The Angry Brain: Neural Correlates of Anger, Angry Rumination, and Aggressive Personality

Thomas F. Denson[1], William C. Pedersen[2*], Jaclyn Ronquillo[3*], and Anirvan S. Nandy[3]

Abstract

■ Very little is known about the neural circuitry guiding anger, angry rumination, and aggressive personality. In the present fMRI experiment, participants were insulted and induced to ruminate. Activity in the dorsal anterior cingulate cortex was positively related to self-reported feelings of anger and individual differences in general aggression. Activity in the medial prefrontal cortex was related to self-reported rumination and individual differences in displaced aggression. Increased activation in the hippocampus, insula, and cingulate cortex following the provocation predicted subsequent self-reported rumination. These findings increase our understanding of the neural processes associated with the risk for aggressive behavior by specifying neural regions that mediate the subjective experience of anger and angry rumination as well as the neural pathways linked to different types of aggressive behavior. ■

INTRODUCTION

Despite the enormous costs of anger and aggression, very little is known about the neural mechanisms guiding these phenomena (Davidson, Putnam, & Larson, 2000). Understanding these neural mechanisms is important because it provides insight into concrete, biological processes that predispose individuals for aggression. The uncovering of these processes has long been a central concern of neuroscience as well as social, clinical, personality, and forensic psychology.

For most people, angry feelings dissipate within 10 to 15 min (Tyson, 1998; Fridhandler & Averill, 1982). However, recent research suggests that dwelling on anger-inducing experiences (i.e., angry rumination) may be particularly harmful because it increases aggression over extended periods of time, even toward the innocent (Bushman, Bonacci, Pedersen, Vasquez, & Miller, 2005; Bushman, 2002). Despite its importance, no study has examined the neural substrate of angry rumination. In the first fMRI experiment to do so, we provide a broad view of the neural processes that occur when angered, when ruminating about an interpersonal insult, and how these processes vary as a function of aggressive personality. The present social neuroscience approach increases our understanding of the neural processes underlying risk for aggression.

[1]University of New South Wales, Sydney, Australia, [2]California State University, Long Beach, [3]University of Southern California
*These authors contributed equally to the research.

Neural Regions Underlying Anger

Identifying the neural foundations of anger has proven difficult because prior work has relied on patients with brain lesions or neuroimaging paradigms that examined anger indirectly. This latter group of just nine studies investigated neural responses to angry faces and brain regions active during the recall of anger-inducing life experiences. Two recent meta-analyses of these studies revealed that some of the most prominent areas of brain activation were the medial prefrontal cortex (mPFC), the ventromedial PFC (vmPFC), the anterior cingulate cortex (ACC), the posterior cingulate cortex (PCC), the lateral PFC, and the thalamus (Murphy, Nimmo-Smith, & Lawrence, 2003; Phan, Wager, Taylor, & Liberzon, 2002).[1]

A social neuroscience approach combines elements of social psychology and cognitive neuroscience. In a review of the literature on aggressive behavior, Anderson and Bushman (2002) refer to interpersonal provocation as ''perhaps the most important single cause of human aggression'' (p. 37). In fact, it is such a reliable means of inducing anger and aggression that it is, by far, the most common experimental manipulation used in social psychological research. No functional imaging study, to our knowledge, has specifically examined an anger-inducing interpersonal insult, despite its ecological validity and relevance to real-world aggression.

Moreover, no study has examined the neural substrate that mediates the *subjective experience* of anger. We hypothesized a special role for the dorsal ACC (dACC) in this regard. Activity in the dACC is associated with a number of negative emotions including the intensity of

social distress following social rejection (Eisenberger, Lieberman, & Williams, 2003), and distress associated with physical pain (Rainville, Duncan, Price, Carrier, & Bushnell, 1997). The dACC has also been discussed in terms of a ''neural alarm system'' because it is active in response to incongruent stimuli and goals (Kross, Egner, Ochsner, Hirsch, & Downey, 2007; Eisenberger & Lieberman, 2004). Interpersonal provocation is likely just such a stimulus. Thus, in response to the interpersonal provocation of the current study, we hypothesized that the dACC would be positively related to the intensity of self-reported anger.

Neural Regions Underlying Angry Rumination

When upset by a provocation, there are a number of emotion regulation strategies one may use to cope with the aversive event. Rumination is one such strategy. Two types of rumination have been examined in relation to anger. One is known as provocation-focused rumination, which involves thinking about and reliving a negative event or an angering incident (Sukhodolsky, Golub, & Cromwell, 2001; Caprara, 1986). A second type is self-focused rumination, which refers to directing attention inward on the self, particularly on one's own negative emotions (Trapnell & Campbell, 1999; Lyubomirsky & Nolen-Hoeksema, 1995; Nolen-Hoeksema & Morrow, 1993). Following a provocation, both types of rumination increase anger and aggression (Denson, Pedersen, & Miller, 2006; Bushman et al., 2005, Experiment 2; Rusting & Nolen-Hoeksema, 1998).

Because angry rumination contains components of self-reflection, social cognition, negative affect, and emotion regulation by maintaining or increasing anger after a provocation, it should involve the recruitment of brain regions associated with these mental events such as the mPFC, the lateral PFC, the insula, and the cingulate cortex (Amodio & Frith, 2006; Lévesque et al., 2003; Ochsner, Bunge, Gross, & Gabrieli, 2002; Phan et al., 2002). Although no neuroimaging study has directly manipulated rumination, Ray et al. (2005) reported that when participants were asked to decrease their negative affective responses to aversive photographs, trait rumination was correlated with ACC and mPFC activity. The mPFC is associated with the self-awareness of emotions and self-relevant cognition (Macrae, Moran, Heatherton, Banfield, & Kelley, 2004; Ochsner et al., 2004; Lane, Fink, Chau, & Dolan, 1997). The mPFC is active when participants are asked to monitor their emotional state, reflect on their feelings, and when reappraising their responses to distressing visual stimuli (Amodio & Frith, 2006; Ochsner et al., 2002, 2004). Moreover, the mPFC also appears related to the personality trait of self-awareness (Eisenberger, Lieberman, & Satpute, 2005). Because rumination involves thinking about and regulating one's affective state, the mPFC should be especially relevant to rumination.

As was the case with anger, we also sought to uncover the neural systems mediating the subjective experience of rumination. Following the above reasoning, we expected activity in the mPFC to be positively correlated with self-reported rumination during the rumination task (e.g., Ray et al., 2005; Ochsner et al., 2002, 2004; Lane et al., 1997). We also expected that regions associated with memory encoding would be especially important in this regard (Kensinger, Clarke, & Corkin, 2003). Because real-world provocations are highly salient and self-relevant, we expected that the hippocampus should be active in response to the provocation. Because deeper encoding should increase the accessibility of the provocation in memory, the degree of hippocampus activity should be a particularly good indicator of the intensity of self-reported rumination. A second, compatible possibility concerns the role that the hippocampus is posited to play in monitoring discrepancies between expected events and actual situations (Gray & McNaughton, 2000). This discrepancy-monitoring system is believed to motivate behavior designed to reduce the problem that produced the discrepancy. Rumination might be one such means of mentally resolving the conflict. Thus, in the context of interpersonal provocation, research and theory suggest that hippocampus activity in response to the provocation and mPFC activity during the rumination task would correlate with self-reported angry rumination.

Neural Regions Underlying Aggressive Personality

Why do some people angrily ''fly off the handle'' in response to provocation, whereas others ''take it out'' on innocents such as their romantic partners? Social psychology has unequivocally demonstrated that even mentally healthy individuals are capable of consequential acts of aggression (Anderson & Bushman, 2002), and some individuals are more prone to aggression than others (Bettencourt, Talley, Benjamin, & Valentine, 2006). People often respond to identical provocations very differently. Recent research supports these notions by providing evidence for the existence of two unique aggressive personality dimensions (Denson et al., 2006).

The first aggressive personality dimension is *general aggression*, which is characterized by frequent anger and direct retaliation in response to interpersonal provocation in both laboratory experiments and real-world settings (Bettencourt et al., 2006; Buss & Perry, 1992). Because we expected the dACC to be related to the subjective experience of anger, we also expected general aggression to be associated with activity in the dACC because this personality dimension is associated with intense anger and impulsive aggression. Indirect support for this hypothesis comes from an fMRI study that manipulated ostracism. In this study, a composite measure of trait anger and hostility was moderately positively

correlated with reactivity in the dACC (Eisenberger, Way, Taylor, Welch, & Lieberman, 2007).

The second aggressive personality dimension is *displaced aggression*, which is characterized by responding to insults with *rumination* instead of immediate aggression, and eventually ''taking out'' aggressive urges on the innocent. When those high in displaced aggression are provoked, they harm innocents in laboratory experiments and report increased levels of romantic partner abuse and driving aggression, whereas those high in general aggression do not (Denson et al., 2006). We expected displaced aggression to be primarily associated with activity in the mPFC and the hippocampus. Because individuals high in displaced aggression report ruminating when provoked, the mPFC and the hippocampus should be especially relevant to individual differences in displaced aggression, but not general aggression.

METHODS

Participants

Twenty right-handed undergraduates (12 women; M_{age} = 18.68, SD_{age} = 0.75, 70% white) volunteered to participate in exchange for extra course credit. In order to reduce suspicion, participants were told that they would be participating in an experiment on cognitive ability and mental imagery.

Materials and Procedure

Initial Questionnaire Session

During an initial session, participants completed a safety screening questionnaire, the 29-item Aggression Questionnaire (AQ; α = .93, M = 3.28, SD = 1.04; Buss & Perry, 1992), and the 31-item Displaced Aggression Questionnaire (DAQ; α = .96, M = 2.80, SD = 1.80; Denson et al., 2006), which assess individual differences in general aggression and displaced aggression, respectively. The AQ is reliable and has proven useful in predicting laboratory and real-world aggression (Bushman & Wells, 1998; Buss & Perry, 1992). The DAQ has good internal consistency, test–retest reliability, convergent validity, and discriminant validity (Denson et al., 2006). By comparison with the AQ, the DAQ is a stronger predictor of laboratory displaced aggression and real-world indicators of displaced aggression such as domestic abuse and road rage (Denson et al., 2006). Participants responded on a scale ranging from 1 (*extremely uncharacteristic of me*) to 7 (*extremely characteristic of me*). These questionnaires were completed as part of a larger packet of measures unrelated to anger and aggression. No participants reported noticing a suspicious relationship between the initial questionnaire session and the imaging experiment.

Provocation Procedure

Approximately 10 to 14 days later, participants returned to the imaging center for the experiment. Upon arrival, participants completed baseline measures of mood with the short version of the Profile of Mood States (POMS; Shacham, 1983). Of particular interest was the anger/hostility subscale (α = .70). Two minutes of functional baseline fixation data were collected while participants were instructed to stare at a green fixation point in the center of the screen visible through mirrors. Using a provocation manipulation adapted from previous research, participants were presented with four easy and eight difficult anagrams for 15 sec each (e.g., Pedersen, Gonzales, & Miller, 2000). They were asked to state their answer out loud or say ''no answer'' if they did not know the answer. As part of the provocation manipulation, the experimenter interrupted participants thrice requesting that they speak louder. During the third interruption, which served as the anger induction, the experimenter stated in a rude, upset, and condescending tone of voice ''Look, this is the third time I have had to say this! Can't you follow directions?'' Immediately following the insult (<500 msec), an additional 2 min of functional data were collected while participants stared at a fixation point. Because the insinuation was that participants were not intelligent enough to follow simple instructions, the provocation manipulation represented the delivery of an unjustified insult. This provocation manipulation has successfully angered participants in prior research (Pedersen et al., 2000).

Directed Rumination Manipulation

In a within-participants design, individuals were assigned to the provocation-focused rumination, self-focused rumination, and distraction conditions in counterbalanced order via a Graeco-Latin square design. In the *provocation-focused rumination* condition, participants were presented with a series of statements on the monitor and asked to think about each statement for 15 sec each (e.g., ''Think about whom you have interacted with in the experiment up to this point,'' ''Think about exactly what you have done from the start of the study until now''). Statements from the self-focused rumination and distraction conditions were taken from Rusting and Nolen-Hoeksema (1998; also used in Bushman et al., 2005). In the *self-focused rumination condition*, participants were asked to think about a series of self-referential statements that did not mention anger or other emotions (e.g., ''Think about why people treat you the way they do,'' ''Think about why you react the way you do''). In the *distraction condition*, participants were asked to think about a series of affectively neutral statements (e.g., ''Think about the layout of the local post office,'' ''Think about a double-decker bus driving down the street''). In each of the three conditions, 12 statements were presented for 15 sec each,

such that each condition took 3 min to complete. The conditions were separated by 16-sec rest periods. Functional EPI whole-brain images were taken during the entire directed rumination period.

Questionnaires

Upon completion of scanning, participants reported potential emotional responses to the provocation with the Positive and Negative Affect Schedule (PANAS-X; Watson & Clark, 1994). Of particular interest was the hostility subscale which assesses angry affect ($\alpha = .81$). We also assessed self-reported rumination during each of the three blocks of rumination. These were ratings of *how often* and *how strongly* (1 = not at all, 7 = very often) participants thought about their performance on the anagram task. All participants were then fully debriefed and thanked.

Image Acquisition

Participants viewed the experimental tasks through mirrors, which were presented on a high-resolution monitor placed at the end of a Siemens Magnetom 3-T scanner. 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 3-D structural images (MP-RAGE, 192 slices, FOV = 256 mm, thickness = 1 mm, TR = 2070 msec, TE = 4.14 msec). Prior to beginning the functional scan, we visually inspected a dummy EPI scan (8 sec) to ensure the quality of the functional data. Whole-brain functional images were acquired with interleaved EPI pulse sequence (29 axial slices, slice thickness = 4 mm, FOV = 24 cm, TE = 68 msec, TR = 2000 msec, 90° flip angle).

Statistical Analysis

All analyses were conducted with Brain Voyager QX (Brain Innovation). Data were preprocessed using motion and scan-time corrections, and smoothed with a Gaussian temporal filter. Brains were normalized via Talairach transformation (Talairach & Tournoux, 1988), and regions were identified using the Talairach Daemon (Lancaster, Summerln, Rainey, Freitas, & Fox, 1997), which is an electronic database of Talairach coordinates. Functional images were coregistered with the normalized structural images. All BOLD responses are expressed in percent signal change. For comparisons between conditions, whole-brain random-effects general linear model (GLM) group analyses were conducted with participant specified as the random factor. The provocation was modeled as the difference in activation during the two fixation blocks (i.e., 2 min provocation fixation > 2 min baseline fixation) adjusted for the hemodynamic response function. For the rumination data, because only minor differences were found between provocation- and self-focused rumination, we averaged these two conditions and contrasted them against the distraction condition (rumination > distraction).[2] The rumination scan was modeled as the difference in activation between the rumination blocks and the distraction condition (rumination > distraction) adjusted for the hemodynamic response function. We controlled Type I error with the false discovery rate (FDR) set at .05, voxelwise $p < .005$.

For correlating the self-report data with BOLD responses, we selected clusters for these analyses based on the results of our whole-brain main-effects analyses. The activity in these clusters was averaged such that a mean percent signal change was calculated for each participant. We then computed correlations between our self-report measures and the average activity in these ROIs.

RESULTS

Manipulation Checks

Participants reported an increase in anger from baseline as a result of the provocation procedure, thus indicating a successful anger induction [$t(15) = 5.32$, $p < .001$, $d = 1.52$]. As expected, participants reported thinking more about the provocation during the rumination block than during the distraction block [$t(19) = 3.44$, $p = .003$, $d = 0.78$], thus indicating a successful rumination manipulation. For the personality variables, we computed partial correlations controlling for gender, because men rated themselves higher than women in general aggression [$t(18) = 3.48, p = .003, d = 1.75$] and displaced aggression [$t(18) = 3.39, p = .003, d = 1.71$]. Consistent with prior research (Denson et al., 2006), general and displaced aggression were significantly correlated ($r = .68, p = .001$).

Anger: Neural Regions, Subjective Experience, and Personality[3]

Table 1 displays regions active in response to the provocation (i.e., provocation > baseline fixation). These data suggest a substantial degree of consistency with prior research using autobiographical recall paradigms and exposure to angry faces (Murphy et al., 2003; Phan et al., 2002). As expected, activation in the left dACC was positively correlated with self-reported feelings of anger ($r = .56, p < .05$), but no other emotions (see Table 2). This provides the first evidence for a neurophysiological basis underlying the intensity of the subjective anger experience. By contrast, activity in the right dACC was correlated with the Guilt subscale of the PANAS-X. We expand upon this finding in the Discussion section.

As expected, general aggression was associated with increased activity in the left dACC following provocation

Table 1. Brain Regions Active after Exposure to an Interpersonal Provocation Relative to Baseline Fixation

	Talairach Coordinates					
Region	*x*	*y*	*z*	*Cluster Size (Voxels)*	*Mean (SE) Percent Signal Change*	*Significance Test*
Dorsal Anterior Cingulate						
Right	8	24	34	787	0.62 (0.07)	$t(15) = 9.23, p < .00001$
Left	−7	22	33	731	0.59 (0.12)	$t(15) = 4.79, p < .001$
Rostral Anterior Cingulate						
$\text{Right}_{\text{region1}}$	5	32	15	446	0.62 (0.13)	$t(15) = 4.85, p < .001$
$\text{Right}_{\text{region2}}$	4	30	−7	719	1.06 (0.29)	$t(15) = 3.63, p < .001$
Left	−3	33	−8	590	1.25 (0.24)	$t(15) = 5.14, p < .001$
Insula						
Right	37	−2	7	637	0.50 (0.11)	$t(15) = 4.66, p < .001$
Left	−37	4	15	767	0.54 (0.09)	$t(15) = 6.13, p < .0001$
Posterior Cingulate						
Right	5	−52	21	796	0.61 (0.08)	$t(15) = 7.48, p < .0001$
$\text{Left}_{\text{region1}}$	−7	−44	23	240	0.50 (0.07)	$t(15) = 7.02, p < .00001$
$\text{Left}_{\text{region2}}$	−2	−21	28	276	0.59 (0.12)	$t(15) = 5.01, p < .001$
Medial Frontal Gyrus						
$\text{Right}_{\text{region1}}$	6	47	13	764	0.72 (0.10)	$t(15) = 7.20, p < .0001$
$\text{Right}_{\text{region2}}$	5	45	19	562	0.59 (0.07)	$t(15) = 8.65, p < .00001$
Medial Frontal Gyrus						
Left	−5	32	−11	555	1.39 (0.28)	$t(15) = 4.92, p < .001$
Lateral Middle Frontal Gyrus						
Right	33	47	7	504	0.76 (0.11)	$t(15) = 6.68, p < .0001$
Left	−32	47	9	617	0.78 (0.15)	$t(15) = 5.10, p < .001$
Hippocampus						
Right	30	−31	−3	1,013	0.49 (0.07)	$t(15) = 6.73, p < .00001$
Left	−30	−31	−3	934	0.60 (0.08)	$t(15) = 7.22, p < .00001$
Thalamus						
Left	−13	−10	3	675	0.60 (0.12)	$t(15) = 5.02, p < .001$

($r = .61$, $p < .05$), but displaced aggression was not ($r = .24$, *ns*). Moreover, displaced aggression was significantly associated with increased activity in the mPFC ($r = .57$, $p < .05$), but general aggression was not ($r = .37$, *ns*) (see Table 3 and Figure 1). Simultaneously regressing BOLD responses on direct aggression, displaced aggression, and gender revealed an identical pattern of results. Specifically, general aggression predicted dACC activity ($b = .49$, $t = 2.59$, $p = .02$), but displaced aggression did not ($b = -.14$, $t = -0.76$, *ns*, $R^2 = .42$). By contrast, displaced aggression marginally predicted mPFC activity ($b = .21$, $t = 1.85$, $p = .09$), but general aggression did not ($b = .01$, $t = 0.10$, *ns*, $R^2 = .33$). Thus, these different personality dimensions were

Table 2. Correlations between BOLD Response in the dACC and State Mood Measures after Exposure to a Verbal Interpersonal Provocation (Provocation > Baseline)

PANAS Subscales	*Left dACC*	*Right dACC*
Hostility	.56*	.40
Guilt	.42	.58*
Sadness	.19	−.01
Fear	.13	.23
Joviality	.10	−.08
Self-assurance	.28	−.11
Attentiveness	.18	.17
Shyness	.05	−.03
Fatigue	.04	−.26
Serenity	−.07	−.17
Surprise	.23	−.06
Basic positive affect	.26	.01
Basic negative affect	.42	.41
Positive emotion	.21	−.07
Negative emotion	.36	.44

The last four subscales are not independent of the preceding subscales.

*$p < .05$.

associated with the recruitment of separate neural regions when confronted with a provocation. Specifically, general aggression was more strongly correlated with a region associated with the intensity of anger, whereas displaced aggression was more strongly correlated with a region associated with self-reflection, the monitoring of negative emotions, and emotion regulation.

Angry Rumination: Neural Regions and Aggressive Personality

Table 4 displays the regions active during the directed rumination task relative to distraction (rumination > distraction). As expected, rumination increased activity in regions associated with emotion regulation, negative affect, and social cognition such as the cingulate cortex, the mPFC, the lateral PFC, and the insula. Also as expected, displaced aggression was positively associated with activity in the left mPFC ($r = .55$, $p = .02$), but general aggression was not ($r = .22$, *ns*) (Table 3). Simultaneously regressing mPFC activity on direct aggression, displaced aggression, and gender revealed an identical pattern of results. Specifically, displaced aggression predicted mPFC activity ($b = .52$, $t = 2.65$, $p = .02$), but general aggression did not ($b = -.20$, $t = -1.00$, *ns*, $R^2 = .35$). These results further support the notion that individuals who exhibit high levels of displaced aggression tend to ruminate to a greater extent following provocations than those who exhibit low levels of displaced aggression.

Angry Rumination: Subjective Experience

We wished to determine whether the degree of neural activity experienced *following the provocation manipulation* (especially in the hippocampus) would be associated with the degree of self-reported rumination about the provocation during the directed rumination task. This was indeed the case. Activity in the hippocampus following the provocation was correlated with self-reported angry rumination ($r = .51$, $p < .05$), suggesting that those who deeply encoded the provocation in memory and/or were deeply affected by the discrepancy between actual and expected events also tended to ruminate about the insult during the subsequent directed rumination task (Figure 2).

Because displaced aggression is characterized by rumination in response to provocation, we also expected individual differences in this trait to correlate with hippocampal activity. Displaced aggression was moderately correlated with hippocampal activation, yet not significantly so ($r = .40$, $p = .14$). Nonetheless, the magnitude of the relationship between general aggression and hippocampal activity was half the size ($r = .21$, $p = .44$). Although not significant, the magnitude and direction of these results are consistent with our theorizing.

Also of interest was that activity in the right insula was correlated with the degree of self-reported rumination ($r = .54$, $p < .05$). This region incorporates physiological information from the body, which some have suggested forms the neural substrate for the subjective sense of self and feeling states (Critchley, Wiens, Rotshtein, Öhman, & Dolan, 2004; Damasio, 1994). Two additional regions commonly involved in negative emotional experience, the rACC and the PCC, were correlated with self-reported rumination (rs = .60 and .52, ps < .05). These findings are especially noteworthy given that brain activity following the provocation *temporally preceded* self-reported rumination. During the directed rumination task, activation in the left mPFC was marginally related to the intensity of self-reported rumination ($r = .42$, $p = .06$). In summary, regions associated with memory encoding, conflict monitoring, the processing of internal states, and negative affective responses to the provocation correlated with subsequent rumination, whereas increased activity in a region associated with self-reflection, emotion regulation, and social cognition (i.e., the mPFC) was correlated with rumination during the task.

DISCUSSION

Our findings provide novel insight into the neural pathways underlying anger, angry rumination, and aggressive personality. Such understanding represents a first step

Table 3. Correlations between BOLD Activation in the Left dACC and the mPFC and Aggressive Personality Dimensions following Provocation (Provocation > Baseline) and during Rumination (Rumination > Distraction)

Brain Region	*General Aggression*	*Displaced Aggression*
Post-provocation		
dACC	.61*	.24
mPFC	.37	.57*
During Rumination		
dACC	.20	.16
mPFC	.22	.55*

*$p < .05$.

toward forming the basis of successful and enduring, evidence-based aggression-reduction interventions. The present research contributes to our knowledge on these topics in a number of ways. First, we provided evidence that the dACC is related to the *subjective experience* of anger. The emerging picture of the role of the dACC in social–affective contexts is that it may be involved in producing feelings associated with the intensity of a number of emotions that are specific to negative social situations such as interpersonal provocation and rejection (e.g., Eisenberger et al., 2003, 2007; Kross et al., 2007). Our data are also consistent with the conceptualization of the dACC as a ''neural alarm system'' that is sensitive to incongruent stimuli and goals (Kross et al., 2007; Eisenberger & Lieberman, 2004). In our case, the interpersonal provocation was likely unexpected and incongruent with participants' positive self-image. Indeed, in the absence of prior knowledge about an individual, people tend to exhibit an initial positivity bias as a default position (Klar & Giladi, 1997; Sears, 1983). Furthermore, although prior research has examined regions associated with angry memories and faces, the current experiment represents a meaningful methodological departure from prior neuroimaging experiments in that previous studies of anger and aggression have relied on autobiographical episodic recall and angry faces as stimuli. By using a provocation that modeled a real-world anger-inducing situation, the current experiment provides a relatively high level of ecological validity, despite participants being in an MRI scanner.

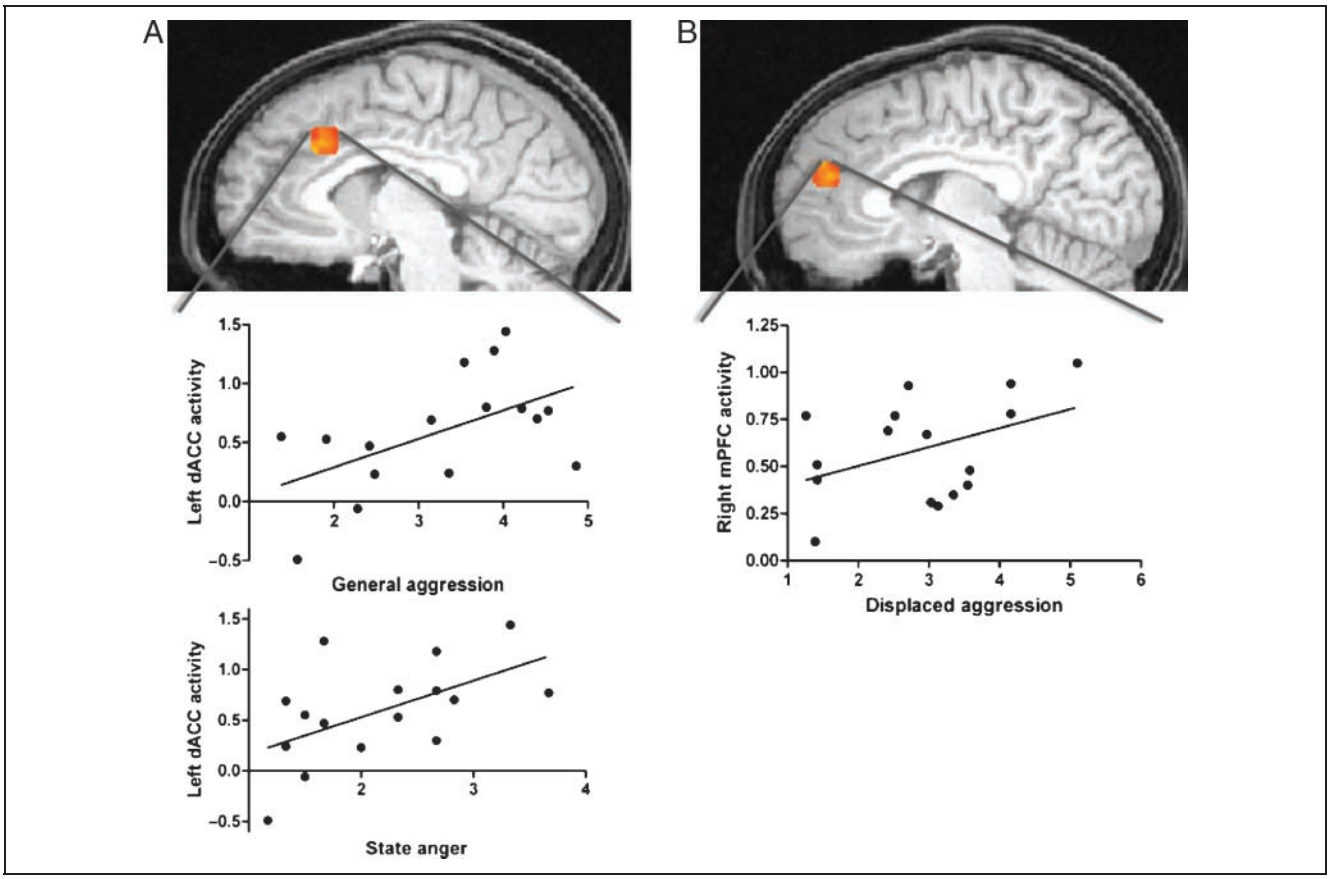

Figure 1. Brain activation following provocation. (A) Activity in the left dACC, which was positively associated with self-reported anger and individual differences in general aggression following the provocation. (B) Activity in the right mPFC, which was positively associated with individual differences in displaced aggression. The scatterplots below each panel depict these correlations. The *y*-axes represent BOLD responses, which are expressed in percent signal change relative to the baseline fixation.

Table 4. Brain Regions Active during Rumination Relative to Distraction

	Talairach Coordinates					
Region	*x*	*y*	*z*	*Cluster Size (Voxels)*	*Mean (SE) Percent Signal Change*	*Significance Test*
Dorsal Anterior Cingulate						
Right	7	15	35	401	0.71 (0.14)	$t(19) = 5.13, p < .001$
Left	−7	15	35	618	0.72 (0.11)	$t(19) = 6.39, p < .00001$
Rostral Anterior Cingulate						
Right	3	35	9	736	0.82 (0.15)	$t(19) = 5.67, p < .0001$
Left	−9	37	13	402	0.69 (0.13)	$t(19) = 5.20, p < .0001$
Insula						
Right	38	−3	7	740	0.68 (0.10)	$t(19) = 6.50, p < .00001$
Left	−38	−3	7	827	0.66 (0.11)	$t(19) = 6.27, p < .00001$
Posterior Cingulate						
$\text{Right}_{\text{region1}}$	6	−16	39	422	0.59 (0.10)	$t(19) = 5.79, p < .0001$
$\text{Right}_{\text{region2}}$	6	−53	25	377	0.60 (0.11)	$t(19) = 5.34, p < .0001$
$\text{Left}_{\text{region1}}$	−6	−15	35	333	0.64 (0.11)	$t(19) = 5.84, p < .0001$
$\text{Left}_{\text{region2}}$	−6	−58	22	488	0.57 (0.11)	$t(19) = 5.00, p < .0001$
Medial Frontal Gyrus						
Right	9	42	15	451	0.69 (0.12)	$t(19) = 6.02, p < .00001$
Left	−9	50	19	339	0.52 (0.13)	$t(19) = 3.89, p < .001$
Superior Frontal Gyrus						
Right	7	48	31	509	0.69 (0.14)	$t(19) = 5.01, p < .0001$
Left	−9	46	33	601	0.82 (0.11)	$t(19) = 7.81, p < .000001$
Precuneus						
Left	−9	−53	34	455	0.63 (0.10)	$t(19) = 6.20, p < .00001$
Lateral Middle Frontal Gyrus						
Right	36	45	15	684	0.74 (0.13)	$t(19) = 5.83, p < .0001$
Left	−37	46	16	368	1.31 (0.27)	$t(19) = 4.83, p < .001$
Lateral Inferior Frontal Gyrus						
Left	−50	23	15	330	0.75 (0.13)	$t(19) = 5.80, p < .0001$
Thalamus						
Left	−13	−21	11	160	0.60 (0.11)	$t(19) = 5.31, p < .0001$

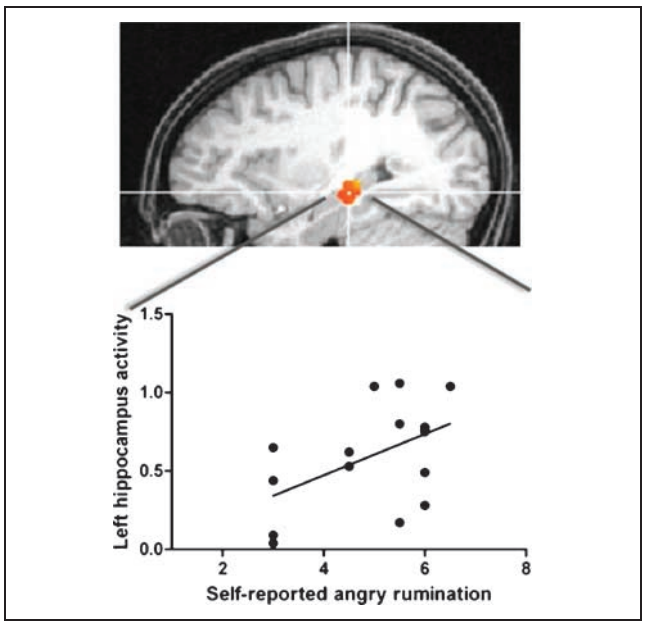

Figure 2. Hippocampal activation following provocation. Activity in the left hippocampus was correlated with the intensity of subsequent self-reported angry rumination during the rumination task. The *y*-axis represents BOLD responses, which are expressed in percent signal change relative to the baseline fixation.

Our data also illustrate the neural regions underlying angry rumination. Activity during angry rumination was apparent in regions associated with the intensity of negative affect as well as ''top–down'' emotion regulation regions such as the lateral PFC and the mPFC. Indeed, the mPFC appears to be associated with the *awareness* and *regulation* of one's negative mood (Macrae et al., 2004; Ochsner et al., 2004; Lane et al., 1997). Broadly relevant to the current study, the mPFC is active during impression formation (Mason & Macrae, 2004) and making attributions and determining the mental states of others (Harris, Todorov, & Fiske, 2005), both of which can occur during rumination. Thus, the current findings are highly consistent with research examining rumination as a multifaceted emotion regulation strategy that maintains or increases negative affect (Denson et al., 2006; Bushman et al., 2005; Ray et al., 2005; Miller, Pedersen, Earleywine, & Pollock, 2003).

We also identified brain regions associated with the subjective experience of rumination. Notably, increased encoding of the provocation in memory as assessed by hippocampus activation predicted subsequent rumination. However, the mechanism whereby this occurs remains unclear. Presumably, increased encoding leads to greater accessibility in memory, yet it is also likely that greater encoding may have occurred due to bewilderment regarding the unexpected and unjustified nature of the provocation. If true, then increased rumination could be a result of cognitively incorporating the provocation into existing knowledge structures or mentally attempting to resolve the conflict. Consistent with this latter view, the hippocampal activity observed in the present research can be interpreted in light of Gray and McNaughton's (2000) theorizing that the hippocampus is involved in comparing discrepancies between expected and actual events. They also propose that this hippocampal comparator mechanism is highly sensitive to signals of punishment, which is relevant to the provocation used in the present research. Furthermore, hippocampal activity is posited to result in behaviors designed to solve the problem that produced the discrepancy. In the context of the present study, rumination may have been just such an attempt at mentally resolving interpersonal conflict.

The final and perhaps most intriguing contribution of our findings is that they suggest a neural basis for differences in aggressive behavior, such that within seconds of being insulted, differences emerged in the degree of activity associated with the dACC and the mPFC as a function of aggressive personality. Individual differences in general aggression were more strongly correlated with exacerbated activity in a region associated with the intensity of anger, pain, social distress, and cognitive conflict monitoring (i.e., dACC), whereas individual differences in displaced aggression were more strongly correlated with a region associated with self-relevant cognition, the self-awareness of emotions, and emotion regulation (i.e., the mPFC). It is highly likely that activity in these regions is at least partially responsible for the observed differences in subsequent cognition, affect, and behavior among those high in general and displaced aggression (Bettencourt et al., 2006; Denson et al., 2006; Buss & Perry, 1992).

One unexpected finding was that activity in the right dACC was correlated with the Guilt subscale of the PANAS-X. In hindsight, these results appear sensible given our manipulation, whereby a high status experimenter communicated (albeit rudely) that the participant was not speaking loud enough. Thus, some participants may have inferred that their actions were invalidating a quite important and expensive experiment. However, we do not believe that this measure truly assesses feelings of guilt as the subscale name implies for two reasons. First, the subscale combines items assessing both shame and guilt despite evidence that they are distinct emotions (e.g., Tangney, 2002). Second, and more importantly, a post hoc analysis of the six individual items in the Guilt subscale revealed that the individual items ''guilty'' and ''ashamed'' were not significantly correlated with dACC activity (right or left). However, three of the individual items from the PANAS-X Guilt subscale demonstrated strong associations with the right dACC. These were ''dissatisfied with self'' ($r = .70$, $p < .01$), ''disgusted with self'' ($r = .46, p = .07$), and ''blameworthy'' ($r = .62$, $p = .01$). In a post hoc analysis, we created an overall ''social distress'' composite by averaging these three items ($\alpha = .80$). Consistent with research demonstrating associations between feelings of social distress and the

dACC in the context of social rejection manipulations (e.g., being left out of a ball-tossing game; Eisenberger et al., 2003, 2007), this social distress variable was correlated with activity in the right (but not left) dACC following the provocation ($r = .72, p = .002$). These results are consistent with the notion that, in addition to anger, the provocation may have elicited feelings of social devaluation and self-reproach rather than true guilt or shame.

There were a number of limitations associated with the current study. Due to practical considerations, the directed rumination task was conducted within participants rather than between participants. Although our significant results suggest otherwise, this might have resulted in carryover effects whereby some individuals were unable to stop ruminating about the provocation even in the distraction condition. Another shortcoming of the current experiment was the temporal placement of the state mood measures that assessed the reaction to the provocation at the end of the experiment. Obtaining self-report data after removing participants from the scanner rather than immediately following the provocation might have introduced retrospective memory biases. However, this was done because we wanted to assess a wide variety of emotional reactions in the current study. To do so would have required an excessive burden on participants if they were asked to rate their mood state on all 65 items in the scanner. We also felt that an earlier positioning of these measures would arouse suspicion by inquiring about the participants' mood immediately following the provocation. Another unresolved issue in the present research is the nature of the functional connections between brain regions. Due to the temporal limitations of fMRI methods, we were unable to specify a temporal pathway for the processes underlying anger and aggression. Future research remains to explore the temporal pattern of activation in response to provocation. Finally, our small sample size limited statistical power. Although small sample sizes are common in neuroimaging research, when examining individual differences and correlations with behavioral data, larger samples are desired in order to detect smaller, yet meaningful, differences. Indeed, most effect sizes in psychology are in the small-to-moderate range (e.g., $r < .30$; Hemphill, 2003). Thus, when it is not possible to increase sample size, it is crucial that researchers select highly reliable and valid instruments when mixing self-report methods with neuroimaging in order to reduce measurement error. Despite these remaining issues, it is our hope that the data presented here may eventually help eliminate the harm associated with anger, angry rumination, and aggressive personality.

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Reprint requests should be sent to Thomas F. Denson, School of Psychology, University of New South Wales, Sydney, NSW 2052, Australia, or via e-mail: t.denson@unsw.edu.au.

Notes

1. The amygdala was not associated with anger.
2. Relative to self-focused rumination, provocation-focused rumination elicited slightly more activity in the right middle frontal gyrus [$M = 0.042$, $SE = 0.012$, $t(19) = 3.58$, $p = .002$], the right PCC [$M = 0.036$, $SE = 0.008$, $t(19) = 4.35$, $p < .001$], and the left precuneus [$M = 0.027$, $SE = 0.007$, $t(19) = 3.73$, $p = .001$. These results suggest that, following a provocation, both types of rumination recruit highly similar neural substrates relative to distraction, even though the content of the two types of rumination may differ.
3. Because of an imaging protocol adjustment, provocation data from the first four participants were removed from analyses.

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