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Transcranial direct current stimulation over the ventrolateral prefrontal cortex in intoxicated individuals during anger regulation

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Abstract

Acute alcohol consumption impairs anger regulation by disrupting prefrontal cortical activity. We tested whether transcranial direct current stimulation (tDCS) over the right ventrolateral prefrontal cortex (rVLPFC) reduces anger in intoxicated vs. sober participants during instructed regulation of recalled anger. One hundred ninety-six healthy adults consumed alcohol or no beverage and received anodal or sham tDCS over the rVLPFC. During stimulation, participants used cognitive reappraisal, distraction, or rumination to regulate recalled anger. As expected, anger was lowest after distraction, intermediate after reappraisal, and highest after rumination. Contrary to predictions, there were no main effects of alcohol, stimulation, or their interaction. Exploratory analyses indicated that among intoxicated participants high in trait anger, anodal stimulation reduced anger following rumination compared to sham. Alcohol also amplified anger in individuals prone to angry rumination. These findings highlight the need for replication and refinement of tDCS protocols to better target intoxicated anger regulation.

Introduction

The ability to regulate the subjective experience of emotions is critical for the healthy functioning of both individuals and societies. Emotion regulation refers to the processes through which individuals shape their emotional experience (Gross & Thompson, 2007). Anger regulation is particularly important because, if left unchecked, anger can lead to aggression and violence (e.g., Anderson & Bushman, 2002; Berkowitz, 2012), as well as adverse health outcomes such as cardiovascular disease (Titova et al., 2022). However, regulating anger and aggression is not always easy, particularly for intoxicated people (Parrott & Giancola, 2004; Neilson et al., 2022). Understanding the brain mechanisms involved in regulating anger when intoxicated and sober may facilitate interventions to reduce the harms associated with anger. In this paper, we will examine the effects of alcohol intoxication and non-invasive brain stimulation on anger during three common emotion regulation strategies: distraction, cognitive reappraisal, and angry rumination.

Distraction involves directing attention away from negative emotion-inducing stimuli to unrelated stimuli. For instance, an angered person engaging in distraction might play a puzzle game on their smartphone or visualize walking around campus (Pedersen et al., 2011; Sheppes & Levin, 2013). Cognitive reappraisal involves mentally re-evaluating a situation to reduce its emotional impact (Gross, 2002). For instance, when reappraising, a provoked person might engage their attention with the provocation by reconsidering the event from a third-person, neutral perspective, or think about lessons they may have learned. Angry rumination refers to perseverative thinking about a personally meaningful anger-inducing event (Bushman, 2002; Denson, 2013). It also involves attentional processes; however, someone engaging in angry rumination will tend to focus on revenge and rehash the provocation repeatedly. Thus, angry

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rumination can increase or maintain the anger experience. A meta-analysis found that cognitive reappraisal was associated with lower anger and rumination was associated with heightened anger (Pop et al., 2025).

Both cognitive reappraisal and distraction are widely considered to be effective strategies for lowering anger (Denson et al., 2012; Etkin et al., 2015; Rusting & Nolen-Hoeksema, 1998; Ursu et al., 2025). In contrast, angry rumination is considered a maladaptive emotion regulation strategy that can maintain or intensify anger and aggression (Bushman 2002; Rusting and Nolen-Hoeksema 1998; Denson 2013; Denson et al. 2011a, b, 2012; Oliva et al. 2023). Relevant to the present research, one study found that alcohol combined with angry rumination increased intimate partner aggression (Watkins et al., 2015).

In healthy and disordered individuals, brain regions associated with emotion regulation are recruited within seconds of an anger-inducing event (Denson et al. 2009; Gilam et al. 2017a, b; Peters et al. 2018). Emotion regulation processes activate a network of prefrontal cortical regions that interact with limbic regions implicated in emotional reactivity (Etkin et al., 2015; Gilam et al., 2015). The prefrontal cortex (PFC) is involved not just in downregulation of negative affect, but also the upregulation of negative affect. For example, the PFC is implicated in cognitive reappraisal and distraction, but is also active during anger rumination (Blair, 2024; Denson et al., 2009; Kelley et al., 2013; Peters et al., 2018).

Within the prefrontal network, the ventrolateral prefrontal cortex (VLPFC) is critical in facilitating adaptive and maladaptive emotion regulation (Buhle et al., 2014; Li et al., 2022). The VLPFC is engaged during emotion recognition and regulation processes (Wager et al., 2008) and may function to suppress the amygdala's role in eliciting negative emotions during reappraisal and distraction (Banks et al., 2007). During angry rumination, the VLPFC may facilitate positive connectivity between the VLFPC and limbic regions such as the amygdala. Peters et al. (2018) found that people with borderline personality disorder, which is characterized by extreme anger, angry rumination, and emotion dysregulation, showed an immediate increase in activity in the VLPFC when insulted compared to healthy participants when provoked in the scanner. The borderline participants also showed greater activity in the nucleus accumbens during directed angry rumination, suggesting that some people may experience positive affect during angry rumination. The authors did not examine functional connectivity, but it seems plausible that there would be positive connectivity between the VLFPC and nucleus accumbens during angry rumination. Thus, it is possible that anodal tDCS to the VLPFC may enhance anger by increasing ruminative processing and its associated rewarding aspects.

Using a very similar design to the present research, another fMRI study asked participants to recall an anger-inducing autobiographical experience while engaging in cognitive reappraisal or two types of angry rumination (Fabiansson et al., 2012). Anger was highest in the angry rumination conditions followed by the reappraisal condition. During rumination, results showed positive functional connectivity between the VLPFC and the amygdala and thalamus, which is consistent with the upregulation of anger during rumination. In another study, increased connectivity between the VLPFC and amygdala following interpersonal anger provocation positively associated with trait anger and predicted long term traumatic stress symptoms (Gilam et al. 2017a, b). Thus, these findings suggest that anodal stimulation to the VLPFC may augment ruminative processing via positive connectivity with limbic regions.

In the present research, we also investigated the effects of acute alcohol intoxication on anger. Alcohol disrupts functioning of prefrontal cortical regions involved in emotion regulation via inhibition through GABA release (an inhibitory neurotransmitter) and by blocking glutamate (an excitatory neurotransmitter) (Heinz et al., 2011). This causes a general dampening effect of alcohol on activity in prefrontal regions. Both acute and chronic exposure to alcohol can alter the structure and function of the prefrontal cortex (Abernathy et al., 2010). Indeed, alcohol consumption is thought to disrupt emotion regulation ability by altering the interaction between prefrontal cortical regions and limbic regions such as the amygdala (Gorka et al., 2013; Heinz et al., 2011). For instance, one fMRI study of heavy drinkers reported that acute alcohol intoxication reduced functional coupling between the orbitofrontal cortex and amygdala when viewing angry faces (Gorka et al., 2013). In another fMRI study, participants at risk of heavy drinking showed reduced functional coupling between the amygdala and frontal lobes when viewing angry faces (McKenna et al., 2022). Another study showed participants with alcohol use disorder and control participants emotional faces and examined effective connectivity. Results showed that there was substantial connectivity between the prefrontal cortex, including the VLPFC, in participants with alcohol use disorder. Furthermore, acute alcohol consumption is associated with increased anger and aggression (Bushman & Cooper, 1990; Duke et al., 2018; Laitano et al., 2022).

Neuromodulation methods such as transcranial direct current stimulation (tDCS) allow researchers to selectively modulate cortical functioning and offer promise as methods of intervention to counteract the alcohol-dampening effects on prefrontal activity. Evidence suggests that in sober participants, anodal tDCS over the VLPFC can reduce negative emotions (e.g., He et al., 2018; Vergallito et al., 2018), and inhibit aggressive behaviour (e.g., Gallucci et al. 2020; Riva

et al. 2015a, b, 2017; Sergiou et al. 2020). However, two recent meta-analyses revealed no significant main effects of non-invasive brain stimulation on anger and aggression (Denson et al., 2025; Romero-Martinez et al., 2025), but the literature was severely underpowered (average sample size of 46 and power of 0.33; Denson et al., 2025). Nonetheless, it remains possible that there are untested moderators. In the present research we examined whether alcohol can moderate the effect of tDCS on anger regulation. Because alcohol exerts a dampening effect on activity within the prefrontal cortex, we expected that the enhanced excitatory effect of anodal stimulation would counteract this effect of acute alcohol intoxication. That is, during cognitive reappraisal and distraction, the enhanced activation in the VLPFC should presumably facilitate greater top-down regulation of the VLPFC over limbic regions, thereby resulting in lower anger. By contrast, during angry rumination, anodal stimulation is expected to counteract the alcohol-dampening effect and thereby increase positive connectivity between the VLPFC and subcortical regions, thereby resulting in greater anger (e.g., Fabiansson et al., 2012).

Here, we test a randomized, sham-controlled, double-blind procedure to examine the efficacy of anodal tDCS over the rVLPFC during explicit anger regulation in intoxicated (vs. sober) people. Although the effects of distraction, cognitive reappraisal, and rumination on anger are well understood, this study fills a critical gap in the literature as the first to examine tDCS effects on anger regulation in intoxicated participants. We predicted that participants in the alcohol condition would report greater anger following the recall task, compared to sober participants. We also predicted that participants would report less anger following cognitive reappraisal and distraction, compared to angry rumination. In line with past research, which found that anodal stimulation over the right DLPFC increased rumination (Kelley et al., 2013), we predicted that in the sober condition, anodal stimulation would increase anger following angry rumination, compared to sham stimulation. Thus, we predicted that anodal stimulation would elicit increased levels of anger in the rumination condition, regardless of alcohol intoxication. Lastly, we predicted that in the alcohol condition, anodal stimulation would reduce anger following reappraisal and distraction, compared to sham stimulation and rumination conditions.

Method

This study was approved by the Human Research Ethics Committee at The University of New South Wales (UNSW; HC200922) and preregistered on the Open Science Framework (https://osf.io/nk5se). Data and R code are available on the Open Science Framework (https://osf.io/mf7zw).

Participants

Participants were required to be aged 18–40, right-handed, not currently depressed or anxious, and consume alcohol at least once per month. Participants were excluded from participating if they had a physical or psychiatric health condition that alcohol might worsen, were taking medications which may interact with alcohol, had a history of head trauma, or have experienced adverse reactions to alcohol. Participants were also excluded if they scored > 15 on the AUDIT, failed to pass a tDCS safety screen, or were currently pregnant.

Consistent with our preregistration, 200 participants were recruited from UNSW and surrounding areas to take part in a study about brain stimulation, alcohol, and recalling past events. Students were remunerated with course credit, and community members were remunerated at a rate of $20/hr. Data from four participants were excluded from analysis (2 = technical difficulties, 1 = did not tolerate tDCS, 1 = suspicion).

The final sample comprised 196 participants (61.73% female; M_{age} = 19.49, SD = 2.50). Participants' reported ethnic backgrounds were Aboriginal/Torres Strait Islander (0.51%), Asian (49.49%), Caucasian (35.20%), Middle Eastern (3.06%), other (11.23%), and did not report (0.51%). Participants reported educational levels at Vocational Qualification (1.02%), Year 12 or equivalent (80.10%), Bachelors' degree (11.23%), Undergraduate diploma (5.01%), Associate Diploma (1.53%), and Masters degree (1.02%).

Design

The study was a 2 (tDCS: sham, anodal) × 2 (alcohol: alcohol, no drink) × 3 (emotion regulation strategy: cognitive reappraisal, angry rumination, distraction) counterbalanced mixed design. Participants were randomly assigned to a single level of each between-subjects factor (alcohol and tDCS). A double-blind procedure was in place for stimulation conditions; however, neither experimenter nor participants were blind to alcohol condition. Emotion regulation strategy was a counterbalanced within-subjects variable (cognitive reappraisal, distraction, rumination). The primary dependent variable was self-reported state anger. Trait anger and aggression, trait angry rumination, trait difficulties in emotion regulation, alcohol consumption habits, breath alcohol concentration, and several state emotions were also measured.

Procedure

Upon arrival, participants provided informed consent and demographic information and recorded a baseline breath alcohol content (BrAC) reading with an Alcolizer LE breathalyzer (Alcolizer, Australia). Participants in the alcohol condition were then administered two alcoholic drinks. Female participants in the alcohol condition were required to return a negative urine pregnancy test before consuming alcohol. All participants completed a series of baseline questionnaires and tDCS electrodes were applied. Participants then completed an autobiographical anger recall task, before stimulation was initiated. Participants were then instructed to complete three emotion regulation tasks (i.e., reappraisal, rumination, distraction) during anodal stimulation or sham. To conclude, participants responded to funnel debriefing questions, were verbally debriefed, and paid. Participants in the alcohol condition were breathalysed immediately before and after the anger recall and emotion regulation tasks. They were required to remain in the laboratory until they recorded a breath alcohol concentration below 0.05%.

Materials

Alcohol administration

Participants in the alcohol condition consumed two alcoholic beverages containing 0.70 g of alcohol per kg of lean body mass (alcohol $M = 40.68$ g, $SD = 12.46$ g; body weight $M = 65.52$ kg, $SD = 12.96$ kg). Each alcoholic beverage comprised 37% ABV vodka and sugar-free lemonade at a ratio of 1:3. A maximum dose of 50 g of alcohol (five standard drinks) was administered to avoid sickness. BrAC was assessed with a calibrated Alcolizer LE5 breathalyser. Participants in the control condition did not consume a drink. We opted not to administer a placebo as placebos can influence neural activity in brain regions implicated in emotion regulation (Price et al., 2008) and alcohol placebos are rarely, if ever, encountered in daily life.

Baseline and trait measures

Participants completed the Alcohol Use Disorders Identification Test (AUDIT; Saunders et al., 1993), Difficulties in Emotion Regulation Scale (DERS; Gratz & Roemer, 2004), Aggression Questionnaire (AQ; Buss & Perry, 1992), Discrete Emotions Questionnaire (DEQ; Harmon-Jones et al., 2016), and the Angry Rumination subscale of the Displaced Aggression Questionnaire (DAQ; Denson et al., 2006).

Anger recall task

As in Denson et al. (2012) participants were asked to recall a time when they felt angry. They were told this should be an event that occurred within the last year that involved another person, and that it should not be a time when they were angry with themselves. Participants were asked to write about the details of this event for 5 min. Following this task, participants rated how angry they felt during the event, how angry they feel about the event now, and how angry they were currently feeling on scales from 1 (*not at all angry*) to 7 (*extremely so*).

Stimulation

Transcranial direct current stimulation (tDCS) was administered with a DC-Stimulator Plus (NeuroConn GmbH, Germany). The anode (5 $cm^2 \times 5$ cm^2) was placed on the scalp at F6, and the cathode (5 $cm^2 \times 7$ cm^2) was placed contralaterally on the scalp at F5. To improve conductivity, each electrode was placed inside a sponge pouch soaked in saline 0.9% sodium chloride. A small amount of antiseptic cream was applied to reduce discomfort. Anodal stimulation was administered for 20 min at a constant current of 1.5 mA. Stimulation included an 8-second fade-in interval and a 5-second fade-out period (Riva et al. 2015a, b). Participants in the sham condition only received active stimulation for a few seconds at 1.5 mA current intensity during the ramp up and ramp down periods with identical fade-in and fade-out parameters. These parameters are effective at blinding participants and replicating the sensations of stimulation without modulating neural circuitry (Riva et al. 2015a, b).

Emotion regulation task

As in past research (e.g., Denson et al., 2012; Fabiansson et al., 2012), participants were instructed to think about the anger inducing event with guided prompts for each of three counterbalanced emotion regulation strategies: cognitive reappraisal, distraction, and rumination. For each strategy, participants were given specific instructions, followed by a series of six prompts (see Table 1). Participants were given 30 s each to respond to each prompt. A 30 s break was included between each condition to mitigate carry over effects. Following each emotion regulation strategy, participants reported how angry they were currently feeling on a 7-point scale ranging from 1 (*not at all*) to 7 (*extremely so*).

Table 1 Instructions and prompts for the emotion regulation strategies

Instruction	Cognitive Reappraisal	Distraction	Rumination
	I want you to think about the anger-inducing event that you wrote down previously. However, this time I want you to write about it in a different, more objective and positive way. Try to write about some positive aspects of the event, such as lessons you have learned, and ways that you could improve in the future if the same event were to arise. Also, try to write about factual, non-emotional details, such as where and when the event occurred.	Please describe the layout of the UNSW campus as you see it in your mind and how you would describe it to someone who has never been here before. Please write a thorough and detailed description.	I want you to think about the anger-inducing event that you wrote down previously. However, this time I want you to write about it in an emotional way. Try to write about your feelings and the emotional aspects of the event, such as anger toward others, and how you might feel if the same event were to occur again. Try to focus on emotional details such as angry feelings and hostile thoughts.
Prompt 1	Think about the non-emotional details of the event, such as where and when it occurred.	Think about where the entrances to the UNSW campus are.	Think about the anger-related physical sensations you experienced during the event.
Prompt 2	Think about the event from the perspective of an objective, impartial third person.	Think about the landscaping of the UNSW campus.	Think about the feelings and emotions you felt toward the other people in the event.
Prompt 3	Think about any valuable lessons you have learned from the event.	Think about the size and scale of the UNSW campus.	Think about exactly what happened during the event.
Prompt 4	Think about the event in a way to maintain a neutral mood.	Think about where specific buildings are located.	Think about how you might like to retaliate in response to the event.
Prompt 5	Think about any positive aspects of the event.	Think about where the amenities such as bathrooms and cafes are located.	Think about your angry mood during the event.
Prompt 6	Think about ways you could improve in the future if the same event were to arise.	Think about how the UNSW campus looks from a bird's eye view.	Think about the feelings and emotions you experienced during the event.

Statistical analyses

All analyses were conducted in R (R Core Team, 2023). Multilevel generalized linear mixed effects models were conducted with the *lme4* (Bates et al., 2015) and *lmerTest* packages (Kuznetsova et al., 2017); R^2 was calculated with Nakagawa and Schielzeth's (2013) method from the p*erformance* package (Lüdecke et al., 2021). We specified participant as a random effect and alcohol, tDCS, emotion regulation task, and their interactions as fixed effects. Outliers defined as having residuals > |3 *SD*s| were omitted from analyses, which resulted in the removal of 0.68% of trials being omitted from our analyses (i.e., 4 out of 588 trials).

Results

Randomisation and manipulation checks

At baseline, experimental groups differed significantly on trait physical aggression and state disgust. Results did not differ when these variables were included in the model (see Supplementary Materials). Experimental groups did not differ on any other demographic, state, or trait measures (see Supplementary Materials).

All participants had a baseline breath alcohol concentration (BrAC) of 0.00%. For participants in the alcohol condition, mean BrAC readings at Time 2 (0.08%), Time 3 (0.08%), Time 4 (0.08%) and Time 5 (0.08%) indicated stable levels of intoxication across the experiment. BrAC at all time points did not differ significantly from each other (adjusted *p*s > 0.918). Within the alcohol condition, BrAC did not differ between anodal tDCS and sham conditions at any of the timepoints (adjusted *p*s > 0.777).

Primary analyses

A 2 (drink: alcohol, no alcohol) × 2 (stimulation: active, sham) × 3 (anger regulation: rumination, distraction, reappraisal) linear mixed effects model examined self-reported anger as a function of the experimental manipulations. The full model was a significantly better fit for the data than an intercept-only model, $c^2(11) = 200.21$, $p < .001$, Adjusted $ICC = 0.527$. The total explanatory power of the full model was greater when including both fixed and random effects ($R^2 = 0.61$) compared to fixed effects alone ($R^2 = 0.17$).

We observed main effects of emotion regulation conditions (Fig. 1; Table 2). Follow-up pairwise comparisons indicated all emotion regulation conditions differed significantly from one another (*p*s < 0.001). No other main effects or interactions were observed.

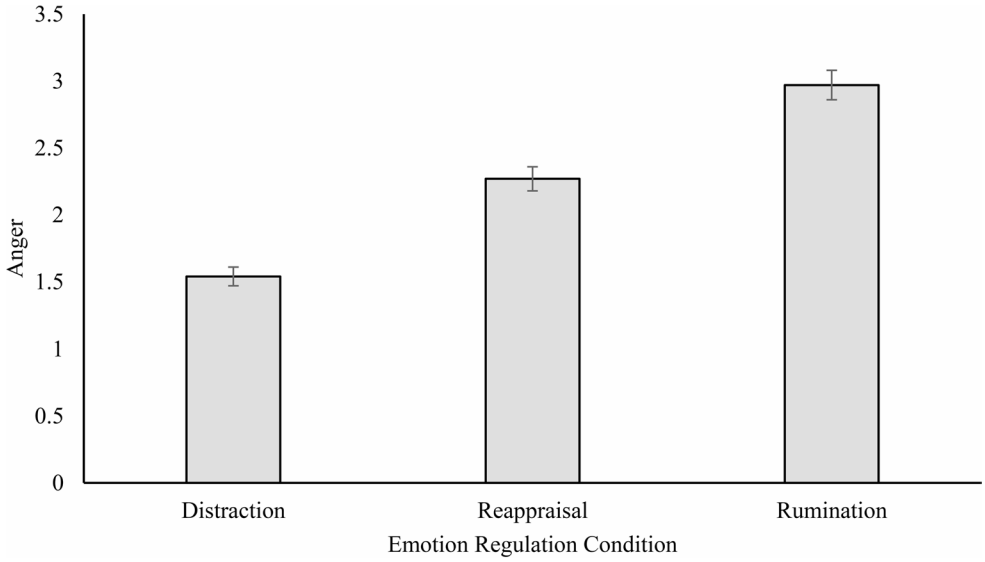


Fig. 1 Self-reported state anger following the anger recall task

Note. Error bars represent standard errors.

Table 2 Interactive effects of alcohol, stimulation, and emotion regulation task on self-reported anger

	Estimate	SE	df	t	Pr(>|t|)
(Intercept)	2.18	0.19	374.18	11.72	.000***
Alcohol	-0.02	0.26	371.42	-0.09	.932
Stimulation	-0.09	0.27	374.53	-0.34	.731
Distraction	-0.60	0.18	380.83	-3.35	.001***
Rumination	0.89	0.18	380.83	4.90	.000***
Alcohol × Stimulation	0.51	0.37	371.67	1.37	.172
Alcohol × Distraction	-0.07	0.25	380.31	-0.27	.784
Alcohol × Rumination	-0.12	0.25	380.31	-0.46	.643
Stimulation × Distraction	0.06	0.26	381.58	0.24	.810
Stimulation × Rumination	-0.04	0.26	381.58	-0.16	.870
Alcohol × Stimulation × Distraction	-0.41	0.36	380.72	-1.13	.258
Alcohol × Stimulation × Rumination	-0.39	0.36	380.72	-1.09	.278

Note. * = $p < .05$; ** = $p < .01$; *** = $p < .001$. Reference groups were no alcohol, sham stimulation, and reappraisal

Exploratory analyses

To examine the influence of theoretically relevant individual difference factors, we ran exploratory models that included trait anger, angry rumination, and difficulties in emotion regulation as fixed factors, respectively. To investigate interactions, we used the *emtrends* package (Lenth, 2023) to estimate the simple slopes for each individual difference at each level of alcohol, stimulation, and emotion regulation.

As past research has observed sex-related differences in tDCS outcomes (e.g., Battacharjee et al., 2025 Gragmena et al., 2025), we also tested the moderating effect of gender. The results of the primary analysis remained unchanged when including gender as a fixed factor (see Supplementary Materials). Additionally, to account for potential alcohol tolerance in participants who more frequently consume alcohol, we also including past-month alcohol use as a covariate. The results of the primary analysis remained unchanged when controlling for alcohol use (see Supplementary Materials).

Trait anger

When trait anger was added to the model, we observed a significant alcohol × stimulation × emotion regulation × trait anger interaction, b = -1.27, SE=0.51, t(372.88) = -2.46, p = .014 (see Table 3; Fig. 2). For intoxicated participants, trait anger slopes significantly differed between anodal and sham stimulation conditions following rumination, estimate = -1.17, SE=0.39, t(358) = -3.01, p = .003. For intoxicated participants who received sham stimulation, self-reported anger following rumination increased by 1.38 points for every one-point increase in trait anger (SE=0.25, CI_{95} [0.89, 1.87]), compared to just 0.22 points in the anodal

Table 3 Model including trait anger

	Estimate	SE	df	t	Pr(>|t|)
(Intercept)	1.05	0.58	363.95	1.81	.072
Alcohol	0.28	0.81	361.03	0.35	.727
Stimulation	0.73	0.84	363.21	0.87	.383
Distraction	0.24	0.56	372.87	0.43	.671
Rumination	0.62	0.56	372.87	1.10	.274
Trait Anger	0.49	0.24	361.22	2.04	.042*
Alcohol × Stimulation	-0.23	1.19	360.58	-0.19	.846
Alcohol × Distraction	-0.46	0.78	372.32	-0.59	.555
Alcohol × Rumination	-2.02	0.78	372.32	-2.58	.010*
Stimulation × Distraction	-0.17	0.81	372.74	-0.22	.830
Stimulation × Rumination	-0.26	0.81	373.27	-0.33	.745
Alcohol × Trait Anger	-0.11	0.35	359.55	-0.33	.740
Stimulation × Trait Anger	-0.35	0.36	364.66	-0.97	.332
Distraction × Trait Anger	-0.37	0.23	372.36	-1.58	.116
Rumination × Trait Anger	0.12	0.23	372.36	0.51	.609
Alcohol × Stimulation × Distraction	-0.49	1.15	372.24	-0.42	.672
Alcohol × Stimulation × Rumination	2.31	1.15	372.51	2.01	.045*
Alcohol × Stimulation × Trait Anger	0.34	0.53	361.08	0.64	.521
Alcohol × Distraction × Trait Anger	0.16	0.33	372.04	0.49	.628
Alcohol × Rumination × Trait Anger	0.88	0.33	372.04	2.63	.009**
Stimulation × Distraction × Trait Anger	0.08	0.35	373.15	0.24	.809
Stimulation × Rumination × Trait Anger	0.11	0.35	374.17	0.32	.751
Alcohol × Stimulation × Distraction × Trait Anger	0.05	0.51	372.40	0.11	.915
Alcohol × Stimulation × Rumination × Trait Anger	-1.27	0.51	372.88	-2.46	.014*

Note. * = $p < .05$; ** = $p < .01$; *** = $p < .001$. Reference groups were no alcohol, sham stimulation, and reappraisal

condition ($SE = 0.30$, CI_{95} [-0.37, 0.80]). No other significant differences were observed.

Angry rumination

When trait angry rumination was added to the model, we observed a significant alcohol × emotion regulation × trait angry rumination interaction, $b = 0.42$, $SE = 0.19$, $t(372.94) = 2.21$, $p = .028$ (see Table 4; Fig. 3). Trait anger slopes differed significantly between alcohol conditions following rumination, estimate = -0.33, $SE = 0.14$, $t(367) = -2.32$, $p = .021$. For participants in the alcohol condition self-reported anger following rumination increased by 0.57 points for every one-point increase in angry rumination ($SE = 0.11$, CI_{95} [0.05, 0.42]), compared to just 0.24 points in the no alcohol condition ($SE = 0.11$, CI_{95} [0.36, 0.78]. No other significant differences were observed.

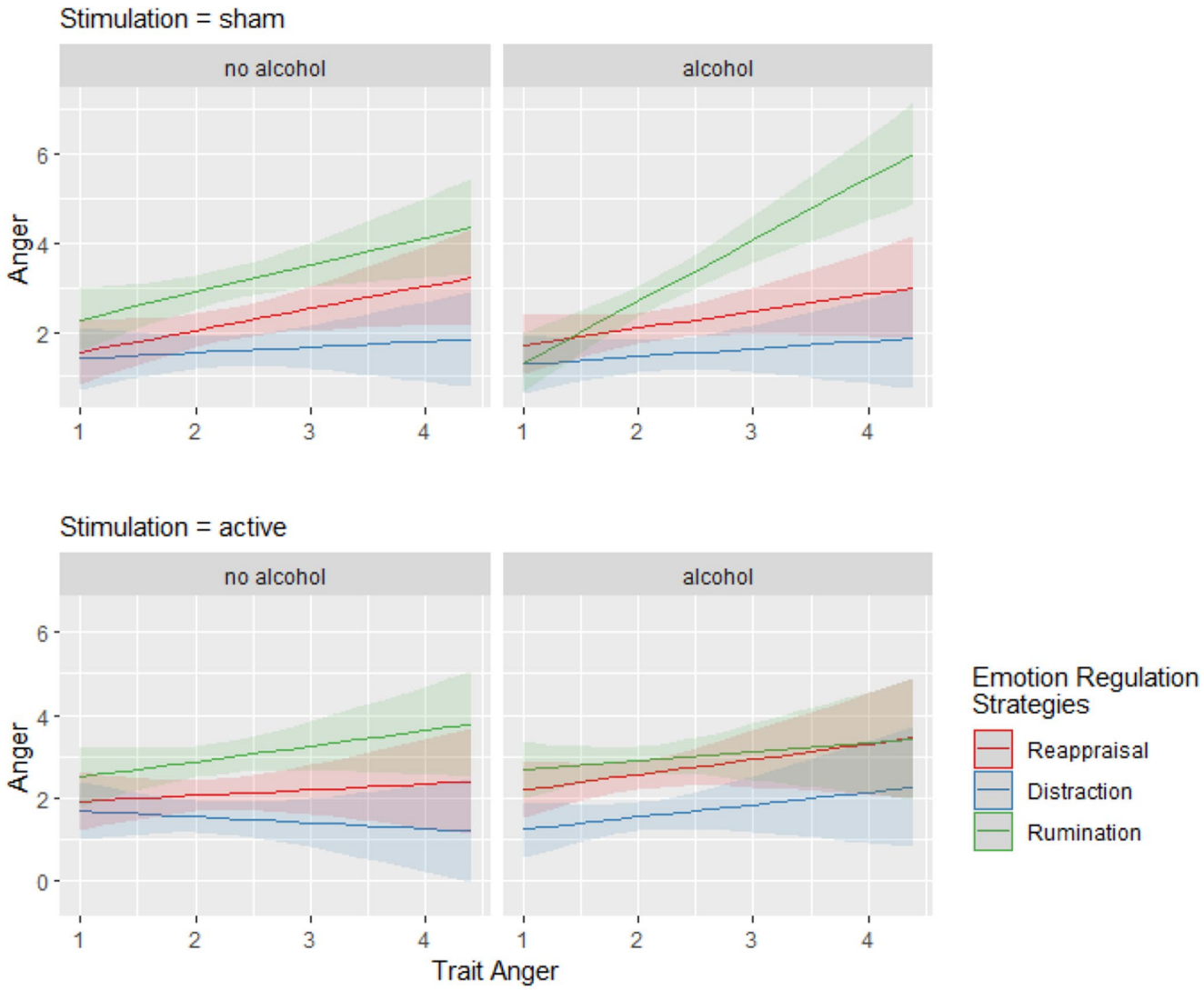
Difficulties in emotion regulation

To account for trait emotion dysregulation, difficulties in emotion regulation (DERS) were included as a fixed factor in the primary model. When DERS scores were added to the model, we observed a main effect of DERS such that greater difficulties in emotion regulation were associated with greater self-reported anger, $b = 0.86$, $SE = 0.37$, $t(373.27) = 2.34$, $p = .020$ (see Table 5). The main effects of distraction and rumination were no longer significant. No other main effects or interactions were observed.

Discussion

This study investigated the efficacy of administering anodal tDCS over the right VLPFC on explicit anger regulation in intoxicated (vs. sober) people. As predicted, distraction reduced anger to a greater extent than reappraisal, and reappraisal reduced anger to a greater extent than rumination. This finding is consistent with the extant literature showing that distraction and reappraisal are effective strategies for anger downregulation (Bushman, 2002; Denson et al., 2012; Fabiansson et al., 2012). Although reappraisal can be more cognitively demanding and processing an anger experience via reappraisal may itself elicit some degree of anger (Denson et al., 2012; Troy et al., 2018). In contrast, rumination generally serves to intensify experiences of anger (Denson, 2013; Pop et al., 2025).

Results did not support the prediction that participants in the alcohol condition would report greater anger following the recall task, compared to sober participants. Others have similarly failed to observe an association between alcohol intoxication and increased state anger (e.g., Eckhardt, 2007;



Note. Shaded areas are 95% CIs.

Fig. 2 Self-reported anger as a function of stimulation condition, alcohol condition, emotion regulation strategy, and trait anger

Eckhardt & Crane, 2008). The proximal effects of alcohol on anger likely depend on participants' trait levels of anger, such that the effects of alcohol on aggression are stronger in individuals high in trait anger (e.g., Eckhardt, 2007; Parrott & Zeichner, 2002). It is likely the case that alcohol intoxication does not influence the likelihood of experiencing anger but impairs one's ability to regulate behavioural urges associated with expressing anger when predisposed to anger experience.

Results also failed to support the prediction that anodal stimulation would reduce anger following reappraisal and distraction in intoxicated participants and increase anger following rumination in sober participants. Anodal stimulation did not influence anger regulation. There may be many reasons for this finding. First, we observed only low levels of anger in our sample (means around the scale midpoint). These scores leave little room to reduce anger within the parameters of the experiment. Second, although we had a sound justification for targeting the right VLPFC, it is possible that other prefrontal areas are better suited as stimulation targets to influence anger regulation. For instance, research suggests that during reappraisal the connection between the VLPFC and amygdala is mediated by the ventromedial prefrontal cortex (VMPFC; Johnstone et al., 2007). Studies targeting the VMPFC may be more effective in modulating intoxicated anger regulation. Third, the brain is a highly interconnected organ. Stimulation of the VLPFC alone may have been insufficient to enhance broader network function. Indeed, the VLPFC is only one region within a broader network implicated in emotion regulation.

Table 4 Model including trait angry rumination

	Estimate	SE	df	t	Pr(>|t|)
(Intercept)	1.35	0.52	388.10	2.61	.009**
Alcohol	0.36	0.75	376.31	0.48	.629
Stimulation	0.38	0.73	381.54	0.52	.601
Distraction	-0.51	0.51	375.70	-1.00	.317
Rumination	0.49	0.51	375.70	0.96	.338
Trait Angry Rumination	0.22	0.13	383.04	1.70	.090
Alcohol × Stimulation	-1.10	1.05	373.15	-1.05	.293
Alcohol × Distraction	-0.05	0.74	373.46	-0.06	.950
Alcohol × Rumination	-1.62	0.74	373.46	-2.20	.028*
Stimulation × Distraction	-0.05	0.73	377.31	-0.07	.942
Stimulation × Rumination	0.20	0.71	374.58	0.29	.774
Alcohol × Trait Angry Rumination	-0.10	0.19	373.57	-0.53	.600
Stimulation × Trait Angry Rumination	-0.12	0.19	376.56	-0.64	.523
Distraction × Trait Angry Rumination	-0.02	0.13	374.74	-0.19	.848
Rumination × Trait Angry Rumination	0.11	0.13	374.74	0.84	.402
Alcohol × Stimulation × Distraction	0.74	1.04	374.27	0.71	.477
Alcohol × Stimulation × Rumination	1.32	1.03	372.92	1.28	.203
Alcohol × Stimulation × Trait Angry Rumination	0.51	0.29	370.33	1.77	.078
Alcohol × Distraction × Trait Angry Rumination	-0.01	0.19	372.94	-0.04	.969
Alcohol × Rumination × Trait Angry Rumination	0.42	0.19	372.94	2.21	.028*
Stimulation × Distraction × Trait Angry Rumination	0.03	0.19	378.32	0.17	.866
Stimulation × Rumination × Trait Angry Rumination	-0.06	0.19	374.03	-0.34	.732
Alcohol × Stimulation × Distraction × Trait Angry Rumination	-0.37	0.28	374.52	-1.30	.196
Alcohol × Stimulation × Rumination × Trait Angry Rumination	-0.48	0.28	372.55	-1.71	.089

Note. * = $p < .05$; ** = $p < .01$; *** = $p < .001$. Reference groups were no alcohol, sham stimulation, and reappraisal

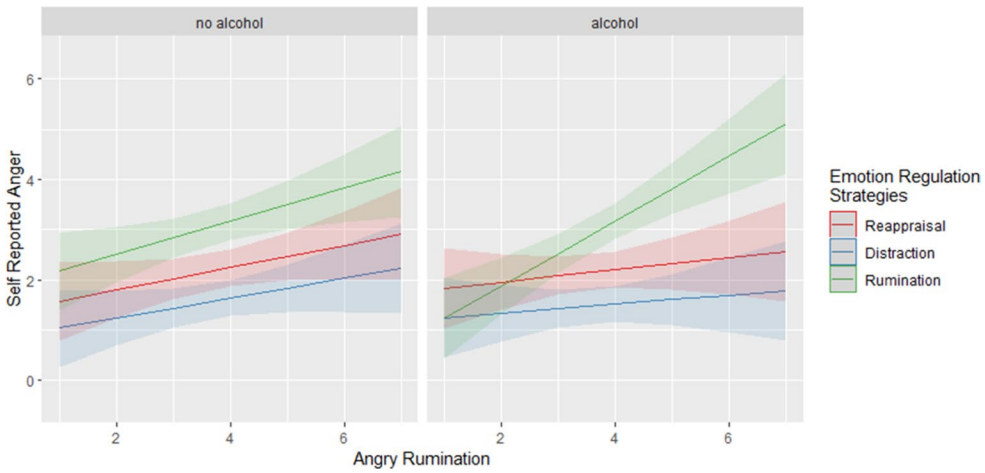


Fig. 3 Self-reported anger as a function of alcohol condition, emotion regulation strategy, and trait angry rumination

Note. Shaded areas are 95% CIs.

Successful anger regulation requires coordinated activity across multiple brain regions (Etkin et al., 2015). For instance, one fMRI study asked participants to reappraise angry faces (Morawetz et al., 2016). They found that not only was greater VLPFC activity associated with greater use of reappraisal, but that also connectivity between the VLPFC and amygdala determined greater emotion regulation success. Thus, connectivity between the VLPFC and limbic regions likely plays a substantial role in the efficacy of anger regulation. Future research combining tDCS with fMRI can provide these details.

We note that there were some significant exploratory results, which are deserving of a caveat. Because these results were exploratory in nature, we hope these findings will be interpreted with caution. Because there were a number of higher order interactions tested, these findings should be useful in generating hypotheses for future research rather than viewing these findings as set in stone. Future

Table 5 Model including trait difficulties in emotion regulation

	Estimate	SE	df	t	Pr(>|t|)
(Intercept)	0.11	0.90	374.86	0.12	.904
Alcohol	-0.06	1.20	373.08	-0.05	.961
Stimulation	0.52	1.30	372.97	0.40	.690
Distraction	0.61	0.90	372.35	0.68	.495
Rumination	0.55	0.90	372.35	0.61	.542
DERS	0.86	0.37	373.27	2.34	.020*
Alcohol × Stimulation	0.23	1.71	372.03	0.13	.895
Alcohol × Distraction	-0.57	1.19	372.00	-0.47	.636
Alcohol × Rumination	-1.18	1.19	372.00	-0.99	.323
Stimulation × Distraction	0.76	1.30	373.57	0.59	.558
Stimulation × Rumination	1.53	1.29	372.77	1.18	.239
Alcohol × DERS	0.03	0.49	372.16	0.07	.946
Stimulation × DERS	-0.22	0.54	371.93	-0.42	.678
Distraction × DERS	-0.50	0.36	372.03	-1.38	.168
Rumination × DERS	0.14	0.36	372.03	0.39	.698
Alcohol × Stimulation × Distraction	-0.31	1.71	372.72	-0.18	.857
Alcohol × Stimulation × Rumination	-0.66	1.71	372.25	-0.39	.700
Alcohol × Stimulation × DERS	0.12	0.72	371.43	0.17	.866
Alcohol × Distraction × DERS	0.20	0.49	371.82	0.41	.683
Alcohol × Rumination × DERS	0.45	0.49	371.82	0.93	.354
Stimulation × Distraction × DERS	-0.33	0.54	374.01	-0.61	.542
Stimulation × Rumination × DERS	-0.68	0.54	372.34	-1.26	.210
Alcohol × Stimulation × Distraction × DERS	-0.04	0.72	372.95	-0.05	.958
Alcohol × Stimulation × Rumination × DERS	0.13	0.71	372.00	0.18	.860

Note. * = $p < .05$; ** = $p < .01$; *** = $p < .001$. Reference groups were no alcohol, sham stimulation, and reappraisal

confirmatory research may be derived from these findings. In particular, exploratory analyses indicated anger following rumination was significantly greater for intoxicated (vs. sober) participants high (vs. low) in trait anger who received sham stimulation. However, this difference was ameliorated for intoxicated (vs. sober) participants high (vs. low) in trait anger who received active stimulation. We recognise the need for replication of this exploratory result. However, we cautiously interpret this finding as promising preliminary evidence that anodal tDCS over the right VLPFC may be an effective tool to reduce anger associated with rumination in intoxicated people predisposed to anger. However, it is possible that this reduction in anger may not reflect increased anger regulation. Rather, stimulation of the VLPFC may have disrupted rumination in people high in trait anger. Thus, the potential clinical utility of these findings should be interpreted cautiously.

Exploratory analyses also indicated that anger following rumination was significantly greater for intoxicated (vs. sober) participants who were high in trait angry rumination. Alcohol Myopia Theory (Parrott & Eckhardt, 2018; Steele & Josephs, 1990) proposes that alcohol intoxication restricts information processing, narrowing attentional scope and restricting the breadth of cue perception. Similarly, anger reduces cognitive scope as well (Gable et al., 2015). Consequently, this finding may reflect decreased attentional scope in intoxicated participants, diminishing their ability to disengage from the rumination process and intensifying their anger experience. This finding is consistent with research showing that alcohol consumption is associated with angry rumination (e.g. Ciesla et al., 2011), and that state and trait angry rumination increase intoxicated aggression (Borders and Giancola 2011; Denson et al. 2011a, b).

The effects of type of emotion regulation strategy on subsequent anger were no longer significant when accounting for emotion dysregulation. This exploratory finding may indicate that changes in self-reported anger for those with dispositional difficulties in emotion regulation may have been caused by an inability to engage with the emotion regulation strategies at all. Individuals high in emotion dysregulation are more likely to endorse engaging in suboptimal emotion regulation strategies such as rumination, and less likely to endorse engaging in cognitive reappraisal (Daros & Ruocco, 2021). However, in the current study, participants high in trait emotion dysregulation may simply have been less able to engage in instructed emotion regulation, regardless of strategy.

We acknowledge the limitations of the current experimental design. Here, participants were exposed to a single session of tDCS which may have been insufficient to cause the predicted effects. Participants sampled in this study were largely young, Asian, and female residents of

Australia completing higher education, which may limit the generalisability of the findings. It is also possible that individual differences in strategy preference, alcohol expectancies, or alcohol tolerance may have contributed to variability in our results; however, random assignment should have ameliorated any such effects. Our choice to use a no-alcohol condition rather than a placebo condition was based on ecological validity concerns. However, we note that failure to include a placebo does not allow us to control for alcohol expectancies, which are shared cultural beliefs that people expect alcohol to make them aggressive. Expectancies may be especially relevant to emotion regulation. For instance, some research suggests that for people who have trouble reappraising, placebos might increase hostility (Stappenbeck & Fromme, 2014). Future research may wish to include a placebo control to account for such effects.

Another limitation is that we relied on self-reported anger as our primary dependent variable. Future research could examine behavioral manifestations of anger such as the Taylor Aggression Paradigm (Chester & Lasko, 2019) and psychophysiological indicators of anger such as increased heart rate and lower heart rate variability. Heart rate variability may be particularly interesting as greater heart rate variability is related to adaptive forms of emotion regulation such as cognitive reappraisal (Applehans & Luecken, 2006; Denson et al. 2011a, b).

A further limitation is that the average levels of anger were fairly low, especially in the distraction and reappraisal conditions. These low levels of anger may have precluded finding significant results. Within ethical constraints, future research could attempt to elicit greater feelings of anger in participants.

Our findings also have relevance for informing future tDCS protocols. Although we focused on the VLPFC due to its role in emotion regulation, other cortical regions have been implicated in anger regulation and aggression as well. For instance, the VMPFC, DLPFC, and OFC have been extensively studied in this context, in particular with functional connectivity with the amygdala (Davidson et al., 2000; Fulwiler et al., 2012; Gilam et al., 2018; Kelley et al., 2013). Experiments that programmatically manipulate activity in these regions would help determine the most effective regions for enhancing anger regulation. Similarly, strengthening the tDCS manipulation with multiple sessions might prove fruitful. For example, Choy et al. (2023) applied tDCS to the VMPFC for three days. They didn't investigate anger, but the three days of stimulation reduced aggression in men. Thus, these findings suggest that multiple sessions may boost the effect of tDCS on anger regulation.

Another implication from this study is that future research may attempt to use stronger anger inductions. Although autobiographical memory recall is a pretty standard method for inducing anger, other work using situational manipulations such as being insulted may elicit greater anger (Siedlecka & Denson, 2019). Eliciting greater anger may then provide participants with more content to regulate, which may produce greater differentiation among the tDCS and emotion regulation conditions. Finally, within the context of anger and the prefrontal-limbic interactions in particular, the field is in need of simultaneous tDCS-neuroimaging studies (e.g., Gilam et al., 2018). Doing so could help identify functional PFC-limbic connectivity induced by tDCS. Uncovering these networks would be a substantial advance toward understanding the brain mechanisms responsible for anger regulation.

Conclusions

This study was the first to test the efficacy of tDCS in modulating intoxicated anger regulation. Although our primary predictions were not supported, exploratory results indicate that anodal tDCS over the rVLPFC may be effective in reducing intoxicated anger following rumination in individuals high in trait anger. Similarly, alcohol should best be avoided for those prone to angry rumination about autobiographical anger experiences. More research is required to replicate these effects and explore the optimal parameters with which to refine the experimental protocol. However, this study represents an important first step in applying tDCS to influence intoxicated anger regulation.

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Author Contributions TD, GG, and PR acquired the funding and designed the study. ES, KF, KT, and JB collected the data. ES analysed the data and drafted the manuscript. All authors contributed to editing the manuscript and approved the final submission.

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Data availability Data and R code are available on the OSF (https://osf.io/mf7zw).

Declarations

Ethics Approval This study was approved by the Human Research Ethics Committee at The University of New South Wales (HC200922) in accordance with the National Statement on Ethical Conduct in Human Research.

Consent to participate All participants provided informed consent prior to participating in this study.

Consent to publish All participants were informed that the study results would be published and provided informed consent with this understanding.

Conflict of interest The authors declare no competing interests.

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