

Coping with Emotions Past: The Neural Bases of Regulating Affect Associated with Negative Autobiographical Memories

Ethan Kross, Matthew Davidson, Jochen Weber, and Kevin Ochsner

Background: Although the ability to adaptively reflect on negative autobiographical experiences without ruminating is critical to mental health, to our knowledge no research has directly examined the neural systems underlying this process.

Methods: Sixteen participants were scanned using functional magnetic resonance imaging (fMRI) as they focused on negative autobiographical memories using cognitive strategies designed to facilitate (feel strategy) versus undermine (analyze and accept strategies) rumination.

Results: Two key findings were obtained. First, consistent with prior emotion regulation research using image-based stimuli, left prefrontal activity was observed during the implementation of all three strategies. Second, activity in a network of regions involved in self-referential processing and emotion, including subgenual anterior cingulate cortex and medial prefrontal cortex, was highest in response to the feel strategy and lowest for the accept strategy. This pattern of activation mirrored participants' self-reports of negative affect when engaging in each strategy.

Conclusions: These findings shed light on the brain regions that distinguish adaptive versus maladaptive forms of reflecting on negative autobiographical memories and offer a novel, ecologically valid route to exploring the neural bases of emotion regulation using fMRI.

Key Words: Autobiographical memory, emotion regulation, fMRI, reappraisal, rumination, subgenual anterior cingulate cortex

The ability to adaptively cope with distressing life experiences is a key self-regulatory challenge. Failing to meet this challenge can be costly, as intrusive and emotionally charged thoughts about these experiences contribute to a variety of clinical disorders (1). Although an explosion of research has examined the neural bases of consciously regulating negative emotions triggered in response to normatively aversive visual or cutaneous shock stimuli (2–19), no research has examined how these findings generalize to coping with such highly idiosyncratic negative emotional memories. This is important because some regions known to be critical to mood disorders have not been consistently identified in prior neuroimaging research on the use of cognitive strategies to regulate emotion. Consider, for example, research on depression, a mood disorder characterized by high levels of self-focused rumination (20,21). Although findings clearly indicate that subgenual anterior cingulate cortex (sgACC) activity tracks closely with depressive symptoms (22–30), research on emotion regulation strategies thought to be relevant to cognitive therapies for depression (e.g., reappraisal) rarely report changes in activity in this region. This discrepancy suggests that some of the brain regions involved in regulating feelings associated with emotional memories may be different than those involved in regulating responses to normatively negative stimuli.

Here, we examined this issue by developing a novel functional magnetic resonance imaging (fMRI) paradigm in which participants recalled a series of highly arousing negative auto-

biographical memories and then focused on them using strategies designed to facilitate versus undermine adaptive self-reflection. The first “feel” strategy directed individuals to focus on the specific feelings that naturally flowed through their mind as they thought about their recalled experiences. This strategy was used because prior research indicates that focusing concretely on negative feelings triggers the kind of negative affect infused, ruminative episodes that are the hallmark of dysfunctional coping (31–33). The second “accept” strategy directed individuals to recognize that the feelings they experienced during recollection were passing mental events that were psychologically distant from the self and did not control them. The instructions for this strategy were adapted from a form of cognitive behavioral therapy that teaches people how to mindfully focus on negative feeling states in ways that are believed to buffer against rumination (34–36). The third “analyze” strategy directed participants to objectively analyze the causes and reasons underlying their feelings and was designed as a memory analog of cognitive reappraisal strategies used in prior fMRI studies (12,13,18,19).

Methods and Materials

Twenty-four Columbia University affiliates (15 female subjects; M age = 20.83, SD = 3.27) provided informed consent. Prospective participants were screened to ensure they were not currently undergoing treatment from a mental health professional, taking mental health-related medication (e.g., Prozac), were claustrophobic, or had metal in their bodies. The sample consisted of 60% European Americans, 24% Asians, 4% African Americans, and 12% other.

Stimuli

Similar to prior studies that have used script-driven methods, cue phrases were used to trigger the recall of negative autobiographical memories in the scanner. To obtain memory cues, participants were asked to describe in writing nine highly arousing negative autobiographical experiences during a screening session and then judge the extent to which thinking about

From the Department of Psychology (EK), University of Michigan, Ann Arbor, Michigan; and Psychology Department (MD, JW, KO), Columbia University, New York, New York.

Address reprint requests to Ethan Kross, Ph.D., University of Michigan, Department of Psychology, 530 Church Street, Ann Arbor, MI 48109-1043; E-mail: ekross@umich.edu.

Received July 10, 2008; revised October 6, 2008; accepted October 13, 2008.

Table 1. Conjunction Analysis Identifying Regions Commonly Active in the Feel, Analyze, and Accept Strategy Conditions Relative to an Active Baseline Task

Region of Activation	Brodmann's Area	<i>t</i>	TAL Coordinates			Voxels
			<i>x</i>	<i>y</i>	<i>z</i>	
Inferior Frontal Gyrus	46	4.46	-51	29	16	218
Cuneus	19	4.46	-12	-88	31	94
Cerebellum		4.11	24	-70	-38	29
Middle Occipital Gyrus	18	3.69	15	-91	10	17
Lingual Gyrus	18	3.89	-6	-73	-2	16

TAL, Talairach.

each memory made them feel aroused ($M = 6.85$; $SD = .65$) and negative ($M = 6.94$; $SD = .55$) using a 7-point scale in which higher numbers corresponded to higher levels of arousal and negativity. Paired sample *t* tests comparing valence and arousal ratings for all memories revealed no significant differences (arousal: $t_s < 1.87$, $p_s > .08$; valence: $t_s < 1.07$, $p_s > .30$).

Training

Upon arrival at the fMRI scanner, participants were reminded of the negative autobiographical memories they generated during the screening session and taught how to quickly recall each memory in response to specific cue words using a computerized protocol. In the first part of the protocol, a cue phrase appeared on screen along with a description of the memory to which it corresponded. Participants were given as much time as they needed to pair the cue and memory so that they would be able to quickly recall each memory when presented with the cue alone. This process repeated until participants saw a cue-memory description pairing for all nine memories. During the

second phase of the protocol, each cue was randomly presented on screen and participants were instructed to press the space bar as soon as they were able to recall the specific negative autobiographical experience it corresponded to. Reaction time data were examined to ensure that participants recalled each memory in less than 10 seconds (i.e., the amount of time participants had to recall their experiences during the experiment). Subsequently, participants received instructions regarding how to implement each strategy during scanning (see introduction for summary of specific strategy instructions).

fMRI Task

Participants viewed three repetitions each of three types of stimulus blocks (feel, accept, or analyze) whose order was counterbalanced. Each block was comprised of three 80-second trials. All trials began with a 10-second cue phrase indicating that participants should recall the autobiographical memory indicated by the cue. Subsequently, a strategy cue word appeared on screen directing them to engage in the feel, accept, or analyze strategy for 30 seconds. Next, participants indicated how aroused and negative they felt using a 5-point scale (1 = not at all aroused/negative; 5 = very aroused/negative). Each question appeared on screen for 5 seconds. Finally, participants engaged in a 30-second spatial perception task in which they saw an arrow pointing left or right and were asked to indicate which direction the arrow was pointing. This task was used as a baseline condition because pilot testing indicated that when participants were asked to recollect memories naturally, they tended to spontaneously engage in the strategies. Therefore, we sought an active baseline task that would not engage the regulatory, memory, and emotional processes of interest, and prior work suggests that this arrows task does not engage these

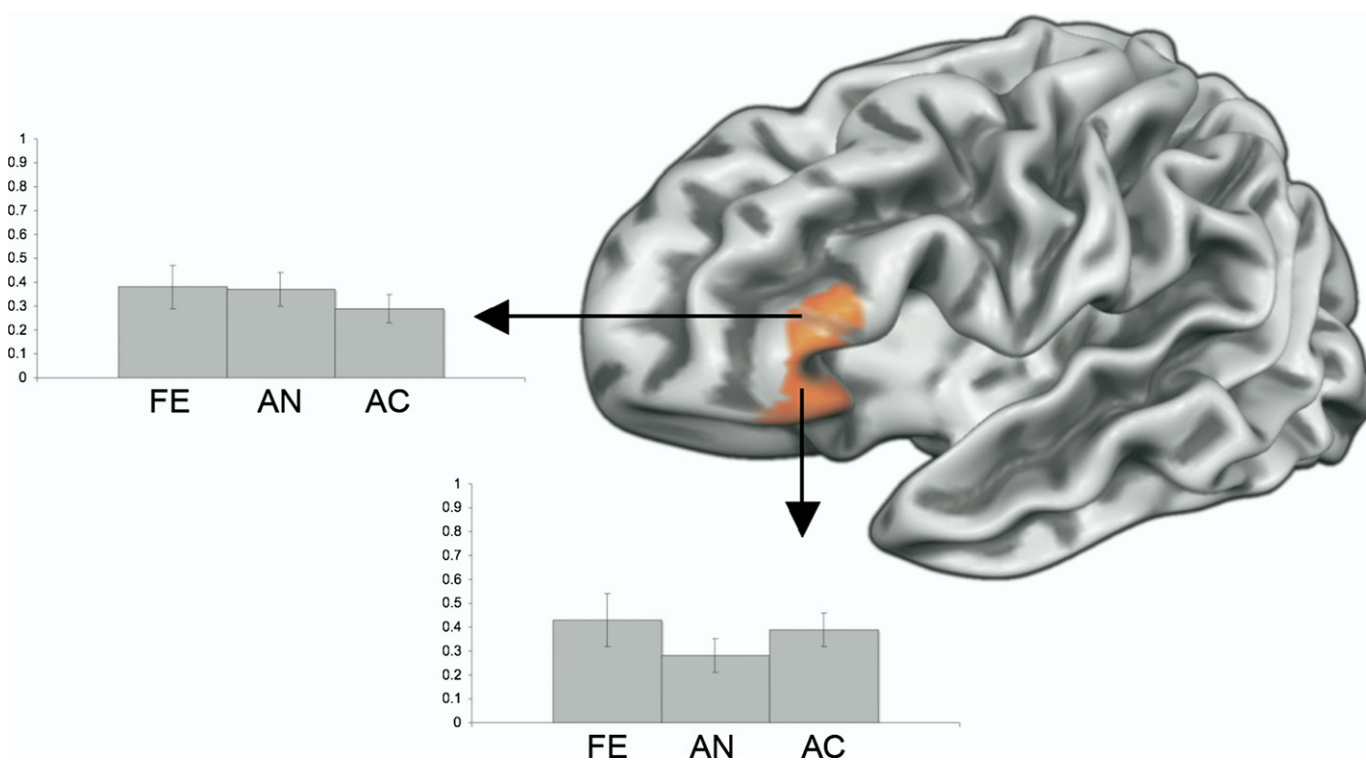
**Figure 1.** Regions of IPFC active for each strategy relative to baseline. Bar graphs illustrate parameter estimates of signal intensity for each strategy versus baseline. Error bars represent SEM. AC, accept; AN, analyze; FE, feel; IPFC, left prefrontal cortex.

Table 2. Activations Revealed from a Within-Subjects ANOVA Examining the Effect of Strategy (Feel vs. Accept vs. Analyze)

Region of Activation	Brodmann's Area	t	TAL Coordinates			Voxels
			x	y	z	
Anterior Cingulate	25	11.67	0	14	-5	55
Medial Frontal Gyrus	10	13.37	6	59	7	49
Cerebellum		8.82	0	-64	-5	56
Cerebellum/Lingual Gyrus	19	12.26	-12	-43	-2	47
Middle Temporal Gyrus	39	10.06	45	-70	25	38
Superior Frontal Gyrus	6	10.47	24	11	49	19
Cuneus	17	8.61	-15	-79	13	17
Cuneus	18	12.89	0	-97	7	12
Inferior Frontal Gyrus	47	8.45	30	23	-17	12

ANOVA, analysis of variance; TAL, Talairach.

processes (37). Postscan debriefings indicated that three participants did not follow instructions. Their data were excluded from subsequent analyses.

fMRI Acquisition and Analysis

Whole-brain functional data were acquired on a GE 1.5 T scanner (General Electric, Milwaukee, Wisconsin) in 24 contiguous axial slices (4.5 mm thick, 1.5 × 1.5 mm in-plane resolution) parallel to the anterior commissure-posterior commissure (AC-PC) line with a T2*-weighted echo-planar imaging (EPI) sequence (repetition time [TR] = 2000, echo time [TE] = 40, flip angle = 60, field of view [FOV] = 22) in three runs of 124 volumes each (248 sec). Structural data were acquired with a T1-weighted spoiled gradient recalled echo (SPGR) scan (124 slices, 1.5 mm thick, in-plane resolution .86 × .86 mm; TR = 19, TE = 5, flip angle = 20, FOV = 220).

Functional scans were slice time and motion corrected using Oxford Centre for Functional MRI of the Brain (FMRIB; Oxford, United Kingdom) Software Library (FSL) tools slicetimer and MCFLIRT and were normalized and smoothed with a Gaussian kernel of 8 mm full-width at half maximum (FWHM) using SPM2 (Wellcome Department of Cognitive Neurology, University College London, London, United Kingdom). Statistical analyses

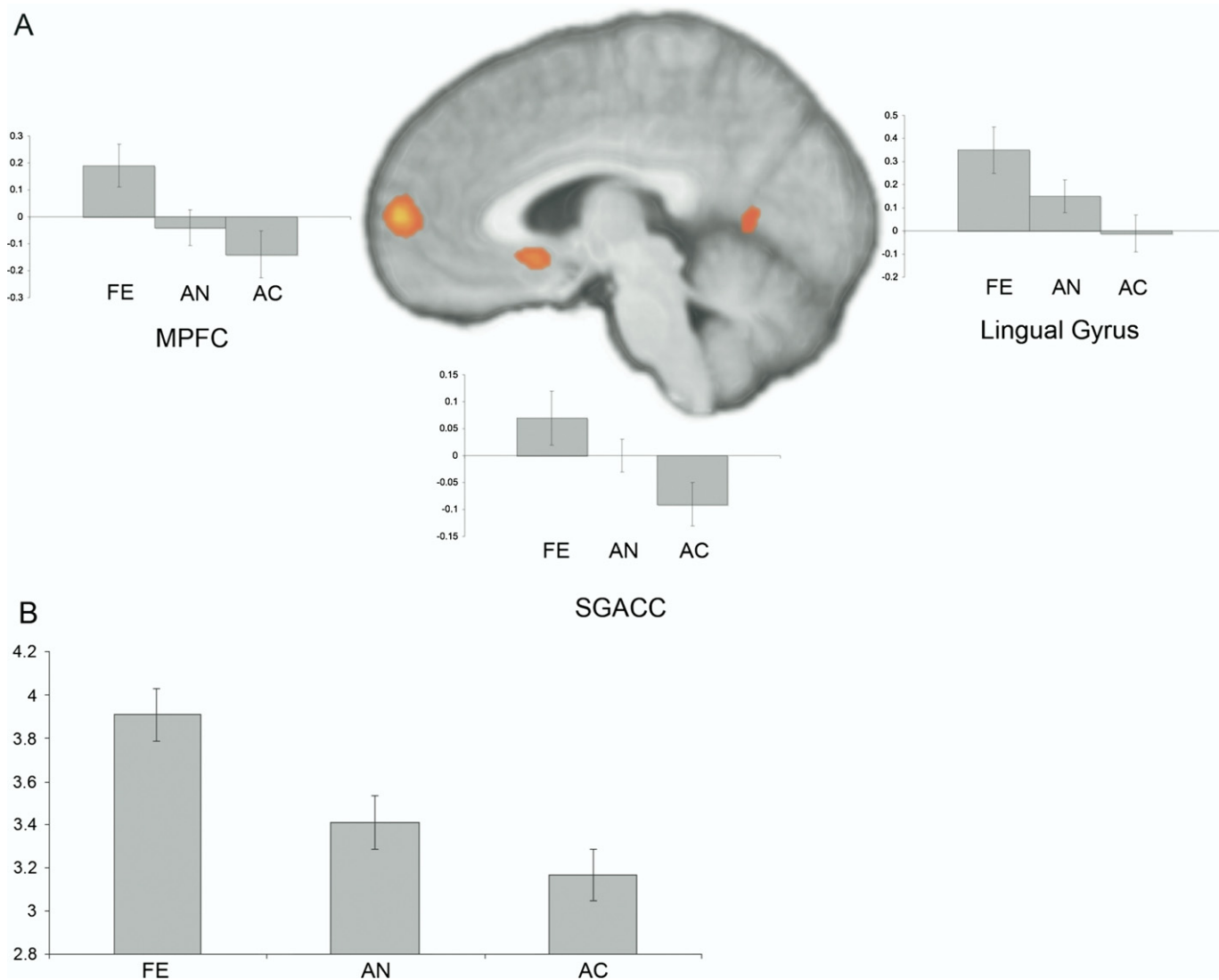


Figure 2. Effects of strategy condition on brain activity and negative affect. **(A)** Regions of mPFC and sgACC displaying a significant linear effect of strategy condition. Bar graphs illustrate parameter estimates of signal intensity for each strategy versus baseline. **(B)** Self-report negative affect as a function of strategy condition. Error bars represent SEM. AC, accept; AN, analyze; FE, feel; mPFC, medial prefrontal cortex; sgACC, subgenual anterior cingulate cortex.

Table 3. Regions Showing a Positive Correlation Between Increases in Self-Reported Negative Affect and Increases in Brain Activity

Region of Activation	Brodmann's Area	t	TAL Coordinates			Voxels
			x	y	z	
Feel > Accept						
Middle Frontal Gyrus	10	5.30	-21	56	19	36
Anterior Cingulate/ Rectal Gyrus	25/11	4.54	-9	14	-17	25
Inferior Frontal Gyrus	47	3.46	-39	23	-17	12
Feel > Analyze						
None						
Analyze > Accept						
Insula	13	4.07	-39	-16	-5	12
TAL, Talairach.						

were conducted using the general linear model (GLM) framework implemented in Brain Voyager (Maastricht, The Netherlands). Boxcar regressors, convolved with the canonical hemodynamic response function (HRF), modeled periods for the 10-sec recall epoch and 30-sec strategy epoch. The arrow task epoch was used as the baseline. Voxelwise statistical parametric maps (SPM) summarizing differences between trial types were calculated for each subject and then entered into random effects group analyses with statistical maps thresholded at $p < .005$ uncorrected for multiple comparisons, with an extent threshold of 12 voxels. These parameters were chosen because they corresponded to an overall alpha level of $p < .05$ corrected for multiple comparisons as calculated by the Monte Carlo simulation method implemented in Analysis of Functional Neuroimages (<http://afni.nimh.nih.gov/afni>), which is widely used in fMRI research (5,15–17,38–40). This technique controls for the familywise error rate (FWE) by simulating null data sets with the same spatial autocorrelation found in the residual images and creates a frequency distribution of different cluster sizes. Clusters larger than the minimum size corresponding to the a priori chosen FWE are then retained for additional analysis. This technique offers an alternative to simple FWE-only voxel-based correction. Preliminary analyses indicated that participants' valence and arousal ratings were highly correlated ($r = .68$, $p < .001$). They were therefore averaged to form a single index of negative affect that was used for subsequent analyses. Data from three participants were excluded because of technical difficulties. In addition, 3 participants were excluded for excessive motion, leaving a total of 16 participants.

Results

We first examined regions commonly active across all three strategies by performing a conjunction analysis on regions active for each strategy versus the baseline task. This analysis revealed increased activity in occipital regions implicated in visualizing recollected events as well as left lateral prefrontal regions previously implicated in studies of cognitive reappraisal using visual stimuli (Table 1; Figure 1).

Next, we used an analysis of variance (ANOVA) with strategy as the within-subjects factor to examine regions whose activity was modulated by the strategy participants implemented. This analysis revealed activations in regions involved in self-referential processing, emotion, and autobiographical memory recall, including right rostral medial and ventrolateral prefrontal cortex (PFC), cuneus, and most notably sgACC (Table 2; Figure 2A). To

determine which strategies drove these activations, we extracted beta values for each condition from each cluster. These analyses revealed highly significant linear effects in 8 of 9 clusters, with activations in each cluster greatest for the feel strategy followed by the analyze strategy and then the accept strategy (F s for all linear effects > 17.08 , all p s $\leq .001$). This pattern directly mirrored participants' self-reported negative affect ratings, which revealed the same highly significant linear relationship [$F(1,15) = 24.12$, $p < .001$; Figure 2B]. The only cluster that did not display this pattern was a small region of activity in right ventrolateral prefrontal cortex (rVLPFC). Consistent with the activations observed in the other clusters, the feel strategy led to significantly more activity in this region than the other two strategies. However, analyze led to lower levels of activation compared with accept, although this difference was not significant ($p = .26$; for strategy vs. strategy comparisons see Table 1 in Supplement 1).

To more directly examine the relationship between neural activity and self-reported negative affect, we next performed a series of simple regression analyses that examined whether self-reported increases in negative affect across strategy conditions (e.g., feel - accept affect difference score; feel - analyze affect difference score; analyze - accept affect difference score) correlated with brain activity identified by the corresponding strategy versus strategy contrast (Supplement 1). We first compared activity on accept and feel trials because these were the conditions that were maximally different on both self-reported negative affect and neural activity identified in the ANOVA described above. These analyses revealed significant positive associations between increases in self-reported negative affect and activity in sgACC and medial PFC (Table 3). Decreases in negative affect on accept versus feel trials were significantly positively correlated with activity in caudate, superior parietal lobule, and medial frontal gyrus, suggesting that these regions were involved in downregulating participants' affective responses (Table 4). The only region correlating with increased levels of negative affect on analyze versus accept trials was the insula (Table 4). No regions correlated with decreases in negative affect on accept versus analyze trials or with increases or decreases in negative affect on feel versus analyze and analyze versus feel trials, respectively.

Discussion

To our knowledge, this study is the first to directly examine the neural systems underlying the ability to regulate emotion by

Table 4. Regions Showing a Positive Correlation Between Decreases in Self-Reported Negative Affect and Increases in Brain Activity

Region of Activation	Brodmann's Area	t	TAL Coordinates			Voxels
			x	y	z	
Accept > Feel						
Caudate		5.08	18	11	22	37
Medial Frontal Gyrus	4	3.93	18	-22	58	23
Superior Parietal Lobule	7	3.84	21	-49	58	14
Analyze > Feel						
None						
Accept > Analyze						
None						
TAL, Talairach.						

adaptively reflecting on negative autobiographical experiences without ruminating. Two key findings were obtained.

First, consistent with prior research using image-based stimuli (2–19,41), left PFC was observed during the implementation of all three strategies. In the context of prior work, this suggests that left PFC implements reappraisal operations regardless of stimulus type or the specific type of cognitive strategy one employs.

Second, activity in a network of regions involved in self-referential processing, autobiographical memory recall, and emotion—including sgACC and medial prefrontal cortex (mPFC)—was highest in response to the feel strategy and lowest for the accept strategy, and this pattern of activation mirrored participants' self-reports of negative affect. Moreover, activity in these regions correlated positively with increases in negative affect on feel versus accept trials, indicating that they were directly related to participants' subjective emotional responses.

These findings have important implications for understanding why depressed individuals, who are known to ruminate (19–20), show activity at rest in a similar set of regions, including sgACC (24,42). They suggest that depressed individuals may spontaneously engage in the same type of self-focused rumination triggered by the feel strategy. Importantly, the present findings demonstrate that this activity can be brought under their cognitive control when the appropriate type of self-regulatory strategy is implemented. More broadly, these findings provide neural corroboration for behavioral research showing that focusing concretely on negative feelings facilitates rumination, whereas focusing on negative feelings as mental events that are psychologically distanced from the self undermines it (31–33).

These findings also suggest that attempts to objectively analyze one's feelings may be a less successful form of reducing negative affect than acceptance when memories are the source of negative feelings rather than standardized visual stimuli. In this vein, it is noteworthy that although both the accept and analyze strategies led to significant group average drops in self-reported affect relative to the feel strategy, these drops correlated with changes in sgACC and mPFC activity only for the feel versus accept contrast. The failure to observe a similar correlation for the analyze versus feel contrast could be attributable to the smaller overall magnitude of regulatory success and the relatively restricted variability in this comparison (Figure 2). However, it may also have to do with the kind of variability: 4 of 16 participants displayed no reduction in negative affect in the analyze versus feel contrast. By comparison, only two participants did not show a drop in negative affect in the accept versus feel contrast. To explore this issue, we reran the correlation analyses leaving out the four nonregulators on the analyze versus feel contrast. This analysis revealed significant positive correlations between increases in self-reported affect and mPFC ($x = 6$, $y = 56$, $z = 1$; $r = .80$, $p < .001$) and sgACC ($x = -1$, $y = 5$, $z = -6$; $r = .60$, $p = .02$) activity. Although exploratory, when considered in the context of the similar correlations found for the feel versus accept comparison, this finding is consistent with the idea that successful regulation diminishes activity in these regions.

Conclusion

The present results raise a number of new questions for future research. To what extent do the neural processes involved in recalling negative autobiographical memories differ from those involved in reflecting on them to enhance or diminish emotional responses? And most important for translational research, how do clinical versus normal populations differ in their ability to

adaptively implement regulatory strategies designed to reduce emotions generated in response to thinking about highly personal emotional memories? Given the pervasive role that thinking about such experiences plays in eliciting distress and dysfunction in everyday life, a key need for future research is to address these questions to refine our understanding of the neural systems that characterize these different forms of self-reflection and their emotional consequences.

Completion of this research was supported by National Institutes of Health (NIH) Grant MH076137.

We thank Nick Weiler for helping with the administration of this experiment and Tor Wager for methodological feedback.

The authors reported no biomedical financial interests or potential conflicts of interest.

Supplementary material cited in this article is available online.

1. American Psychiatric Association (1994): *Diagnostic and Statistical Manual, 4th ed.* Washington, DC: American Psychiatric Press.
2. Ochsner KN, Gross JJ (2008): Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Curr Dir Psychol Sci* 17:153–158.
3. Beauregard M, Levesque J, Bourgouin P (2001): Neural correlates of conscious self-regulation of emotion. *J Neurosci* 21:RC165.
4. Eippert F, Veit R, Weiskopf N, Erb M, Birbaumer N, Anders S (2006): Regulation of emotional responses elicited by threat-related stimuli. *Hum Brain Mapp* 28:409–423.
5. Goldin PR, McRae K, Ramel W, Gross JJ (2008): The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biol Psychiatry* 63:577–586.
6. Harenski CL, Hamann S (2006): Neural correlates of regulating negative emotions related to moral violations. *Neuroimage* 30:313–324.
7. Kalisch R, Wiech K, Critchley HD, Seymour B, O'Doherty JP, Oakley DA, et al. (2005): Anxiety reduction through detachment: Subjective, physiological, and neural effects. *J Cogn Neurosci* 17:874–883.
8. Kalisch R, Wiech K, Herrmann K, Dolan RJ (2006): Neural correlates of self-distraction from anxiety and a process model of cognitive emotion regulation. *J Cogn Neurosci* 18:1266–1276.
9. Kim SH, Hamann S (2007): Neural correlates of positive and negative emotion regulation. *J Cogn Neurosci* 19:776–798.
10. Levesque J, Eugene F, Joannette Y, Paquette V, Mensour B, Beaudoin G, et al. (2003): Neural circuitry underlying voluntary suppression of sadness. *Biol Psychiatry* 53:502–510.
11. Levesque J, Joannette Y, Mensour B, Beaudoin G, Leroux JM, Bourgouin P, et al. (2004): Neural basis of emotional self-regulation in childhood. *Neuroscience* 129:361–369.
12. Ochsner KN, Bunge SA, Gross JJ, Gabrieli JD (2002): Re-thinking feelings: An fMRI study of the cognitive regulation of emotion. *J Cogn Neurosci* 14:1215–1229.
13. Ochsner KN, Ray RD, Cooper JC, Robertson ER, Chopra S, Gabrieli JDE, et al. (2004): For better or for worse: Neural systems supporting the cognitive down- and up-regulation of negative emotion. *Neuroimage* 23:483–499.
14. Phan KL, Fitzgerald DA, Nathan PJ, Moore GJ, Uhde TW, Tancer ME (2005): Neural substrates for voluntary suppression of negative affect: A functional magnetic resonance imaging study. *Biol Psychiatry* 57:210–219.
15. Schaefer SM, Jackson DC, Davidson RJ, Aguirre GK, Kimberg DY, Thompson-Schill SL (2002): Modulation of amygdalar activity by the conscious regulation of negative emotion. *J Cogn Neurosci* 14:913–921.
16. Urry HL, van Reekum CM, Johnstone T, Kalin NH, Thurow ME, Schaefer HS, et al. (2006): Amygdala and ventromedial prefrontal cortex are inversely coupled during regulation of negative affect and predict the diurnal pattern of cortisol secretion among older adults. *J Neurosci* 26:4415–4425.
17. van Reekum CM, Johnstone T, Urry HL, Thurow ME, Schaefer HS, Alexander AL, Davidson RJ (2007): Gaze fixation predicts brain activation during the voluntary regulation of picture-induced negative affect. *Neuroimage* 36:1041–1055.

18. Wager TD, Davidson M, Hughes BL, Lindquist MA, Ochsner KN (2008): Prefrontal-subcortical pathways mediating successful emotion regulation. *Neuron* 59:1037–1050.
19. Ray RD, Ochsner KN, Cooper JC, Robertson ER, Gabrieli JD, Gross JJ (2005): Individual differences in trait rumination and the neural systems supporting cognitive reappraisal. *Cogn Affect Behav Neurosci* 5:156–168.
20. Nolen-Hoeksema S (2000): The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *J Abnorm Psychol* 109:504–511.
21. Nolen-Hoeksema S, Wisco BE, Lyubomirsky S (2008): Rethinking rumination. *Perspect Psychol Sci* 3:400–424.
22. Liotti M, Mayberg HS, Brannan SK, McGinnis S, Jerabeck P, Fox PT (2000): Differential limbic-cortical correlates of sadness and anxiety in healthy subjects: Implications for affective disorders. *Biol Psychiatry* 48:30–42.
23. Siegle GJ, Cameron CS, Thase ME (2006): Use of fMRI to predict recovery from unipolar depression with cognitive behavior therapy. *Am J Psychiatry* 163:735–738.
24. Drevets WC, Bogers W, Raichle ME (2002): Functional anatomical correlates of antidepressant drug treatment assessed using PET measures of regional glucose metabolism. *Eur Neuropsychopharmacol* 12:527–544.
25. Osuch EA, Ketter T, Kimbrell T, George M, Benson B, Herscovitch M, Post RM (2000): Regional cerebral metabolism associated with anxiety symptoms in affective disorder patients. *Biol Psychiatry* 48:1020–1023.
26. Mayberg HS, Lozano A, Voon V, Mcneely H, Seminowicz D, Hamani C, *et al.* (2005): Deep brain stimulation for treatment-resistant depression. *Neuron* 45:651–660.
27. Ressler K, Mayberg HS (2007): Targeting abnormal neural circuits in mood and anxiety disorders: From the laboratory to the clinic. *Nat Neurosci* 10:1116–1124.
28. Kennedy SH, Konarski JZ, Segal ZV, Lau MA, Bieling PJ, McIntyre RS, Mayberg HS (2007): Differences in brain glucose metabolism between responders to CBT and venlafaxine in a 16-week randomized controlled trial. *Am J Psychiatry* 164:778–788.
29. Siegle GJ, Carter CS, Thase ME (2006): Use of fMRI to predict recovery from unipolar depression with cognitive behavior therapy. *Am J Psychiatry* 163:735–738.
30. Farb NAS, Segal ZV, Mayberg H, Bean J, McKeon D, Fatima Z, Anderson AK (2007): Attending to the present: Mindfulness meditation reveals distinct neural modes of self-reference. *Soc Cogn Affect Neurosci* 2:313–322.
31. Kross E, Ayduk O, Mischel W (2005): When asking “why” does not hurt: Distinguishing rumination from reflective processing of negative emotions. *Psychol Sci* 16:709–715.
32. Ayduk O, Kross E (2008): Enhancing the pace of recovery: Self-distanced analysis of negative experiences reduces blood pressure reactivity. *Psychol Sci* 19:229–231.
33. Kross E, Ayduk O (2008): Facilitating adaptive emotional analysis: Distinguishing distanced-analysis of depressive experiences from immersed-analysis and distraction. *Pers Soc Psychol Bull* 34:924–938.
34. Teasdale J, Segal Z, Williams J, Ridgeway V, Soulsby J, Lau M (2000): Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *J Consult Clin Psychol* 68:615–623.
35. Hayes SC, Strosahl K, Wilson KG (1999): *Acceptance and Commitment Therapy: An Experimental Approach to Behavior Change*. New York: Guilford Press.
36. Linehan M (1993): *Skills Training Manual for Treating Borderline Personality Disorder*. New York: Guilford Press.
37. Stark CEL, Squire LR (2001): When zero is not zero: The problem of ambiguous baseline conditions in fMRI. *Proc Natl Acad Sci U S A* 98:12760–12766.
38. Forman SD, Cohen JD, Fitzgerald M, Eddy WF, Mintun MA, Noll DC (1995): Improved assessment of significant activation in functional magnetic resonance imaging (fMRI): Use of a cluster-size threshold. *Magn Reson Med* 33:636–647.
39. Konishi S, Nakajima K, Uchida I, Kikyo H, Kameyama M, Miyashita Y (1999): Common inhibitory mechanism in human inferior prefrontal cortex revealed by event-related functional MRI. *Brain* 122:981–991.
40. Wood JN, Romero SG, Makale M, Grafman J (2003): Category-specific representations of social and nonsocial knowledge in the human prefrontal cortex. *J Cogn Neurosci* 15:236–248.
41. Ochsner KN, Gross JJ (2007): Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Curr Dir Psychol Sci* 17:153–158.
42. Greicius MD, Flores BH, Menon V, Glover GH, Solvason HB, Kenna H, *et al.* (2007): Resting-state functional connectivity in major depression: Abnormally increased contributions from subgenual cingulate cortex and thalamus. *Biol Psychiatry* 62:429–437.