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The impact of self-control training on neural responses following anger provocation

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ABSTRACT

Self-control training (SCT) is one way to enhance self-controlled behavior. We conducted a novel and exploratory functional magnetic resonance imaging experiment to examine how SCT affects neural responses in a situation that elicits a self-control response: anger provocation. Forty-five healthy young men and women completed two-weeks of SCT or a behavioral monitoring task and were then insulted during scanning. We found significant changes in functional activation and connectivity using a lenient error threshold, which were not observed using a stricter threshold. Activation in the posterior insula was greater for the control compared to the SCT group at post-provocation, trait aggression correlated with neural responses to SCT, and SCT was associated with specific amygdala-cortical connections. Neural changes occurred even though SCT did not affect participants' performance on an inhibition task, reports of trying to control their anger, or their experience of anger. This dissociation prevented clear interpretation about whether the neural changes were indicative of specific anger or anger control processes. Although replication with high-powered studies is needed, we provide evidence that SCT affects neural responses in the context of anger provocation.

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Self-control training; anger; posterior insula; dorsal anterior cingulate; fMRI

Self-control is the ability to consciously or effortfully regulate thoughts, feelings, and behaviors in order to attain long-term goals (e.g., Baumeister et al., 2007; De Ridder et al., 2017). Efficacious self-control correlates with a host of positive outcomes including improved academic performance, quality interpersonal interactions, health, and wellbeing (e.g., Tangney et al., 2004). Given the widespread benefits of self-control, strategies that improve self-control may have tangible benefits for individuals and communities. Self-control training (SCT) is one such method and involves the repeated practice of self-control over time (Baumeister et al., 2007; Berkman, 2016). Research has provided much information about the behavioral and self-reported effects of SCT, but less is known about the concomitant neural effects. Knowledge about neural correlates may help to explain the effects of SCT demonstrated in previous research.

Self-control training (SCT)

Effective SCT exercises include using one's non-dominant hand for common tasks, practicing better posture, and avoiding unhealthy foods (for a review, see Berkman, 2016). These exercises are acts of self-control because they involve monitoring ongoing behavior, detecting a habitual response, and overriding the habitual response with a preferred response (inhibition). Many SCT experiments have shown that practicing self-control in one domain for a minimum of two-weeks increases self-control in unrelated domains (e.g., Allom & Mullan, 2015; Bertrams & Schmeichel, 2014). This effect is called far transfer (Shipstead et al., 2012). Two independent meta-analyses estimated that the overall far transfer effect of SCT on a range of self-controlled outcomes was small-to-medium (k = 29, g = +0.36, CI_{95} [0.25, 0.48] in Beames et al., 2017; k = 33, g = +0.30, CI_{95} [0.17, 0.42] in Friese et al., 2017).

SCT experiments to date have primarily examined self-reported or behavioral outcomes. Whether, and how, SCT affects neural responses in situations that require self-control has received less empirical attention. To the best of our knowledge, only one functional magnetic resonance imaging (fMRI) experiment has investigated the neural correlates of SCT in the context of far transfer. Sixty healthy adults completed 10 sessions of Stop Signal Task training (SST) or a control task (sham SST) over three-weeks (Beauchamp et al., 2016). The extent of far transfer was quantified as changes in neural activation during an emotion regulation task, between baseline and post-training. Reduced activation in

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Supplemental data for this article can be accessed here.

the inferior frontal gyrus (IFG) was found for the training group, but not the control group. The IFG is involved in emotion regulation and inhibition (Aron et al., 2014; Buhle et al., 2014), and decreased activation might have reflected increased automaticity in inhibitory processing. These neural changes occurred in the absence of changes in self-reported emotional distress. In sum, SCT might affect activation in the IFG, but it remains to be seen whether these effects generalize to other self-controlled contexts.

To address this gap in the literature, we conducted a novel experiment to examine SCT-induced neural changes in the context of anger provocation. Anger provocations often involve receiving negative feedback or an unjustified insult. Such provocations are relevant to self-control because they produce a negative affective state that people find difficult to regulate and elicit an approach-motivated desire to retaliate (Carver & Harmon-Jones, 2009; Denson et al., 2015). Self-control is important in regulating anger in order to avoid potential negative consequences, such as reactive aggression (Denson et al., 2012; Wilkowski & Robinson, 2010). The potential contributions of this work are two-fold: Results could facilitate knowledge about how SCT affects behavioral and self-reported outcomes, and clarify the neural regions involved in anger and anger control.

SCT and anger provocation

Two published behavioral experiments have demonstrated that SCT reduces provoked anger and aggression. In both experiments, healthy participants assigned to the SCT group used their non-dominant hand for two-weeks for a variety of common tasks (Denson et al., 2011; Finkel et al., 2009). Relative to participants in the control group, Finkel et al. (2009) found that participants in the SCT group rated themselves as less likely to be physically aggressive toward their intimate partner upon provocation to hypothetical scenarios. Denson et al. (2011) extended these findings by showing that compared to the control group, SCT reduced subjective feelings of anger following provocation. Trait aggression and state anger positively correlated with aggressive behavior in the control group but not in the SCT group. These findings suggest that anger provocation elicits a self-control response and is therefore is a feasible method for investigating the effects of SCT on the brain.

Neural correlates of provoked anger and anger control

Neuroimaging research suggests that the correlates of self-control, or anger-control, are also relevant to anger. A review identified four neural circuits that are likely involved in the subjective experience of anger: (1) threat detection, arousal, and negative affect; (2) saliency and interoception; (3) emotion regulation; and (4) mentalizing (Gilam & Hendler, 2015; also see Alia-Klein et al., 2020 for similar circuits). The threat detection network involves the thalamic, limbic, and brainstem regions (Siever, 2008). The insula and dorsal anterior cingulate cortex (dACC) form a joint saliency detection network in which the former mediates emotional experience and the latter recruits top-down control systems and subsequent behavior modification (Craig, 2011; Gasquoine, 2013). The emotion regulation network involves the ventromedial prefrontal cortex (vmPFC)/orbitofrontal cortex (OFC) and lateral PFC. These regions are important for cognitive control and regulation of negative emotions (Buhle et al., 2014; Etkin et al., 2015). Finally, the mentalizing system, which involves the dorsomedial prefrontal cortex (dmPFC), posterior cingulate, temporal poles, and temporal parietal junction (e.g., Denny et al., 2012; Schurz et al., 2014), has been activated in anger induction studies. In the context of provoked anger, these circuits are likely involved in developing and monitoring an emotional experience, signaling a need for control, and anger control (Gilam & Hendler, 2015).

fMRI studies support the neural substrates of provoked anger and anger control specified in Gilam and Hendler's (2015) model. Recent fMRI studies have used in vivo anger social interactions such as the anger-infused Ultimatum Game (aiUG) to provoke participants. In this paradigm, a confederate offers the participant an unfair amount of money embedded with insulting provocations (for a description of the task, see Gilam et al., 2019). One study found that increased vmPFC and decreased brainstem activity correlated with decreased self-reported anger and reactive aggression (Gilam et al., 2015). A follow-up study used brain stimulation to enhance vmPFC activity, which decreased anger and aggression in the aiUG (Gilam et al., 2018). Moreover, enhanced vmPFC activity was coupled with decreased ACC and insula activity. Self-reported anger was associated with increased activity in the dmPFC, temporo-parietal junction, thalamus, and bilateral insula.

Other fMRI studies have provoked participants by insulting their ability to follow simple instructions on a difficult anagram task. Provocation increased activation in prefrontal areas following provocation, namely the dlPFC, dmPFC, and cingulate cortex, as well as subcortical areas including the insula, thalamus, and hippocampus (Denson et al., 2009). dACC activation positively correlated with self-reported anger and a measure of trait aggression (Denson et al., 2009). Two additional experiments similarly showed that people who are hormonally or genetically at risk for aggression had

increased activation in brain regions involved in anger and anger control (Denson et al., 2014, 2013). One of these studies found increased functional connectivity between the amygdala and a top-down prefrontal cortical control network, including the dlPFC, left dACC and left OFC, during induced anger control (Denson et al., 2013).

Summary of neural correlates

The neural regions involved in anger and anger control are overlapping. On the one hand, evidence suggests that the vmPFC/OFC, lateral PFC, and dACC are linked to anger control following provocation. Increased activation in the vmPFC has been associated with decreased anger; enhanced vmPFC activity has been coupled with decreased dACC and insula activity (saliency detection); and enhanced coupling between this prefrontal control network and the amygdala (anger experience) has been observed during anger control. On the other hand, evidence suggests that similar prefrontal regions are also implicated in the experience of provoked anger. Increased activation in the dmPFC, dACC, thalamus, insula, and hippocampus has been associated with increased anger, and anger provocation increases activation in the dlPFC and vmPFC (Denson et al., 2009; also see Fanning et al., 2017). One explanation for increases in prefrontal and subcortical regions following provocation is that anger expression (and reactive aggression) occurs when regulatory regions are compromised or overwhelmed by the salience of the anger cue (Blair, 2012). Individuals with reduced self-control or increased impulsivity might be more likely to show these neural effects following provocation, because they need to exert greater effort to down-regulate emotional reactivity (e.g., Blair, 2012; Denson, 2014). Increasing self-control though training will likely influence how these regions activate and connect following a provocation, although the nature of this influence is somewhat unclear.

The present research

We conducted an exploratory fMRI experiment to examine differences in neural responses among healthy young men and women who practiced self-control for two-weeks (or not) and were subsequently provoked. Replicating prior behavioral research, we expected that SCT would decrease subjective feelings of anger following provocation relative to the control group (Denson et al., 2011). Given the exploratory nature of the study and overlapping neural regions, we did not make specific a priori predictions about how SCT would affect neural activation and connectivity, or how this would relate to trait aggression, anger, and anger control. Our view was that there were too many competing predictions available to meaningfully select one over another.

Method

Participants and design

An Australian community sample between the ages of 18–30 was recruited from the University of New South Wales' (UNSW) School of Psychology paid research participation recruitment platform. Participants received AUD$45 for attending each face-to-face session, and a bonus AUD$10 for completing measures related to SCT adherence. Exclusion criteria were left-handedness, MRI contraindications, and lack of English language proficiency. Due to the collection of saliva samples to test for hormone concentrations, participants were asked to abstain from eating, drinking, smoking, and chewing gum 30 min prior to the experiment. Hormone analyses are not reported in this article.

The sample size was set at 60 due to funding. Using the repeated measures within-between interaction function in G*Power 3.0.10, we had 63% power to detect an effect size of $f = 0.15$ (or $d = 0.30$) with $\alpha_{\text{two-tailed}} = .05$. Participants were randomly assigned to either the SCT or the control group. Data from 15 participants were excluded from analysis due to: attrition ($n = 7$); not believing the provocation ($n = 4$); abnormal structural scans determined by a radiologist ($n = 2$); excessive movement (i.e., ± 3.00 mm) that could not be motion corrected ($n = 1$), and technical errors during scanning ($n = 1$). The final sample consisted of 45 participants ($M_{age} = 20.60$ years, $SD = 2.50$, 18–31; 20 men) who were predominantly Caucasian (31.1%) or Asian (62.2%). Twenty-four participants were in the SCT group; 21 were in the control group. One participant aged 31 years was accidently admitted into the experiment but was included in analyses. All participants reported that English was their native or best language. None of the demographic variables varied by group (see Tables S1 and S2 in the Supplementary Materials). This experiment was approved by the UNSW ethics committee and all participants provided written informed consent.

Materials and procedure

Session 1

This experiment was conducted over two sessions that were separated by a two-week interim. During the first session in the laboratory, participants were screened for

right-handedness and MRI safety. Eligible participants were informed by the experimenter that the experiment was about cognition and brain activity. To avoid demand and awareness of the manipulation, self-control was not mentioned. After obtaining demographics, all participants completed a battery of measures (see Supplementary Materials for a full list). Only the Aggression Questionnaire (Buss & Perry, 1992) was analyzed as part of the current experiment. The Aggression Questionnaire consists of four subscales: anger, hostility, verbal aggression, and physical aggression. Because the specific facets of trait aggressiveness that might affect the neural correlates of SCT are unknown, we used the average of the four subscales in a covariate analysis (α = .84; 1 = *extremely uncharacteristic of me* to 5 = *extremely characteristic of me*). There were no group differences in trait aggression, $F(1, 43) = 0.19$, $p = .66$, $d = 0.13$ (see Table S2 in the Supplementary Materials). Participants then provided a saliva sample.

Two-week interim

During the two-week interim, participants in the SCT group engaged in a verbal regulation task that increases self-control (e.g., Finkel et al., 2009). The effects of SCT manipulations like the verbal regulation task are due to enhanced self-control rather than awareness of self-control, self-fulfilling prophecies, or self-efficacy (Muraven, 2010). In the verbal regulation task, participants were required to monitor and regulate their speech as often as possible between 8 am and 6 pm each day. They were given specific examples to guide the manner of their speech regulation, including speaking in full and complete sentences, and refraining from using slang, abbreviations, and colloquialisms. This task requires self-control because participants must monitor their verbal behavior, detect a discrepancy between using habitual language and the goal of adhering to the task requirements, and then override the use of habitual language.

Participants were required to complete online, time-stamped diaries every second day (seven total) to evaluate task adherence and difficulty throughout the two-weeks. For the diaries, participants rated the frequency with which they regulated their speech (1 = *not at all*, 10 = *consistently*). Participants were instructed to be as accurate as possible and were told that no penalty would apply for reports of noncompliance. Participants in the control group were asked to monitor, but not to regulate, the time that they spent watching TV each day. To keep task requirements consistent across the two groups, control participants also completed online diaries about their TV watching habits. Participants in both groups also received text messages every second day to remind them to complete the speech tasks.

Session 2

The second session took place at the MRI facility at Neuroscience Research Australia. There was no difference between the average number of days between session one and session two for the SCT ($M = 14.04$, $SD = 0.20$) and control groups ($M = 15.00$, $SD = 4.36$), $F(1, 43) = 1.16$, $p = .29$, $d = 0.31$. All participants first reported their baseline mood using a modified version of the Positive and Negative Affect Scale (PANAS; Harmon-Jones et al., 2009). This measure contains 36 items (1 = *very slightly or not at all*, 5 = *extremely*) that assess state general positive affect, general negative affect, and angry affect. Only the angry affect scale was analyzed as part of the current experiment (α = .55).[1]

A 3D structural scan was taken before participants completed two tasks in the scanner. The first task, not described here, involved responding to emotional faces while a functional scan was taken. A 2-min pre-provocation (or resting state) functional scan was then taken to record baseline activity. Following prior research, the focal task involved completing a series of anagrams, which was used to provoke participants (Denson et al., 2014, 2009, 2013). On each trial, participants were instructed to verbalize their answer aloud if they solved the anagram, or to say "no answer" if they were unable to solve the anagram. As part of the provocation, an audio recording of one of the coauthors (TD) interrupted participants to instruct them to speak louder. During the third interruption of the recording (i.e., the provocation), TD insulted participants by saying "Look, this is the third time I have had to say this! Can't you follow directions?" This and similar provocation procedures that undermine participants' general intelligence and competence successfully increase anger, aggression, and blood pressure (Denson et al., 2009; Memedovic et al., 2010). A second 2-min functional scan was taken after the provocation. Participants stared at a blank screen during the structural scan and a green circle during the pre- and post-provocation functional scans.

Next, outside of the scanner, participants completed another modified-PANAS (angry affect, α = .93) and five items assessing anger control (e.g., "I tried hard to control my emotions during the scanning", 1 = *not at all*, 5 = *extremely*, α = .72). Mean scores were computed for all self-report measures. Then participants completed a modified version of

[1]Four of the eight items were removed from the reliability analyses because all participants endorsed the lowest score on the scale (i.e., angry, furious, hostile, and mad).

the cued Go No-Go Task (Fillmore, 2003). There were no effects of group on the number of inhibition errors made by participants following the provocation, incidence rate ratio = 1.06, [CI_{95} = 0.61, 1.83], p = .85, d = 0.03 (see the Supplementary Materials for a description of the task and generalized linear model output [Table S3]). Since we did not find evidence for group effects on Go No-Go performance, we do not consider if further. Participants were then probed for suspicion, thanked, debriefed, and compensated.

Image acquisition

The anagram task was presented to participants in a Philips Achieva X-Series 3-Tesla whole-body scanner on a high-resolution monitor. Participants viewed the stimuli through mirrors. The scanner consisted of a 32-channel head coil and parallel imaging system. Padded foam head constraints were used to control head movements. We obtained a T1 anatomical 3D structural dataset (180 slices, FOV = 180 mm × 256 mm × 256 mm, voxel size = 1 × 1 × 1 mm), as well as a functional whole-brain EPI pulse sequence with sagittal slices and 2.5 SENSE acceleration (49 ascending slices without gaps, slice thickness = 3 mm, voxel size = 2.26 × 2.26 × 3.00 mm, FOV = 253 × 253 mm, volumes = 48 per scan, TE = 51 ms, TR = 2500 ms, 90° flip angle). The first four volumes were discarded.

Preprocessing

First, BET from the FSL package (Smith et al., 2004) was used to remove the components of the EPI images that captured non-brain tissues. This step was necessary because the sagittal EPI slices contained substantial non-brain tissue that had the potential to interfere with motion correction procedures. Second, the data were imported to BrainVoyager QX, which was used to conduct the remaining preprocessing. Third, the functional images were slice scan time corrected using cubic-spine interpolation, 3D motion corrected, and spatially smoothed with a 5.00 mm FWHM Gaussian filter. Fourth, the functional images were manually linearly coregistered with the structural images. Fifth, the coregistered functional images were normalized via Talairach transformation (Talairach & Tournoux, 1988). Sixth, all functional scans were adjusted for the hemodynamic response function and head-movement realignment parameters for each participant were added as additional nuisance predictors.

Statistical analyses

Excluding outliers (i.e., ± 3 *SD*s from the mean) from the self-report data did not change the pattern of results, and so all data points are included in the relevant analyses.[2] None of the neuroimaging results were significant at the false discovery rate, q(FDR) < .05. For all analyses described below, correction of brain activation maps for multiple comparisons was performed by setting a voxel-level threshold at p < .005 (uncorrected) with a minimum cluster-size of 10 contiguous functional voxels (where each voxel corresponds to an anatomical volume of 3 × 2.26 × 2.26). This approach produces a balance between Type I and II error rates (Lieberman & Cunningham, 2009).

Neural activation

Inferential statistics were conducted with BrainVoyager QX and SPSS v25. We used a paired sample t-test and univariate Analysis of Variances (ANOVAs) to test the efficacy of the SCT manipulation. We also used univariate ANOVAs to test the efficacy of the provocation procedure on self-reported anger and anger control. For BOLD responses, we computed a single whole-brain random effects (RFX) General Linear Model (GLM). We corrected for temporal autocorrelations using a second-order autoregressive model. Our analytic strategy involved three random effects contrasts: (1) the provocation main effect (post-provocation > pre-provocation); (2) the group main effect (control > SCT); and (3) the provocation × group interaction. Subsequent analyses on the resulting clusters of activation were performed by extracting beta values (averaged across all cluster voxels) and tested for significance with t-tests.

Correlation analyses. Based on behavioral research suggesting that SCT may be most beneficial for individuals with high trait aggression (Denson et al., 2011), we used an RFX ANCOVA to correlate this individual difference measure with whole-brain BOLD responses for the post-provocation > pre-provocation contrast. We also conducted post hoc correlation analyses between neural activation, anger, and anger control.

[2]One outlier from the anger control measure, three from the angry affect subscale of the PANAS measured at pre-provocation, and one from the same subscale measured at post-provocation.

None of the correlations were significant, *rs* < |.16|, *ps* > .49 (see the Supplementary Materials for statistical approach and results [Table S4]).

Neural connectivity

We conducted whole-brain psychophysiological interaction (PPI; Cisler et al., 2014; O'Reilly et al., 2012) random effects GLM analyses using previously developed scripts in MATLAB v.9.0 to examine functional connectivity (Gilam et al., 2015). We selected the bilateral amygdala a priori as the seed ROI, centered at ± 21, y = −6, and z = −14 as determined by the Brede Toolbox (http://neuro.imm.dtu.dk/services/bredeatabase/WOROI_36.html) and Neurosynth (http://neurosynth.org/analyses/terms/amygdala/) web applications (see Figure S1 in the Supplementary Materials). Our rationale for selecting the amygdala was that it is often influenced by provocation and is involved in anger and anger regulation (e.g., Davidson et al., 2000; Denson et al., 2013; Fanning et al., 2017). For example, the amygdala and vmPFC share rich connections and correlate with emotional reactivity and regulation networks (e.g., Carré et al., 2011; Gilam & Hendler, 2015). In our PPI analyses, we focus on associations between the amygdala and other regions relevant to anger and anger control.

Results

Manipulation checks and mood

SCT tasks

Participants in the SCT group completed an average of 4.75 diaries (out of a possible 7; *SD* = 1.82). On average, participants reported engaging in the SCT tasks each day to a moderately high level: *M* = 5.65 (out of 10; *SD* = 1.73). As expected, participants reported that they experienced difficulty completing the SCT tasks (*M* = 5.70, *SD* = 1.32), yet they also reported performing the tasks with greater frequency during the second week of SCT compared to the first week of SCT, *t*(20) = 3.16, *p* = .005, *d* = 0.69. These results indicate sufficient engagement with the SCT tasks.

Angry affect and anger control

Participants reported increased anger from pre-provocation (*M* = 1.10, *SD* = 0.19) to post-provocation (*M* = 1.83, *SD* = 0.86), *F*(1, 42) = 31.67, *p* < .001, *d* = 1.19 (see Table S5 in the Supplementary Materials for additional descriptive statistics). There was no significant main effect of group, *F*(1, 42) = 0.34, *p* = .57, *d* = .17, and contrary to our hypothesis, no significant time × group interaction, *F*(1, 42) = 0.59, *p* = .45, *d* = −0.43. However, participants in the control group (*M* = 1.93, *SD* = 0.75) did not report significantly greater anger than the SCT group (*M* = 1.75, *SD* = 0.95) following provocation, *F*(1, 42) = 0.48, *p* = .49, *d* = 0.21.

Participants reported controlling their emotions during the study at a level significantly different from the scale endpoint of "not at all" (*M* = 2.55, *SD* = 0.73), *t*(43) = 23.08, *p* < .001, *d* = 3.47. This finding conceptually replicated the level of anger control reported in Denson et al. (2013). Contrary to our hypotheses, however, there was no difference between groups in the extent to which they reported trying to control their anger in response to the provocation (SCT: *M* = 2.44, *SD* = 0.72; control: *M* = 2.68 *SD* = 0.75), *F*(1, 42) = 1.11, *p* = .30, *d* = .33.

Neuroimaging results

Neural activation

Figure 1 summarizes the significant RFX GLM contrasts (also see Table S6 in the Supplementary Materials). There were main effects of group in the right middle frontal gyrus (MFG) and right fusiform gyrus, but no main effects of provocation. There were significant group × provocation interactions within the left inferior parietal lobule, left superior frontal gyrus (SFG), left medial frontal gyrus, right cuneus, right precuneus, and right posterior insula. Activation in the left SFG was lower in the control group compared to the SCT group at pre-provocation, *t*(43) = −3.54, *p* = .001, *d* = −1.05. In the control group, activation in the right cuneus was lower at pre-provocation compared to post-provocation, *t*(20) = −2.38, *p* = .03, *d* = −0.65. Activation in the right posterior insula was greater in the control group compared to the SCT group at post-provocation, *t*(43) = 2.75, *p* = .009, *d* = 0.82. None of the remaining follow-up *t*-tests were significant, *ts* < |1.9|, *ps* > .07 (see Table 1 for descriptive statistics).

Correlations with trait aggression. For each group, we correlated trait aggression scores with BOLD signal for the post-provocation > pre-provocation contrast. For participants in the SCT and control groups, trait aggression was positively correlated with relatively greater post-provocation activation in frontal, temporal, and subcortical regions (Figure 2; also see Table S7 in the Supplementary Materials). Trait aggression was also negatively correlated with activation in the left cingulate gyrus for the control group.

Neural connectivity

No significant differences were found when examining pre- and post-provocation predictors

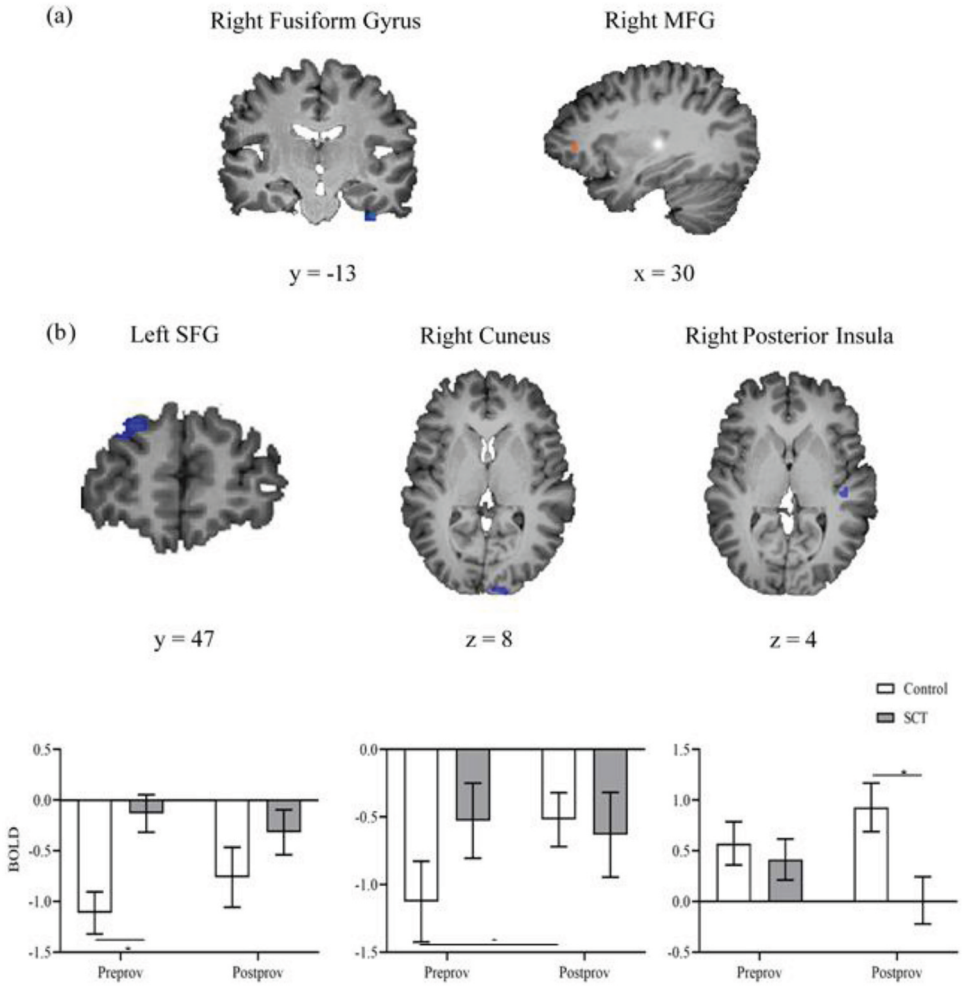

Figure 1. Significant RFX GLM contrasts. All regions arising from whole-brain random-effects analysis, presented at a threshold of $p < 0.005$ with a minimum cluster size of 154 contiguous anatomical (1 mm^3) voxels. (a) Main effects of group (Control > SCT). Brain areas colored in blue/red represent clusters in ROIs showing decreased/increased activation in the control compared to the SCT group; (b) Group × provocation interactions that yielded significant simple effect contrasts. Mean beta values extracted from interaction, demonstrating the effect seen in the corresponding brain activity map. Error bars indicate SEM. MFG = middle frontal gyrus; SFG = superior frontal gyrus; Preprov = Pre-provocation; Postprov = Post-provocation.

Table 1. Descriptive statistics for simple effect contrasts from the group × SCT interaction.

	Pre-Provocation				Post-Provocation			
	Control		SCT		Control		SCT	
Brain Region	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
Inferior Parietal Lobe	−0.29	1.33	0.04	1.11	0.46	1.13	−0.17	2.15
SFG	−1.11	0.95	−0.13	0.91	−0.76	1.36	−0.32	1.09
Medial Frontal Gyrus	−1.73	1.95	−1.68	1.40	−1.60	1.39	−1.78	1.65
Cuneus	−1.13	1.37	−0.53	1.36	−0.52	0.92	−0.63	1.54
Precuneus	−0.48	1.29	−0.59	1.07	0.07	1.22	−0.47	1.36
Posterior Insula	0.57	0.98	0.41	0.99	0.93	1.09	0.01	1.14

$n_{Control} = 21$, $n_{SCT} = 24$. SFG = superior frontal gyrus.

simultaneously. We then conducted separate PPI analyses at pre- and post-provocation to test for differences in functional connectivity between groups. At pre-provocation, there was a positive association between activity in the bilateral amygdala and right MFG for the SCT group, but a negative association for the control group. At post-provocation, there was a positive association between the bilateral amygdala and right middle temporal gyrus (MTG) for the SCT group, but a negative association for the control group (see Figure 3; also see Tables S8 and S9 in the Supplementary Materials).

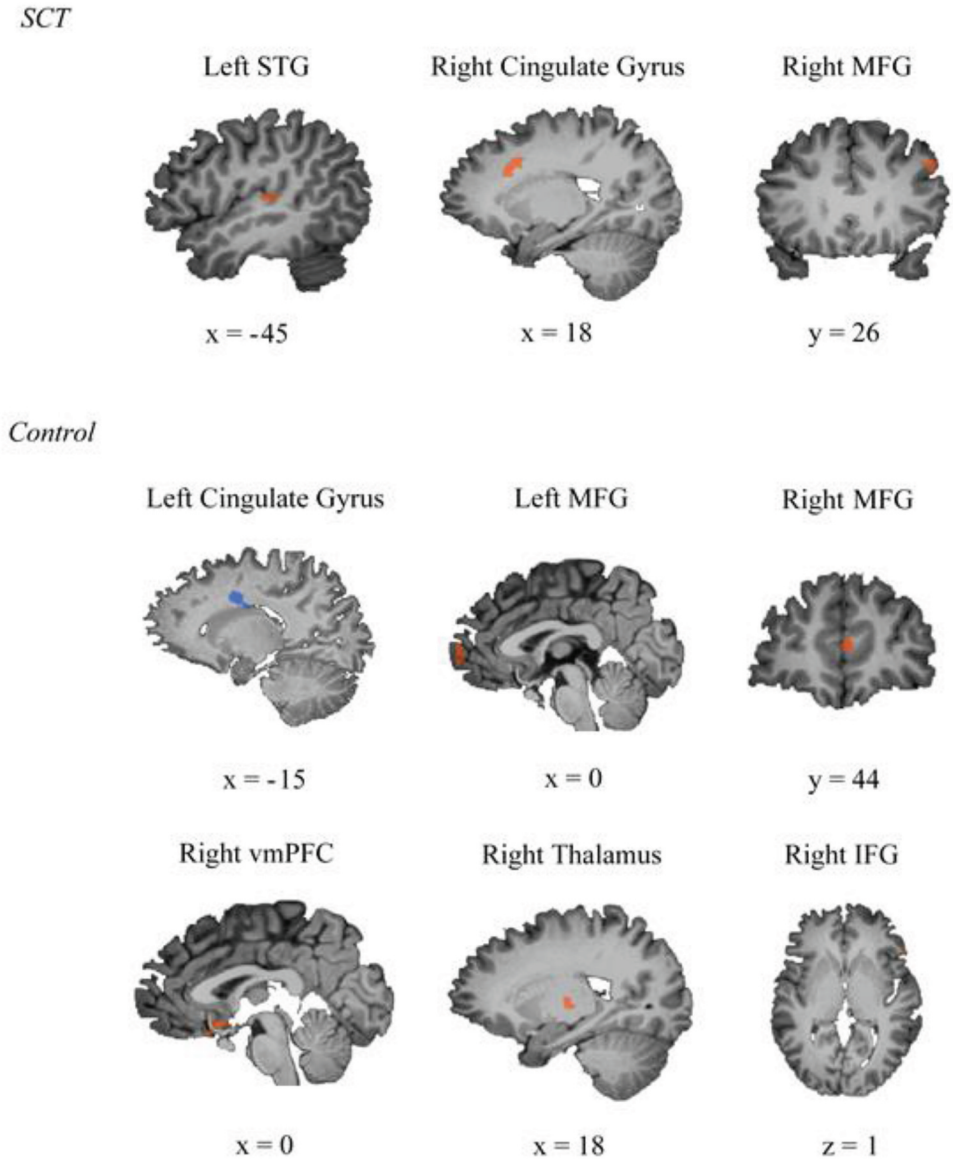

Figure 2. Correlation analyses. The top and bottom panes show correlations between trait aggression and BOLD signal for the post-provocation > pre-provocation contrast for the SCT and control groups, respectively, for select clusters. Brain areas colored in blue/red represent clusters in ROIs showing negative/positive correlations with greater post-provocation activation. All regions arising from whole-brain random-effects analysis, presented at a threshold of $p < 0.005$ with a minimum cluster size of 154 contiguous anatomical (1 mm^3) voxels. MFG = middle frontal gyrus; vmPFC = ventromedial prefrontal cortex; IFG = inferior frontal gyrus.

Discussion

This exploratory study examined the neural responses associated with SCT in the context of provoked anger. We found preliminary evidence that SCT affects neural regions related to anger, anger control, and self-control more broadly (albeit with a lenient statistical threshold). The changes in neural activation and connectivity associated with SCT emerged even though SCT did not significantly affect participants' performance on a self-control task, or their reports of trying to control their anger or its experience. This dissociation, although noteworthy, means that it is difficult to interpret the neural changes observed in this study. We offer some interpretations by drawing from previous research but acknowledge that we cannot conclude whether these changes were specifically associated with anger or anger control.

Neural activation

Neural correlates of SCT

Resting state activation in the SFG was lower in the control group compared to the SCT group. The activated cluster was located in the posterior dorsolateral region of the SFG (Li et al., 2013). This result is consistent with prior research showing that the dlPFC is involved in cognitive control, working memory, and attention (e.g.,

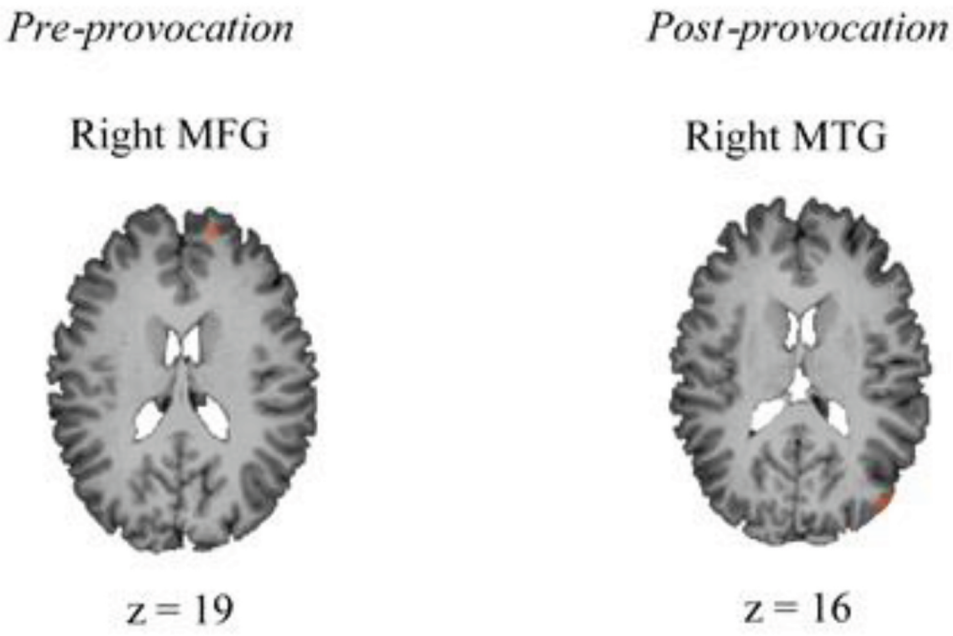

Figure 3. Functional connectivity analyses. The bi-lateral amygdala was a significant seed ROI. The left and right panes show the control > SCT contrast at the level of pre-provocation and post-provocation, respectively, for select clusters. All regions arising from whole-brain random-effects analysis, presented at a threshold of $p < 0.005$ with a minimum cluster size of 154 contiguous anatomical (1 mm^3) voxels. MFG = middle frontal gyrus; SFG = superior frontal gyrus.

Brosnan & Wiegand, 2017; Duncan & Owen, 2000; Li et al., 2013). SCT might have prepared participants for a later self-control conflict, such as a provocation, by increasing the efficiency with which they could exert self-control (or decreasing the effort that they needed to do so). This interpretation is preliminary given that SCT did not affect performance on a self-control task (i.e., Go No-Go) relative to the control group, and we did not measure baseline neural activation prior to the two-week manipulation. These limitations reduce our capacity to infer whether resting state activation does (or does not) reflect processes related to self-control.

Neural correlates of SCT following provocation

Our results suggest that the posterior insula might be an important neural region for SCT. Activation in the posterior insula was greater for the control compared to the SCT group at post-provocation. Increased insula activation in the control group might reflect greater processing of arousal caused by the provocation; participants might have been more aware of provoked somatic responses. This result is consistent with activation in the saliency detection network proposed within Gilam and Hendler's (2015) model of anger. The insula activity is also consistent with a neuroimaging experiment that found increased activation in the dorsal posterior insula during more angering provocations (particularly for participants who were better able to regulate; Gilam et al., 2015). Given that posterior insula activity did not correlate with any self-reported outcomes in the current study, is not clear whether this activity is specific to anger or anger control.

Correlations with trait aggression

We found correlations between hemodynamic activity in diffuse neural regions and trait aggression. There was indirect evidence that anger prone participants in the control group had increased brain activity in dmPFC, vmPFC, thalamus, and IFG following provocation. These regions have been implicated in processing anger experience and aggressive behavior (Fanning et al., 2017; Gilam et al., 2015; Rosell & Siever, 2015) as well as self-control (Denson, 2014; Maier & Hare, 2017). Relatedly, these regions are involved in key neural circuits considered in Gilam and Hendler's (2015) model of anger: the threat detection network (e.g., thalamus), emotion regulation network (e.g., vmPFC), and mentalizing system (e.g., dmPFC). We also found inverse correlations between trait aggression and cingulate activation across the two groups. Participants higher in trait aggression in the control group showed reduced anterior cingulate activity after provocation. Similar to Denson et al.'s (2009) findings, participants high in trait aggression in the SCT group showed heightened dorsal anterior cingulate activity after provocation. Enhanced dACC activation has been linked to anger control networks through its saliency detection role, but also to increased anger experience. In the context of this previous work and lack of correlation with self-

reports in the current study, it is difficult to differentiate whether these correlations reflect increased anger and/or anger control.

Neural connectivity

Our connectivity results showed opposing amygdala-cortical associations for the SCT and control groups. Amygdala activity correlated positively with MFG activity before provocation in the SCT group, but negatively in the control group. After provocation, the same pattern (positive for SCT, negative for control) was observed for correlations between amygdala activity and the MTG. Previous research has implicated the MFG and MTG in emotion regulation and social perception (Grecucci et al., 2013). Assuming this regulatory role, these associations provide *some* support for regulation potentiation (or at least alteration) in SCT subjects. If so, these results have implications for self-controlled outcomes beyond the domain of provoked anger; SCT might produce far transfer by affecting neural networks with a broad array of functions.

Dissociation between neural and behavioral changes

The dissociation between behavioral and neural SCT effects is consistent with the findings from Beauchamp et al.'s (2016) training study, as well as from clinical research with healthy participants (e.g., Schweizer et al., 2019). One reason for dissociation is that neural activation might be a precursor to later behavioral changes, particularly in healthy individuals. Another related reason is that neural substrates are more responsive to interventions or experimental manipulations than other outcomes. For relatively healthy individuals, behavioral effects might be modulated by other study-specific and individual difference factors (e.g., personality traits or the nature task stimuli/paradigm; Schweizer et al., 2019). Given that other SCT experiments have found far transfer, it is likely that methodological characteristics of the current experiment play some role in our failure to observe effects in self-report data. For example, anger may have dissipated naturally over the course of the post-provocation scan. In support of this interpretation, participants were asked to report how angry they felt in the current moment after the post-provocation scan rather than when they were provoked or during the scan.

Limitations and future research

This experiment was the first attempt to investigate the neural mechanisms of SCT in response to provocation. Although the experiment provided initial insight into the neural substrates of SCT, it was limited in some aspects of sampling and methodological design. We did not find significant results for the analyses using a stricter threshold (i.e., the FDR). Insufficient power, due to a between-subjects design and a relatively small sample size, might have prevented detection of small effects. Our approach of using cluster thresholding might have produced false-positive results, meaning that any BOLD and connectivity differences observed between groups might be related to confounding variables or noise (Eklund et al., 2016). It is difficult to rule out this explanation until further research with greater power is conducted. Although our results were generally consistent with the extant literature, inferences based on a liberal threshold must be taken with caution.

SCT did not significantly affect self-reported anger or performance in a self-control related task. Given that the SCT manipulation was ineffective in this regard and there were no correlations of anger or anger control with functional activity or connectivity, it is difficult to infer how these results relate to neural changes. Future work could build on the foundations provided by our study but recruiting a larger sample and using measures of self-control or affect that are more sensitive to change.

Our sample was qualitatively different from clinical and criminal populations who are prone to violence and aggressive behavior, and thus results may not generalize to individuals with severe aggression, poor self-control, or excessive impulsivity. We also limited our investigation to the neural correlates of anger *after* a provocation had occurred. More recent studies have examined activation and functional connectivity *during* anger provocation using the aiUG (e.g., Gilam et al., 2015). Understanding how SCT affects neural responses in a healthy community sample, during the entire time-course of provocation, paves the way for future studies with more aggressive populations.

Conclusions

Despite the growing literature on the behavioral effects of SCT, the role of the brain remains relatively unexplored. We provide preliminary evidence to suggest that SCT might affect activation in the posterior insula and dACC, the latter more so in aggression prone individuals. We also provide evidence that SCT is associated with amygdala-cortical connections that are potentially involved in emotion regulation, which differ from those observed in the control group. Finally, we found a dissociation between neural and self-reported/behavioral outcomes. This dissociation prevents clear conclusions about how SCT produces far transfer, as well as the

neural regions associated with anger and anger control. Despite the limitations, our study represents a novel approach to exploring SCT and highlights a need for further neuroimaging research that identifies brain-behavior links in the context of provoked anger.

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Disclosure statement

No potential conflict of interest.

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Data Availability Statement

The data and materials are available upon request from the corresponding author. The experiment was not preregistered.

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