Cognitive: Executive Function

Don't look back in anger: Neural correlates of reappraisal, analytical rumination, and angry rumination during recall of an anger-inducing autobiographical memory

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ABSTRACT

Despite the enormous costs associated with unrestrained anger, little is known about the neural mechanisms underlying anger regulation. Behavioral evidence supports the effectiveness of reappraisal in reducing anger, and demonstrates that rumination typically maintains or augments anger. To further understand the effects of different anger regulation strategies, during functional magnetic resonance imaging 21 healthy male and female undergraduates recalled an anger-inducing autobiographical memory. They then engaged in three counterbalanced anger regulation strategies: reappraisal, analytical rumination, and angry rumination. Reappraisal produced the least self-reported anger followed by analytical rumination and angry rumination. Rumination was associated with increased functional connectivity of the inferior frontal gyrus with the amygdala and thalamus. Understanding how neural regions interact during anger regulation has important implications for reducing anger and violence.

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Introduction

When recalling an anger-inducing event, the extent to which one becomes angered depends on how one mentally processes the event (Mauss et al., 2007; Memedovic et al., 2010; Ray et al., 2008). This process of emotion regulation refers to how we experience and express our emotions. Failing to regulate frequent anger can lead to violence, social dysfunction, and poor mental and physical health (Anderson and Bushman, 2002; Friedman and Booth-Kewley, 1987; Houston and Vavak, 1991; John and Gross, 2004). In the present functional magnetic resonance imaging (fMRI) study, participants cognitively processed an anger-inducing autobiographical memory in three ways: namely, by engaging in reappraisal, analytical rumination, and angry rumination. Our goal was to understand the neural mechanisms involved in these three forms of anger regulation.

Of these three types of emotion regulation, the most effective in reducing anger is cognitive reappraisal (Denson et al., 2011a; Mauss et al., 2007; Memedovic et al., 2010; Ray et al., 2008). Cognitive reappraisal involves reinterpreting an emotional event in order to reduce its negative emotional impact (Gross, 1998; 2001). For instance, one may think about an anger-eliciting event from the perspective of a neutral third party. Behavioral studies have shown that reappraisal decreases anger and increases healthy patterns of cardiovascular responding (Denson et al., 2011a, in press; Mauss et al., 2007; Memedovic et al., 2010; Ray et al., 2008); however, the neural regions involved in reappraising an anger-inducing event have not been investigated. Outside of the anger context, reappraising negative affective stimuli increases activation in regions implicated in meaning processing, self-reflection, cognitive control, and reward. The most robust finding is that reappraisal activates the dorsal and/or ventral lateral prefrontal cortex (PFC) (for a review, see Ochsner and Gross, 2008). The inferior frontal gyrus (IFG) in the lateral PFC has been implicated in a variety of tasks requiring cognitive and inhibitory control including emotion regulation (Lieberman, 2007; Tabibnia et al., 2011). Other regions often activated by reappraisal include the medial PFC (mPFC), lateral and medial orbitofrontal cortex (OFC), anterior cingulate cortex (ACC), amygdala, and caudate (McRae et al., 2008, 2009; Ochsner and Gross, 2008; Ochsner et al., 2002, 2004).

In addition to reappraisal, we investigated two forms of rumination. Analytical rumination involves focusing on why an event occurred by analyzing the event’s causes, consequences, and meaning (for a review, see Watkins, 2008). When analytical rumination is conducted from a “cool”, self-distanced perspective, it reduces anger and cardiovascular reactivity relative to “hot”, emotionally evocative, self-immersed angry rumination (Ayduk and Kross, 2008; Kross et al., 2005). Similarities have been drawn between self-distanced analytical rumination and reappraisal, such that both involve an attempt to...
create meaning. Furthermore, taking a distanced perspective has been conceptualized as an aspect of reappraisal (Ayduk and Kross, 2010; Ochsner and Gross, 2008). By contrast, angry rumination involves focusing on one’s angry feelings and thoughts of revenge, and results in increased anger and aggression (Bushman, 2002; Caprara, 1986; Denson et al., 2006; Sukhodolsky et al., 2001). Angry rumination increases or maintains anger, aggression, blood pressure (Bushman, 2002; Bushman et al., 2005; Denson et al., 2011b; Pedersen et al., 2011) and increases activation in the insula, ACC, mPFC, and dorsal and ventral lateral PFC (Denson et al., 2009).

To our knowledge, only three studies have examined the neural correlates of anger regulation, and they have primarily done so indirectly (Alia-Klein et al., 2007, 2009; Denson et al., 2009), Alia-Klein et al. (2007) asked men to listen to the word no, which tends to be associated with anger, and yes, which does not tend to be associated with anger. Relative to neutral words, hearing the word yes increased activity in the lateral OFC, whereas hearing the word no decreased activity in the lateral OFC. Self-reported trait anger control was positively correlated with lateral OFC activity while listening to the word no. Additional research suggests that OFC lesions are associated with deficits in self-regulation and impulsive aggression (Blair, 2004; Grafman et al., 1996; Krisingerbach and Rolls, 2004). Consistent with this notion, Mehta and Beer (2010) found that blunted activation in the medial OFC was correlated with increased reactive aggression in an economic bargaining game. The lateral and medial OFC have also been implicated in regulating general negative affect (Banks et al., 2007; Ochsner et al., 2002, 2004; Phan et al., 2005). Ochsner et al. (2004) found that using reappraisal to decrease negative affect was associated with increased lateral PFC and OFC activation. In sum, the research to date suggests a role for top-down control processes supported by the lateral PFC and OFC in anger regulation.

The present research

In this experiment, we identified regions commonly activated during reappraisal, analytical rumination, and angry rumination. We then tested two complementary hypotheses that may account for the differential effectiveness of each strategy in reducing anger. The mean level hypothesis is that mean levels of activation in the regions differ as a function of type of anger regulation. For instance, reappraisal may initiate relatively greater activation in regions implicated in cognitive control and reward (e.g., IFG, OFC, dACC, caudate); whereas angry rumination may initiate greater activation in regions implicated in negative emotions and arousal (e.g., amygdala, insula, thalamus). To test the mean level hypothesis, we conducted a conjunction analysis to identify regions that are active during all three anger regulation strategies relative to a baseline period in which participants were asked to relax. We then examined mean differences in activity during the three strategies.

A second possibility is the functional connectivity hypothesis which is that the emotion regulation strategies differ in terms of functional connections that exist between neural regions. Past research on reappraising negative affect has implicated downregulation of subcortical regions by the PFC (Banks et al., 2007; Heatherton, 2011; Ochsner et al., 2002; Urry et al., 2006). In contrast to reappraisal, rumination may involve upregulation of subcortical limbic activation by the PFC. For instance, thinking about the anger experienced and planning revenge may be supported by positive connectivity between cortical and subcortical regions. Such a reciprocal feedback loop in which participants become increasingly “worked up” could explain how rumination (or at least angry rumination) increases arousal and anger. To test the functional connectivity hypothesis, we performed psychophysiological interaction (PPI) analyses examining activity during each emotion regulation strategy. We expected that regions associated with top-down prefrontal control would modulate activity in subcortical regions.

Method

Participants and design

Twenty-three right-handers graduated from the University of New South Wales were reimbursed AUD$40 for voluntary completion of the study. Participants were recruited using listings on the university careers website. One participant was excluded for excessive movement during the scan. Another participant had an abnormal left frontal lobe and was referred to a medical professional. This left a total of 21 participants (11 women; Mage = 21, SDage = 3.19; 57% Asian, 38% Caucasian, and 5% other). A counterbalanced within-participants block design was used. No gender differences were observed for self-reported data or BOLD responses in any of the regions of interest.

Materials and procedure

Initial questionnaire session

In the laboratory, participants completed handedness and safety-screening questionnaires. Next, participants recalled an anger-inducing memory of an event that occurred within the past 12 months. Using a mood adjective checklist (MACL; Nowlis, 1965; e.g., angry, sad, disgusted, happy), participants rated how they felt when they originally experienced the event and after they recalled the event. Participants also indicated how vivid, emotionally intense, and distressing the event was at the time when they experienced it and when they later recalled the event. Scales ranged from 0 (not at all) to 10 (extremely so). To ensure sufficient anger, eligible participants rated their anger as ≥6 when the event occurred and ≥4 following recall.

Imaging session

Approximately 3–4 weeks later, participants arrived at the neuroimaging facility. Participants were instructed to use the memory that they recalled during the initial session and were told that they would be asked to think about this memory from different perspectives. Whole-brain 3D structural images were followed by functional images. Functional scans started with participants engaging in a 2-min relaxation baseline, during which they were instructed to stare at a green circle in the center of the screen (visible through mirrors) and relax. The word “relax” appeared below the circle. Participants were then asked to briefly recall the anger-inducing autobiographical memory via instructions on the screen. All participants verbally indicated that they recalled the memory in less than 45 s.

Following memory recall, participants engaged in 3 counterbalanced emotion regulation conditions: reappraisal, analytical rumination, and angry rumination. Each condition consisted of a set of instructions presented for 45 s and 6 randomized statements that were presented for 25 s each (see Appendix A for materials). The instructions informed participants how to think about the anger-inducing memory. The specific statements helped guide participants by elaborating on the instructions. In the reappraisal condition, participants were asked to think about the event “...in a different, more objective and positive way”. These instructions were adapted from previous studies (Gross, 1998; Ray et al., 2008; Richards and Gross, 2000). In the analytical rumination condition, participants were instructed to think about the memory “...in a way that brings to mind the causes and consequences of the event” (e.g., Watkins, 2008; Watkins and Moulds, 2005). The angry rumination condition was adapted from Denson et al. (2009) (e.g., “...feelings and emotional aspects of the event”). A relaxation baseline was used in between each condition for 30 s to reduce carry-over effects and distinguish reappraisal from relaxation. This involved viewing a green circle and the word “relax”.
Post-scan questionnaire

Following the scan, participants were taken to another room where they rated how they felt during each emotion regulation block using a MACL assessing angry affect (e.g., angry, annoyed, grouchy; α = .80). Participants completed emotion regulation manipulation checks such as “To what extent did you focus on angry thoughts?” for each of the 3 blocks (Appendix B; reappraisal α = .79, analytical rumination α = .50, and emotion-focused α = .65). These scales ranged from 1 (not at all) to 7 (extremely so).

MRI acquisition

Participants viewed the tasks through mirrors, which were presented on a high-resolution monitor placed at the end of a Philips Achieva X-Series 3-Tesla whole-body scanner with an 8-channel head coil and parallel imaging system. Padded foam head constraints controlled participant movement. Once participants were situated in the scanner, a localizer scan was conducted to ensure proper image acquisition. Next, we acquired a T1 anatomical 3D structural dataset (180 slices, FOV = 256 mm, voxel size = 1 × 1 × 1 mm). Functional images were acquired with a single scan using a whole-brain EPI pulse sequence with sagittal slices and 2.5 mm SENSE acceleration (50 slices, slice thickness = 2.5 mm, voxel size = 2.14 × 2.14 × 2.5 mm, FOV = 240 mm, TE = 30 ms, TR = 3000 ms, 90° flip angle). This sequence was chosen after trying a number of sequences as it provided the best signal levels and smallest distortion in the OFC. The initial 4 fMRI volumes were discarded by the scanner.

Statistical analyses

The sagittal EPI slices imaged substantial amounts of non-brain tissue that could interfere with motion correction. Accordingly, as a first step BET from the FSL package (Smith et al., 2004) was used to remove all non-brain components in the EPI images. After this step the data were imported to BrainVoyager QX where all subsequent preprocessing was performed. Images were 3D motion corrected and spatially smoothed with a 4.28 mm Gaussian filter. Brains were normalized via Talairach transformation (Talairach and Tournoux, 1988), and regions of interest (ROIs) were checked against the Talairach Daemon which is an electronic Talairach atlas (Lancaster et al., 1997). Functional images were coregistered with the normalized structural images. Type I error was controlled for in all functional imaging using a cluster-size threshold (Forman et al., 1995). These values were selected based on the AlphaSim Monte Carlo simulation method from the National Institute of Mental Health’s Analysis of Functional Neuroimaging collaborative (http://afni.nimh.nih.gov/afni). This method, which is frequently used in fMRI research (e.g., Goldin et al., 2008; Kross et al., 2009; McRae et al., 2009; Urry et al., 2006), allows for estimation of the probability of a false detection based on the researchers’ own neuroimaging parameters and autocorrelation. Our cluster-size threshold (p < .01, 25 contiguous voxels) controlled for Type I error for the whole brain at p < .007. We relied on random effects general linear model (GLM) group analyses for the inferential statistics. In a within-participants block design, the regressors included the 4 tasks of interest: relaxation baseline, reappraisal, analytical rumination and angry rumination. All blood-oxygen-level dependent (BOLD) responses were adjusted for the hemodynamic response function.

To test for mean differences in BOLD responses between the conditions, we selected clusters for these analyses based on the results of our whole-brain conjunction analysis. The conjunction analysis simultaneously tested each condition against baseline (e.g., [reappraisal + 1, baseline − 1] ^ [analytical rumination + 1, baseline − 1] ^ [angry rumination + 1, baseline − 1]). The activity in these clusters was averaged such that mean signal intensity was calculated for each participant for each cluster listed in the tables. The instruction periods were not modeled in our analyses, nor were the relaxation periods between conditions. Thus the mean activation was calculated on 50 time points per condition for each participant. These BOLD responses were exported to SPSS and we then computed one-way repeated measures ANOVAs on the average activations in these clusters using regions identified in the conjunction.

To test for functional connectivity between top-down prefrontal control regions (i.e., lateral PFC and OFC) and subcortical activity, we conducted psychophysiological interaction (PPI) analyses (Friston et al., 1997). The mean signal intensity of the time courses from the active clusters identified in the whole-brain analyses were exported to SPSS (50 time points per condition). We used 6 regions as seeds: IFG (BA44), IFG (BA45), IFG (BA47), posterior OFC, anterior OFC, and medial OFC. Activation was averaged over the whole cluster. We dummy coded the conditions and created interaction terms of the z-transformed clusters with the condition variable. We then entered the main effects and interaction term in hierarchical regression analyses. In the presence of a significant interaction, follow-up tests were performed to determine the correlation between the cortical control regions and subcortical activity within each condition. All statistical tests are two-tailed (α = .05), unless otherwise stated. For all analyses conducted in SPSS, familywise error was controlled with the false discovery rate, q(FDR) < .05.

Results

Manipulation checks

A 3 (emotion regulation condition) × 3 (manipulation check type) repeated measures ANOVA examined the extent that participants reported engaging in reappraisal, analytical rumination, and angry rumination in each emotion regulation condition. As expected, there was a significant interaction between the emotion regulation strategy participants were instructed to use and the emotion regulation strategy that participants reported using during each of the 3 conditions, F(4, 80) = 40.07, p < .001, d = 2.78. Specifically, when participants were instructed to reappraise they reported doing so to a greater extent (M = 4.70, SD = 0.98) than when they were instructed to engage in analytical rumination, M = 4.09, p < .001, d = 1.14, (M = 3.26, SD = 1.40), or angry rumination, M = 8.09, p < .001, d = 1.77, (M = 2.33, SD = 1.23). Participants reported engaging in more analytical rumination during the analytical rumination condition (M = 5.14, SD = 0.75) than during the reappraisal condition, M = 4.28, p < .001, d = .93, (M = 3.79, SD = 1.16), or angry rumination condition, M = 6.25, p < .001, d = 1.38, (M = 3.21, SD = 1.35). During the angry rumination condition they reported engaging in more angry rumination (M = 5.62, SD = 1.06) than during the reappraisal condition, M = 8.70, p < .001, d = 1.90, (M = 2.58, SD = 1.19), or analytical rumination condition, M = 7.93, p < .001, d = 1.78, (M = 3.50, SD = 1.40). These data suggest effective emotion regulation manipulations.

Self-reported anger

A one-way repeated measures ANOVA was used to assess angry affect during reappraisal, analytical rumination, and angry rumination. There was a significant main effect of emotion regulation condition, F(2,40) = 12.40, p < .001, d = 1.56 (see Fig. 1). Follow-up comparisons revealed that as hypothesized, participants reported experiencing less anger during reappraisal than during analytical rumination, t(20) = −2.82, p = .01, d = −.61, or angry rumination, t(20) = −4.46, p < .001, d = −.97. As predicted, participants reported experiencing less anger during analytical rumination than during angry rumination, t(20) = −2.55, p = .01, d = −.55. These results suggest that reappraisal was the most effective method for regulating anger, followed by analytical rumination. Angry rumination was the least effective anger
regulation strategy. Self-reported anger did not significantly decrease across conditions over time, $F<1$.

Functional imaging results

**Testing the mean level hypothesis**

To identify which regions were commonly activated when regulating anger, we conducted a whole-brain conjunction analysis (Friston et al., 1999) simultaneously contrasting BOLD responses from all three emotion regulation conditions against the relaxation baseline period (Table 1). Regions implicated in cortical control including the IFG and OFC were active during all three conditions, observed relative to baseline. The putamen and regions implicated in emotion such as the amygdala, insula and thalamus were also active compared to baseline. Repeated measures ANOVAs using regions identified in the conjunction analysis showed no significant effects. These results suggest that the strength of neural activation between the three emotion regulation conditions did not differ.

**Testing the functional connectivity hypothesis**

PPI analyses using regions identified in the conjunction analysis were conducted to determine whether regions implicated in top-down emotion regulation (i.e., the OFC and IFG) would modulate activity in limbic and subcortical regions (i.e., amygdala, thalamus, insula, putamen, and caudate). A significant IFG (BA 44)×condition interaction was obtained when predicting amygdala activity, $b=-11.04, SE=4.18, t(146)=-2.54, p=.01$. Follow-up tests revealed significant positive associations between the IFG (BA44) and amygdala during analytical rumination and angry rumination, but no correlation during reappraisal (Fig. 2, panel A).

There was a significant IFG (BA45)×condition interaction for the lateral posterior thalamus, $b=-6.87, SE=2.71, t(146)=-2.54, p=.01$. Follow-up tests revealed a significant positive association between the IFG and lateral posterior thalamus during analytical rumination (Fig. 2, panel B), but no correlations during reappraisal and angry rumination.

There was also a significant IFG (BA45)×condition interaction with the ventral lateral thalamus, $b=-7.37, SE=2.64, t(146)=-2.79, p=.01$. Follow-up tests indicate that there was a significant positive association between the IFG and ventral lateral thalamus during both analytical and angry rumination, but no association during reappraisal (Fig. 2, panel C).

Following Meng et al. (1992), a one-tailed procedure was used to compare the magnitude of functional connectivity between dependent correlation coefficients. Specifically, there was significantly greater functional connectivity between the IFG (BA45) and lateral posterior thalamus during analytical rumination ($Z=-2.12, p=.02$) and angry rumination ($Z=-1.94, p=.03$) than during reappraisal (Fig. 2, panel B). There was also greater functional connectivity between the IFG (BA45) and ventral lateral thalamus during analytical rumination ($Z=-2.21, p=.01$) and angry rumination ($Z=-2.1, p=.02$) in comparison to reappraisal. There were no other significant differences in the magnitude of the correlations.

**Discussion**

The present research provides insight into a phenomenon with important social and economic implications: anger regulation. This work is the first to directly investigate the neural regions recruited during three different types of anger regulation. Consistent with past research, we found that reappraisal produced the lowest levels of self-reported anger (Denson et al., 2011a; Mauss et al., 2007; Memedovic et al., 2010; Ray et al., 2008) and analytical rumination produced less self-reported anger than angry rumination (Ayduk and Kross, 2008; Kross et al., 2005). In terms of BOLD responses, a conjunction analysis identified top-down activation in the IFG and OFC, which is consistent

<table>
<thead>
<tr>
<th>Region of activation</th>
<th>Hemisphere</th>
<th>Brodmann area</th>
<th>Talairach coordinates</th>
<th>Cluster size</th>
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</thead>
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<tr>
<td>Medial orbital gyrus</td>
<td>Right</td>
<td>11/47</td>
<td>x: 20 y: 26 z: -10</td>
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<tr>
<td>Anterior orbital gyrus</td>
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<td>x: 23 y: 37 z: -5</td>
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<tr>
<td>Posterior orbital gyrus</td>
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<td>47</td>
<td>x: 27 y: 24 z: -5</td>
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<tr>
<td>Inferior frontal gyrus</td>
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<td>47</td>
<td>x: 23 y: 30 z: -8</td>
<td>508</td>
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<tr>
<td>Inferior frontal gyrus</td>
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<td>45</td>
<td>x: 42 y: 20 z: 4</td>
<td>675</td>
</tr>
<tr>
<td>Insula</td>
<td>Right</td>
<td>13</td>
<td>x: 40 y: 16 z: 5</td>
<td>872</td>
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<tr>
<td>Amygdala</td>
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<td>x: 43 y: 6 z: 7</td>
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<tr>
<td>Thalamus ventral lateral nucleus</td>
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<tr>
<td>Lateral globus pallidus</td>
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with studies investigating anger regulation indirectly (Alia-Klein et al., 2007; Mehta and Beer, 2010) and the regulation of general negative affect (e.g., Ochsner et al., 2002). Anger regulation also recruited regions associated with interoceptive awareness including the right insula, IFG, and thalamus (Critchley et al., 2004; Pollatos et al., 2007). Greater interoceptive awareness has been associated with increased self-reported negative emotion (Critchley et al., 2004). We also obtained increased activation in regions associated with emotion processing

Fig. 2. Functional connectivity analyses during reappraisal, analytical rumination, and angry rumination. Panel A displays a significant positive association between the inferior frontal gyrus (BA 44) and amygdala during both analytical rumination and angry rumination. Panel B displays a significant positive association between the inferior frontal gyrus (BA 45) and lateral posterior thalamus during analytical rumination. Panel C shows a significant positive association between the inferior frontal gyrus (BA 45) and ventral lateral thalamus during analytical rumination and angry rumination. Signal intensity values are z-transformed. *p < .05, **p < .01, two-tailed.
including the amygdala (Hare et al., 2005), and the thalamus, which has been implicated in mood, arousal and episodic memory (Taber et al., 2004). We also found increased activation in the right putamen, which is active when viewing hated individuals (Zeki and Romaya, 2008) and is involved in goal directed behavior and motor control (Balleine et al., 2007; Gray, 1990).

Of particular interest was increased in the medial, anterior, and posterior OFC. Many neuroscientifically-informed theories emphasize dysregulation in a neural circuit involving the OFC as a risk factor for aggressive and violent behavior (Blair, 2004; Davidson et al., 2000; MacDonald, 2008; Raine, 2008; Siever, 2008). Functional distinctions have been made between the lateral-medial OFC and the anterior-posterior divisions of the OFC. The medial OFC is involved in evaluating the affective value of rewarding stimuli (Kringelbach and Rolls, 2004) and may also be involved in anger regulation (Mehta and Beer, 2010). By contrast, the lateral OFC is activated by reversal learning tasks that require inhibiting behavioral responses to stimuli that have been previously paired with a reward (Elliot et al., 2000; O’Doherty et al., 2003). We did not find activation in the lateral OFC, but such activation has sometimes been observed in prior research on emotion regulation (Alia-Klein et al., 2007; Phan et al., 2005). The anterior and posterior OFC are thought to be involved in the processing of abstract and primary reinforcers respectively (Kringelbach and Rolls, 2004). Abstract reinforcers in the context of anger regulation may have included representations of relationship restoration whereas primary reinforcers may have included re-experienced social pain. Future research on the role of these subregions of the OFC may shed light on the neural basis of effective anger regulation.

Although the mean level of activation did not significantly differ between the three emotion regulation strategies, the strategies induced differences in functional connectivity. Functional connectivity between the IFG, amygdala, and thalamus distinguished reappraisal from rumination. Specifically, during both types of rumination the IFG was positively correlated with increased amygdala and thalamus activation. Moreover, these correlations were larger during rumination than during reappraisal. These findings are consistent with two possible explanations. The first is that rumination requires increased recruitment of top-down control processes to regulate increased subcortical activation — but is ultimately ineffective in reducing anger. The second possibility is that the increased positive connectivity between subcortical limbic activation and the IFG forms a feedback loop in which abstract functions supported by the PFC, such as reflecting on angry feelings and planning revenge, may be supported by positive connectivity between cortical and subcortical regions. This notion explains how rumination heightens arousal and anger. The thalamus activation is noteworthy because it plays a general role in emotion including emotional processing, emotion experience, representing feelings, and emotional control (Hooker et al., 2008; Marchand, 2010; Reiman, et al., 1997). Increased thalamic activity has been found in studies involving the recall of angry autobiographical memories (Damasio et al., 2000; Kimbrell et al., 1999), but also for other emotions and a variety of other emotion induction techniques such as film clips (Lane et al., 1997). The thalamus also has a number of different functions including a role in different types of memory such as episodic memory (Taber et al., 2004; Wiggs et al., 1999). Although activation of the thalamus in the present research is consistent with all of these functions, we suggest the pattern of functional connectivity in the present study is most consistent with the role of the thalamus in affective processes.

Evidence from primate studies reveals that few direct connections between the lateral prefrontal cortex and amygdala exist (Ghashghaei and Barbas, 2002). Nonetheless, there may be a number of other pathways that mediate this connection. The lateral prefrontal cortex has connections with the OFC (Barbas, 2007), and the OFC has direct connections with the amygdala (Ghashghaei and Barbas, 2002). It is also worth noting that contrary to some prior research, an inverse relationship between the IFG and subcortical regions during reappraisal was not observed (Goldin et al., 2008; Ochsner et al., 2002). Nonetheless, connectivity between the IFG and thalamus were in the hypothesized negative direction, consistent with the notion that reappraisal involves the downregulation of subcortical activity.

The present results consistently displayed a pattern of right laterality. Although negative affective states tend to activate the right PFC, anger is an approach-oriented negative emotion, which is typically associated with relatively greater left PFC activity (Harmon-Jones and Sigelman, 2001). One potential explanation for the increased right cortical activity found in the present study could be the nature of the anger-inducing memory recalled. The memories that participants recalled occurred in the past year and may have varied in terms of how resolved the events were. Similarly, Harmon-Jones et al. (2003) examined prefrontal laterality among undergraduates facing an anger-inducing tuition increase. Participants were led to believe that they could sign a petition against the increase or do nothing. Participants showed increased left activation when they could sign the petition, but this effect was ameliorated when participants could do nothing to prevent the anger-inducing event. Another possibility is that increased interoceptive awareness associated with anger regulation may account for the right lateralization. Right lateralization has been found in studies examining interoceptive awareness of cardiovascular responses (Pollatos et al., 2007).

One shortcoming of the current experiment was the placement of the self-reported anger measure at the end of the study rather than immediately following each emotion regulation block. It is possible that obtaining self-report data after removing participants from the scanner might have introduced retrospective memory biases. In addition, the small sample size likely reduced statistical power to detect effects such as mean-level differences between the conditions. Because we examined healthy undergraduates, it remains to be seen whether these findings are applicable to other populations of interest. For instance, future research could investigate the neural underpinnings and functional connectivity involved in different types of anger regulation in individuals prone to frequent anger and aggression (e.g., domestic violence offenders).

The present research contributes to the literature by revealing differences in functional connectivity that differentiate reappraisal from rumination. Understanding interactions between neural regions during different emotion regulation strategies may eventually help form the basis of interventions aimed at reducing the negative consequences of poor anger regulation such as violence and cardiovascular disease.

Appendix A. Anger regulation stimuli

Reappraisal

‘I want you to think about the anger-inducing event that you wrote down previously. However, this time I want you to think about it in a different, more objective and positive way. Try to think 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 think about factual, non-emotional details, such as where and when the event occurred’.

1. Think about the non-emotional details of the event, such as where and when it occurred.
2. Think about the event from the perspective of an objective, impartial third person.
3. Think about any valuable lessons you have learned from the event.
4. Think about the event in a way to maintain a neutral mood.
5. Think about any positive aspects of the event.
6. Think about ways you could improve in the future if the same event were to arise.
‘I want you to think about the anger-inducing event that you wrote down previously. However, this time I want you to think about it in a way that brings to mind the causes and consequences of the event. Try to think about the reasons for and the causes of the event, what it means that it happened the way it did, and the future implications of the event. Try to think about why people acted the way they did and what the event means to you.’

1. Think about why the other people involved in the event acted the way they did.
2. Think about why the event happened.
3. Think about what it means that you experienced the event.
4. Think about the future implications and consequences of the event.
5. Think about the thoughts that ran through your mind during the event.
6. Think about why the event unfolded the way it did.

**Analytical rumination**

‘I want you to think about the anger-inducing event that you wrote down previously. However, this time I want you to think about it in an emotional way. Try to think 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’.

1. Think about the anger-related physical sensations you experienced during the event.
2. Think about the feelings and emotions you felt toward the other people in the event.
3. Think about exactly what happened during the event.
4. Think about how you might like to retaliate in response to the event.
5. Think about your angry mood during the event.
6. Think about the feelings and emotions you experienced during the event.

**Appendix B. Anger regulation manipulation check items**

**Reappraisal**

1. To what extent did you re-consider the event from an objective perspective?
2. To what extent did you consider the positive aspects of the event?
3. To what extent did you learn how to deal with frustrating or anger-inducing events?

**Analytical rumination**

1. To what extent did you think about why the event happened?
2. To what extent did you think about the future implications of the event?
3. To what extent did you think about the causes of the event?

**Angry rumination**

1. To what extent did you focus on angry thoughts?
2. To what extent did you focus on your anger toward others?
3. To what extent did you focus on your emotional response to the event?

**References**


