this study and others like it (Berger et al. 2020; Dignath et al. 2019; Elkins-Brown et al. 2016, 2017; Lindström et al. 2013) imposes psychometric constraints on measured responses to error commission independently of and/or in addition to the constraints imposed by the type of measurement (i.e., subjective, neuro/psychophysiological). Thus, future studies should parametrically manipulate the frequency of error commission.

Keeping in mind its strengths and weaknesses, the current study has implications for understanding the acute effects of alcohol on cognition. Specifically, its findings suggest that the acute effects of alcohol on canonical cognitive control tasks sometimes may reflect acute effects of alcohol expectancy and pharmacology on the affective underpinnings of action monitoring and, more broadly, cognitive control. Continued work on the affective underpinnings of cognitive control (Dignath et al. 2020; Inzlicht et al. 2015) stands to improve our basic understanding of not only decision-making and self-regulation but also how the latter might be affected by the acute and/or chronic effects of psychoactive substances, including alcohol.

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Compliance with ethical standards

All procedures were approved by the University of Missouri Institutional Review Board.

Conflict of interest The authors declare that they have no conflict of interest.

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