Journal of Abnormal Psychology
2012, Vol. 121, No. 1, 61–72

0021-843X/11/$12.00 DOI: 10.1037/a0025294

Neural Correlates of Automatic Mood Regulation in Girls at High Risk for Depression

Jutta Joormann
University of Miami

Rebecca E. Cooney, Melissa L. Henry, and Ian H. Gotlib
Stanford University

Daughters of depressed mothers are at significantly elevated risk for developing a depressive disorder themselves. We have little understanding, however, of the specific factors that contribute to this risk. The ability to regulate negative affect effectively is critical to emotional and physical health and may play an important role in influencing risk for depression. We examined whether never-disordered daughters whose mothers have experienced recurrent episodes of depression during their daughters' lifetime differ from never-disordered daughters of never-disordered mothers in their patterns of neural activation during a negative mood induction and during automatic mood regulation. Sad mood was induced in daughters through the use of film clips; daughters then recalled positive autobiographical memories, a procedure shown previously to repair negative affect. During the mood induction, high-risk girls exhibited greater activation than did low-risk daughters in brain areas that have frequently been implicated in the experience of negative affect, including the amygdala and ventrolateral prefrontal cortex. In contrast, during automatic mood regulation, low-risk daughters exhibited greater activation than did their high-risk counterparts in brain areas that have frequently been associated with top-down regulation of emotion, including the dorsolateral prefrontal cortex and dorsal anterior cingulate cortex. These findings indicate that girls at high and low risk for depression differ in their patterns of neural activation both while experiencing, and while repairing negative affect, and suggest that anomalies in neural functioning precede the onset of a depressive episode.

Keywords: depression, cognition, emotion, fMRI, memory

Given the high personal and societal costs associated with Major Depressive Disorder (MDD), efforts to identify vulnerability factors for the onset of this disorder are particularly pressing. In this context, having parents who have experienced MDD is associated with a three- to fivefold increase in the risk to the offspring for developing a depressive episode during adolescence (Hammen, 2009). In particular, maternal depression has been found to be associated with an earlier onset and more severe course of depression in the offspring (Lieb, Isensee, Hoefler, Pfister, & Wittchen, 2002). The mechanisms underlying this risk, however, are not well understood. In this context, sustained negative affect is a hallmark feature of depression. Thus, individual differences in the ability to regulate and effectively respond to negative affect may play a critical role in influencing risk for this disorder (Campbell-Sills & Barlow, 2007; Joormann, Yoon, & Siemer, 2010). The construct of emotion regulation has received considerable attention in the developmental literature; indeed, adaptive self-regulation of negative affect is generally regarded as a key developmental task in childhood and adolescence (Cole, Martin, & Dennis, 2004; Eisenberg & Morris, 2002). Although it is not yet clear how difficulties in emotion regulation develop over time, numerous studies have linked risk for depression to impaired emotion regulation in children, adolescents, and adults, and have discussed the potential adverse impact of parental depression on this important ability (Goodman, 2007; Goodman & Gotlib, 1999; Kovacs, Joormann, & Gotlib, 2008). It is now clear that the responses that enable children and adolescents to modulate distress emerge relatively early in life and are affected by a complex set of psychosocial variables that interact with the children's innate neurobiology; it is also apparent that learning mechanisms, as well as interactions with caregivers, shape the acquisition of these responses (e.g., Morris et al., 2007). Thus, in the present study we focus on emotion regulation in the offspring of depressed mothers as a risk factor for the development of depression in later life.

An increasing number of studies have examined the neural bases of emotional responding and of efforts to consciously regulate negative emotions in response to aversive stimuli such as disgusting pictures and shocks (Beauregard, Levesque, & Bourgouin, 2001; Eippert, Veit, Weiskopf, Erb, Birbaumer & Anders, 2007; Goldin, McRae, Ramel, & Gross, 2008; Ochsner & Gross, 2008). In addition to identifying subcortical and prefrontal brain regions that mediate emotion processing and regulation, recent research has also examined how neural circuitry that subserves emotional responding and emotion regulation is altered in children and adolescents (see Monk, 2008, for a recent review). Few investigators,

This article was published Online First September 5, 2011.

Jutta Joormann, Department of Psychology, University of Miami; Rebecca E. Cooney, Melissa L. Henry, and Ian H. Gotlib, Department of Psychology, Stanford University.

This research was supported by National Institute of Mental Health Grant MH074849 awarded to Ian H. Gotlib and a NARSAD Young Investigator Award to Jutta Joormann.

Correspondence concerning this article should be addressed to Jutta Joormann, PhD., Department of Psychology, University of Miami, 454 Flipse Boulevard, Coral Gables, FL 33124. E-mail: jjoormann@psy.miami.edu

however, have examined these processes in clinically depressed individuals (Johnstone, van Reekum, Urry, Kalin, & Davidson, 2007) or in the offspring of depressed individuals. In the present study we examine neural activation during emotion generation and regulation in adolescent daughters of mothers with a history of depressive episodes.

Investigators have generally used self-report questionnaires to assess the frequency of use of emotion regulation strategies such as suppression and reappraisal (Campbell-Sills, Barlow, Brown, & Hofmann, 2006; Garnefski & Kraji, 2006; Garnefski & Kraji, 2007; Gross & John, 2003; Ehring, Fischer, Schnuelle, Boesterling, & Tuschen-Caffier, 2008); few researchers have examined whether depression is associated with a reduced effectiveness of specific strategies. One noteworthy exception is research examining the use of positive recall to repair negative affect. A number of studies have now demonstrated that people retrieve pleasant thoughts and memories to regulate unpleasant moods (e.g., Josephson, Singer, & Salovey, 1996). In this context, it is important to note that there are at least two broad but distinct classes of affect regulation processes. One class reduces negative affect by generating thoughts and appraisals that directly alter the perceptions of the situation that elicited the emotion or the experience of the negative affect (e.g., reappraisal). This form of emotion regulation is effortful and strategic. In contrast, a different type of regulation alleviates negative affect less directly by strengthening mood-incongruent (e.g., positive) thoughts and associations and by evoking competing affective states. This form of emotion regulation is frequently referred to as automatic (Mauss, Bunge, & Gross, 2007). Importantly, two recent studies that used a mood-repair task to investigate more automatic aspects of emotion regulation found that dysphoric participants, currently depressed participants, and participants with a history of depressive episodes but who are not currently depressed differ from control participants in their ability to effectively use recall of positive memories to repair their mood (Joormann & Siemer, 2004; Joormann, Siemer, & Gotlib, 2007). Even though depressed participants did not differ from nondepressed participants in the positivity of the memories they recalled, recalling these memories did not repair their negative mood. Indeed, the use of positive recall may be particularly critical to understanding depression because it involves the processing of idiosyncratic and, therefore, highly self-relevant material (Kross, Davidson, Weber, & Ochsner, 2009). In the current study, we examined the neural correlates of this automatic emotion regulation process in adolescents at high familial risk for depression.

Investigators examining the neural correlates of sad mood states have reported activations in the medial prefrontal cortex (MPFC), medial temporal lobes, anterior cingulate cortex (ACC), the insula, and the amygdala (Phan, Wager, Taylor, & Liberzon, 2002). The few studies that have assessed the neural substrates of the regulation of negative affect have focused primarily on correlates of reappraisal (Ochsner, Bunge, Gross, & Gabrieli, 2002) and suppression (Lévesque et al., 2003; Phan et al., 2005). In general, these studies have implicated a top-down modulation of ventral and limbic structures by dorsal brain regions, such as the dorsolateral prefrontal cortex (DLPFC) and ACC, in the regulation of affect (Ochsner & Gross, 2005; Kalisch, Wiech, Critchley, & Dolan, 2006). Importantly, previous studies have found similar activations in children compared to adults when examining neural correlates of sad mood and of the use of emotion regulation strategies (Monk, 2008; Durston et al., 2006).

In addition to dorsal structures, investigators have recently found ventromedial and ventrolateral areas, including the orbitofrontal cortex (OFC; BA 11), superior frontal gyrus (BA 10), the subgenual ACC (BA 25, 32), and the amygdala to be associated with the generation and regulation of emotional responses (Mauss, Bunge, & Gross, 2007; Phelps & LeDoux, 2005; Quirk & Beer, 2006). Given these findings, researchers have suggested that different emotion regulatory strategies recruit different brain regions (Ochsner & Gross, 2005). Importantly, using a design similar to the one utilized in this study with an unselected sample of adults, Cooney et al. (Cooney, Joormann, Atlas, Eugene & Gotlib, 2007) found mood-incongruent recall to be associated with activations in ventrolateral and ventromedial prefrontal cortices, including OFC.

Researchers are now beginning to use functional imaging to examine the neural correlates of specific emotion regulation processes. For example, Johnstone et al. (2007) reported increased activation of bilateral PFC during the reappraisal of negative affect, as well as anomalous connectivity between the ventromedial prefrontal cortex (VMPFC) and amygdala, in depressed compared with healthy control individuals. Similarly, Beauregard and colleagues (Beauregard, Paquette, & Lévesque, 2006) found that activation in BA 10 and the amygdala were associated with greater effort during suppression of sadness in depressed, but not in nondepressed, participants. In both studies, increased involvement of the VMPFC was interpreted as evidence of cortical inefficiency in depressed individuals during the voluntary down-regulation, or decrease, of negative affect. Importantly, it is not known whether cortical inefficiency during affect regulation is a symptom of a depressive episode, a "scar" of a previously experienced depressive episode or, alternatively, whether it is a risk factor that precedes the onset of depression. In this context, it is critical to examine the neural basis of various forms of affect regulation in individuals who are vulnerable to developing MDD but who have no past or current diagnosable emotional disorders.

The present study was designed to examine the neural correlates of both the induction of a sad mood state and the recall of positive autobiographical memories during the experience of a sad mood in girls at high and low risk for depression. Based on findings of studies examining neural correlates of sadness and of emotion regulation in depression, we predicted that, compared to the low-risk girls, the high-risk girls would exhibit greater activation in limbic areas and in the ventrolateral PFC as they experienced sad mood, and less activation in dorsal areas such as the dorsal ACC and DLPFC as they recalled positive memories following the induction of a sad mood in the service of repairing negative affect.

Method

Participants were 47 girls between the ages of 9 and 14 years with no current psychopathology and no history of any Axis I disorder according to the Diagnostic and Statistical Manual of Mental Disorders, 4th ed. (DSM, American Psychiatric Association, 1994). We recruited participants in this age group because girls younger than 9 years of age are likely to have difficulties with the task instructions, and because daughters of depressed mothers older than 14 years of age are likely to have experienced a depressive episode themselves (Angold, Costello, & Worthman,

1998). Twenty-seven girls had biological mothers with no current or past DSM Axis I disorder, and 20 girls had biological mothers with a history of recurrent MDD during their daughter's lifetime. All daughters lived with their biological mother. Participants were recruited through advertisements posted within the local community. A telephone screen established that both mothers and daughters were fluent in English and that daughters were between 9 and 14 years of age. Daughters were excluded if they had experienced severe head trauma, learning disabilities, and/or current or past depression.

Assessment of Psychopathology

Trained interviewers assessed the diagnostic status of daughters by administering the Schedule for Affective Disorders and Schizophrenia for School-Age Children-Present and Lifetime version (K-SADS-PL; Kaufman, Birmaher, Brent, Ryan, & Rao, 2000) separately to the daughters and to their mothers (about the daughters). The K-SADS-PL has been shown to generate reliable and valid child psychiatric diagnoses (Kaufman et al., 1997, 2000). A different interviewer administered the Structured Clinical Interview for the *DSM–IV* (SCID; First, Spitzer, Gibbons, & Williams, 1995) to the mothers. Both K-SADS-PL and SCID interviewers had previous experience administering structured clinical interviews. To assess interrater reliability, an independent rater who was blind to group membership evaluated 30% of our SCID and K-SAD-PL interviews by randomly selecting audiotapes of equal numbers of high-risk and low-risk pairs. In all cases these diagnoses matched those assigned by the original interviewer.

Daughters in the high-risk group (RSK) were eligible to participate in the study if: (a) they did not meet criteria for any past or current DSM Axis-I disorder according to both the parent and child K-SADS-PL; and (b) their mothers met the DSM criteria for at least two distinct episodes of MDD since the birth of their daughters, but did not currently meet criteria for MDD. In addition, mothers in the high-risk group were included only if they had no current diagnosis of any DSM Axis-I disorder, although they could have a past diagnosis. Daughters in the low-risk control group (CTL) were eligible to participate if: (a) they did not meet criteria for any past or current DSM Axis-I disorder based on both the parent and child K-SADS-PL, and (b) their mothers did not meet criteria for any DSM Axis-I disorder during their lifetime.

Questionnaires

Daughters completed the 10-item version of the Children's Depression Inventory (CDI-S; Kovacs, 1992). Self-report Tanner Staging (Tanner & Whitehouse, 1976) was used to assess pubertal status. Daughters also completed mood rating sheets before and after the mood induction. Ratings were made on a 5-point scale consisting of drawn face pictures, ranging from 1 = *very sad* to 5 = *very happy* (Taylor & Ingram, 1999). Finally, the vocabulary section of the verbal subtest of the Wechsler Intelligence Scale for Children-IV (WISC-IV; Wechsler, 2003) was administered to the daughters to ensure that the CTL and RSK groups did not differ in intellectual ability.

Procedure

Mood repair task. To minimize demand effects, participants were not told that this task assesses mood regulation, but were told instead that the task examines their ability to recall personal memories. The girls practiced recalling autobiographical memories outside the scanner to ensure that they understood the task instructions and were able to generate positive memories. In addition, we asked all girls to give a description of the memories they recalled while they were being scanned after the mood repair task was completed. A 1-min baseline scan was conducted while participants focused on a fixation cross. Participants then saw a screen with a text prompt that remained on for one minute asking them to recall a happy, positive memory from a recent event (*Positive Recall 1; PR1*). They then viewed a sad film clip depicting a young girl dying of cancer (4 minutes). Before watching the film clip, participants were instructed via audiotaped instructions to "get into the feeling" of the movie as intensely as possible and to imagine what they would feel if they were in this situation. After they watched the film clip, participants heard audiotaped instructions asking them to focus on the feelings associated with that event (2 minutes). Participants then saw a screen with a text prompt for one minute asking them to elaborate their sad mood by "really getting

Table 1
Demographic Characteristics and Mood Ratings

	CTL	RSK	
N	27	20	
Age	11.74 (1.29)	11.90 (1.21)	$t(45) < 1$
Ethnicity (% Caucasian)	70	60	$\chi^2(1,47) = 0.55, p > .05$
CDI	1.00 (1.24)	1.60 (1.64)	$t(45) = 1.43, p > .05$
WISC	50.27 (8.54)	51.79 (5.09)	$t(43) < 1$
Tanner−Breast	3.11 (0.81)	3.17 (1.25)	$t(43) < 1$
Tanner−Hair	2.96 (1.25)	3.06 (1.06)	$t(43) < 1$
Mood−Baseline	2.70 (0.87)	3.06 (0.73)	$t(43) = 1.41, p > .05$
Mood−PR 1	3.15 (0.83)	3.11 (0.83)	$t(42) < 1$
Mood−ME	2.16 (0.80)	2.33 (0.84)	$t(41) < 1$
Mood−PR 2	3.59 (0.51)	3.33 (0.49)	$t(27) = 1.35, p > .05$

Note. CTL = control group; RSK = high-risk group; CDI = Children Depression Inventory; WISC = Wechsler Intelligence Scale for Children; PR 1 = positive recall 1; ME = mood elaboration; PR 2 = positive recall 2. Standard deviations are presented in parentheses.

into the feeling" generated by the sad film clip and instructions (*Mood Elaboration; ME*). They were then prompted to recall a second autobiographical memory for one minute (*Positive Recall 2; PR2*). The text prompt remained on the screen for the duration of that scan. Before and after each scan, participants rated their mood. Four separate 1-min scans were collected: Baseline; Positive Recall 1; Mood Elaboration; and Positive Recall 2. Participants then described the events they had recalled in the scanner.

fMRI Data Acquisition and Analysis

Scans were conducted on a 1.5T GE Signa scanner. Functional images were acquired using a $T2^*$ in-/out- spiral pulse sequence (TE = 40 ms, flip = 90; Glover & Law, 2001) consisting of 24 4 mm interleaved slices acquired axially (in-plane resolution 3.75 × 3.75 mm, no gap) at a temporal resolution of 2s (2.00 TR). High-resolution structural scans were collected using a T1-weighted spoiled grass (TR = 100 ms; TE = 7 ms, flip = 90) sequence.

All preprocessing and analyses were performed with Analysis of Functional Neuro Images (AFNI; Cox, 1996). Voxel time series data were concatenated, slice time corrected, and corrected for motion, excluding participants who moved more than 1.5 mm. Data were spatially smoothed with a 4 mm Gaussian smoothing kernel, and high-pass filtered. Prior to analysis, functional images were coregistered to anatomical images. The preprocessed time series data for each participant were analyzed with multiple regression. Four contrast vectors were created: ME versus baseline, PR1 versus baseline, PR1 versus PR2, and PR2 versus baseline.

Table 2
Areas of Increased Activation in Response to Contrasts of Interest Within the CTL Group

Contrast	Region	BA	Vox	X	Y	Z	Max Z
Mood Elaboration							
(Mood Elaboration > Baseline)							
	MFG	9	18	−7.5	41.2	26.9	5.30
	dACC	32	10	−11.2	22.5	30.6	3.87
	Amygdala		7	−30	0	−18.1	3.99
	MFG		7	−11.2	48.8	0.6	3.48
	Claustrum		7	37.5	−11.2	11.9	3.79
	PG	6	6	48.8	0	34.4	3.88
	Claustrum		5	37.5	0	−6.9	3.87
	Putamen		5	30	−15	−6.9	4.23
	Thalamus		5	−3.8	−7.5	4.4	4.74
	ACC	24	5	7.5	0	34.4	4.31
	PCC	31	5	22.5	−33.8	38.1	3.39
Mood-Incongruent Recall							
(Positive Recall 2 > Baseline)							
	SFG	9	34	26.2	45	30.6	4.11
	dACC	24	23	−3.8	26.2	19.4	3.80
	dACC	32	20	−11.2	22.5	34.4	3.83
	Putamen		11	−18.8	11.2	−3.1	3.55
	dACC	32	8	7.5	18.8	34.4	3.42
	ITG	20	7	−52.5	−30	−14.4	4.04
	Caudate		7	−7.5	11.2	15.6	3.98
	MFG	10	6	−3.8	63.8	11.9	3.67
Mood Regulation							
(Positive Recall 2 > Positive Recall 1)							
	dACC	24	54	−7.5	15	26.9	4.52
	dACC	32	36	11.2	18.8	41.9	4.42
	Claustrum		30	30	−7.5	19.4	4.23
	MFG	8	27	30	15	34.4	3.83
	SFG	9	26	26.2	45	30.6	4.11
	mid dACC	24	21	11.2	3.8	34.4	4.02
	IFG	47	14	−22.5	30	−10.6	3.57
	dACC	32	14	7.5	7.5	45.6	3.85
	dACC	24	13	−18.8	−11.2	34.4	3.50
	Claustrum		12	30	7.5	15.6	4.11
	dACC	32	12	−11.2	37.5	19.4	3.47
	Caudate		10	−18.8	3.8	19.4	4.39
	MFG	9	9	22.5	26.2	23.1	3.62
	dACC	32	8	7.5	30	26.9	3.67
	PG	6	7	48.8	0	34.4	3.52
	PG	6	6	−56.2	0	19.4	3.87
	dACC	24	6	15	−7.5	45.6	3.11
	PCC	31	6	11.2	−22.5	41.9	3.72
	MFG	9	5	−26.2	33.8	26.9	3.45
	Insula	13	5	41.2	−41.2	26.9	2.94
	MFG	8	5	−7.5	26.2	45.6	2.98

Models included regressors for each contrast vector and included terms for residual motion regressors and trend regressors (i.e., baseline, linear, and quadratic). Resulting individual participant *t*-statistic maps were transformed into *z*-scores and warped into Talaraich space. The low- and high-risk group maps were analyzed with two-sample *t* tests for each contrast. Using the AFNI software program, AlphaSim, a joint voxel-wise and cluster-size threshold was determined for the between-groups comparison. A Monte Carlo simulation with 10,000 iterations was conducted with a voxel-wise threshold of $p < .005$ and a Gaussian kernel smoothing of 3.75 mm^3 (equivalent to one original voxel). A final corrected cluster threshold of $p < .05$ with a cluster minimum of 6 voxels was applied to whole-brain contrasts. Because of the large number of activation points generated by this same thresholding for the within-group comparisons, we present the activations for these contrasts at an uncorrected threshold of $p < .001$ with a cluster minimum of 5 voxels.

Results

Participant Characteristics, Mood Ratings, and Ratings of Memories

The RSK and CTL groups did not significantly differ in age, ethnicity, intellectual ability, pubertal development, or level of depressive symptoms (see Table 1). The transcribed recordings of each participant's memories were rated by two independent raters on a 10-point scale (1 = *not at all happy* to 10 = *extremely happy*) with respect to how happy the recalled event would be for an average person. Because of random technical difficulties with the recordings, data are missing for participants in both groups (CTL: 44%; RSK: 40%); there were no group differences in the proportion of participants for whom recordings of their memories are missing. The interrater agreement was $r = .89$ for the first memory and $r = .87$ for the second memory. The two memories recalled by the participants did not differ in their average happiness ratings (CTL: $M = 6.60$, $SD = 2.56$; $M = 7.29$, $SD = 1.27$, $t(13) = 1.28$, $p > .05$; RSK: $M = 7.00$, $SD = 2.36$, $M = 7.17$, $SD = 1.80$, $t(11) < 1$). The groups did not differ in the positivity of the memories recalled at Time 1 or Time 2, all $ts(25) < 1$. A two-way (Group: CTL, RSK; Time: Baseline, PR1, ME, PR2) repeated-measures analysis of variance (ANOVA) conducted on the mood ratings yielded a significant main effect for time, $F(3, 75) = 29.78$, $p < .01$ but no significant main effect of group, $F(1, 25) < 1$, or interaction of group and time, $F(3, 75) < 1$. Follow-up paired *t* tests indicate that participants' mood ratings decreased significantly after the negative mood induction, $t(42) = 5.91$, $p < .01$, and increased again after recalling the second positive autobiographical memory, $t(27) = 7.58$, $p < .01$, indicating that the sad mood induction was successful for both the CTL and RSK groups and that recalling positive memories improved participants' mood state.

Within-Group fMRI Contrasts

Sad mood elaboration. To identify brain regions involved in the generation and elaboration of the sad mood induced by the film clip, we contrasted the ME scan with the initial baseline scan. Table 2 presents regions activated during this contrast in the CTL group and Table 3 presents regions activated in the RSK group. Importantly, while activations were present in both groups in the amygdala, the CTL girls also showed extensive activation in middle frontal gyrus (MFG; BA 9), dorsal ACC (BA 32), ACC (BA 24), and putamen. The RSK girls showed additional activation in inferior frontal gyrus (IFG; BA 47), fusiform gyrus, subcallosal (BA 47) and subgenual cingulate gyrus (BA 25).

Automatic mood regulation. We examined two specific contrasts designed to assess the neural substrates associated with the use of mood-incongruent recall to repair a sad mood. The first contrast (PR2 vs. baseline) was designed to yield activations associated with recall of positive autobiographical memories following a sad mood induction. This contrast yielded activations in DLPFC (BA 9), dorsal ACC (BA 24,32), ITG (BA 20), putamen and caudate in the CTL girls, and in amygdala and fusiform gyrus (FFG) in the RSK girls. While important, this contrast cannot differentiate activation associated generally with the recall of positive autobiographical memories from activation associated specifically with mood-incongruent recall. Therefore, the second contrast (PR2 vs. PR1) was designed to identify neural activations that are unique to the

Table 3
Areas of Increased Activation in Response to Contrasts of Interest Within the RSK Group

Contrast	Region	BA	Vox	X	Y	Z	Max Z
Mood Elaboration							
(Mood Elaboration > Baseline)							
	IFG	47	10	−26.2	7.5	−14.4	3.40
	FFG	20	5	45	−3.8	−21.9	3.45
	SCG	47	5	15	15	−14.4	3.85
	Amygdala		5	30	0	−14.4	3.61
	sgACC	25	5	−3.8	3.8	−6.9	3.62
Mood-Incongruent Recall							
(Positive Recall 2 > Baseline)							
	FFG	20	7	45	−3.8	−25.6	3.65
	Amygdala		5	30	−7.5	−21.9	3.92
Mood Regulation							
(Positive Recall 2 > Positive Recall 1)							
	MTG	21	10	45	0	−21.9	3.92
	MTG	21	10	52.5	−11.2	−18.1	4.45
	Caudate		5	18.8	18.8	8.1	3.82

recall of *mood-incongruent* positive autobiographical memory (i.e., areas that are differentially active during recall of a positive memory while in a sad mood state). Areas of activation in this contrast are listed in Tables 2 and 3. Both the CTL and RSK groups showed activation in caudate. In addition, the CTL girls showed activations in parts of the dorsal ACC (BA 24, 32), the DLPFC, and the IFG (BA 47); in contrast, the RSK girls exhibited extensive additional activations in medial temporal gyrus (BA 21).

Between-Group Comparisons

Sad mood elaboration. Table 4 presents regions that were differentially activated in RSK versus CTL girls during the mood elaboration scan (see also Figure 1). Compared to the RSK girls, CTL girls showed greater activations in occipital regions implicated in visualizing remembered events, including the right lingual gyrus (LG) and the left FFG (e.g., Burgess, Maguire, Spiers, O'Keefe, 2001; Maguire, Frackowiak, & Frith, 1997), as well as in regions involved in self-referential processing and autobiographical recall, such as the cuenus and precuneus (e.g., Lou et al., 2004). In contrast, the RSK girls showed increased activation in regions frequently associated with the experience of negative affect, including the left ventrolateral PFC (IFG; BA 47).

Automatic mood regulation. The first contrast (PR2 vs. baseline) yielded greater activation in RSK than in CTL girls in the

Table 4
Areas of Increased Activation In Response To Contrasts of Interest

Contrast	Region	BA	Vox	X	Y	Z	Max Z
Mood Elaboration							
(Mood Elaboration > Baseline)							
RSK > CTL	MTG/FFG	37	13	48.8	−33.8	−10.6	2.75
	PG	4	12	−30.0	−15.0	49.4	2.78
	PG	4	11	−30.0	−26.2	56.9	3.02
	IFG	47	9	−18.8	7.5	−18.1	2.64
	IFG	9	9	45.0	−3.8	19.4	2.67
CTL > RSK	Cuneus	18	25	18.8	−82.5	26.9	−3.04
	LG	17	17	22.5	−82.5	0.6	−2.38
	MTG	39	17	33.8	−60.0	26.9	−3.02
	Cuneus	19	16	−7.5	−86.2	30.6	−2.59
	Precuneus	7	14	−18.8	−75.0	41.9	−2.92
	FFG	19	13	−33.8	−71.2	−14.4	−2.37
	FFG	37	12	−41.2	−48.8	−18.1	−2.79
	IFG	46	11	−52.5	37.5	8.1	−2.71
	Precuneus	31	9	15.0	−63.8	26.9	−2.42
	Precuneus	7	9	3.8	−75.0	41.9	−2.44
Mood-Incongruent Recall							
(Positive Recall 2 > Baseline)							
RSK > CTL	MFG	11	27	30.0	33.8	−10.6	2.86
	PG		26	−26.2	−15.0	49.4	3.40
	Thalamus		13	−7.5	−15.0	19.4	3.17
	Parahippocamp/Amygdala		11	22.5	−11.2	−10.6	2.69
	PG	4	11	33.8	−22.5	60.6	2.69
	MFG	6	9	−30.0	−7.5	60.6	2.64
CTL > RSK	FFG	19	19	−22.5	−56.2	−10.6	−2.66
	IFG	46	14	−48.8	41.2	11.9	−2.91
	dACC	32	14	22.5	15.0	26.9	−3.02
	dACC	32	14	−18.8	7.5	34.4	−2.83
	OG	19	11	41.2	−63.8	−6.9	−2.62
	FFG	37	10	−41.2	−48.8	−14.4	−3.12
Mood Regulation							
(Positive Recall 2 > Positive Recall 1)							
RSK > CTL	MFG	11	23	30.0	33.8	−10.6	3.50
	PG	6	14	−30.0	−11.2	53.1	2.45
	Thalamus		9	−7.5	−26.2	15.6	2.53
CTL > RSK	LG	17	196	15.0	−82.5	0.6	−3.57
	Precuneus	7	71	3.8	−75.0	41.9	−3.18
	FFG	37	64	−33.8	−56.2	−10.6	−3.08
	Cuneus	18	18	18.8	−82.5	26.9	−3.04
	Precuneus		14	26.2	−52.5	26.9	−2.91
	dACC	32	13	−18.8	7.5	34.4	−2.98
	MFG	9	12	−26.2	33.8	26.9	−3.17
	SFG	10	11	−30.0	56.2	11.9	−2.73
	Caudate		11	−15.0	3.8	15.6	−3.56
	FFG	37	10	−48.8	−45.0	−14.4	−2.96
	Cuneus	17	10	−18.8	−93.8	4.4	−2.45
	dACC	32	9	18.8	15.0	26.9	−2.37

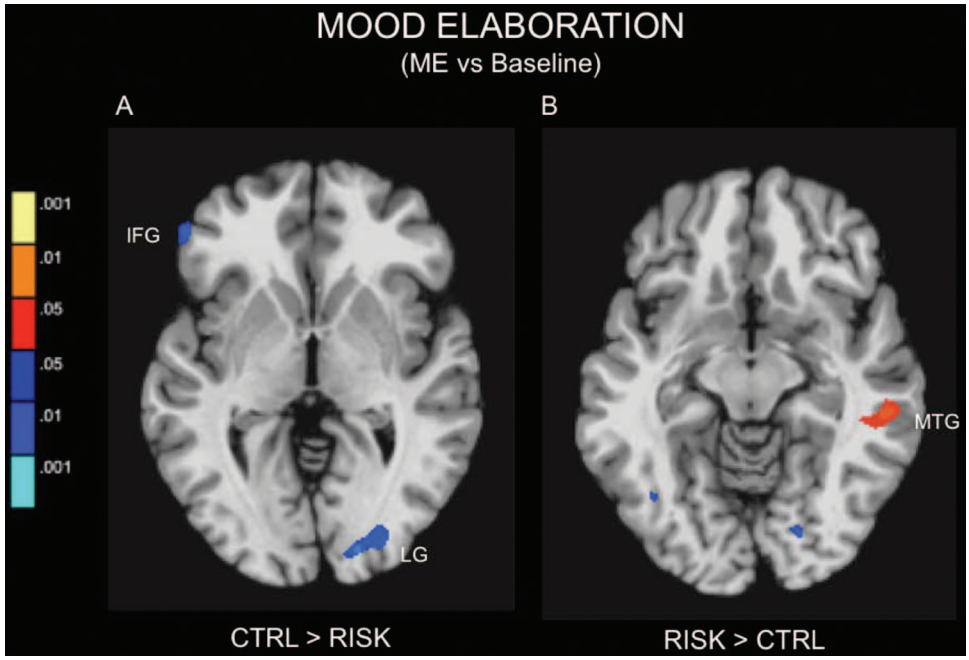

Figure 1. Mood Elaboration. Group differences in activation during the instantiation of a sad mood state compared with baseline (Mood Elaboration vs. Baseline). A) Activations depicted in blue (CTL > RISK): indicate greater activation in inferior frontal gyrus, IFG, and lingual gyrus, LG, in CTL relative to RSK participants. B) Activation depicted in red (RISK > CTL): indicates greater activation in medial temporal gyrus, MTG, in RSK relative to CTL participants.

OFC (MFG; BA 11) and in parahippocampus/amygdala (see Figure 2). In contrast, CTL girls exhibited greater activation than did RSK girls in dorsal areas frequently associated with voluntary emotion regulation, such as dorsal ACC. Again, while important, this contrast cannot differentiate activation associated generally with the recall of positive autobiographical memories from activation associated specifically with mood-incongruent recall. Therefore, the second contrast (PR2 vs. PR1) was designed to identify neural activations that are unique to the recall of *mood-incongruent* positive autobiographical memory (i.e., activations that are differentially active during recall of a positive memory in a sad mood state). Areas of activation that differentiated the CTL and RSK girls during this contrast are listed in Table 4 and depicted in Figure 3. Most strikingly, the RSK girls continued to show greater activation in the OFC/MFG than did the CTL girls, who continued to show increased activation in the dorsal ACC, and activation within the superior frontal gyrus (SFG), lingual gyrus (LG), cuneus (CU), precuneus, and caudate.

ROI Analysis

Region of interest (ROI) analysis was conducted by extracting percent signal change (PSC) from predefined AFNI masks for three regions: right amygdala, right OFC, and dACC (BA 32). These regions were examined because each yielded significant between-groups differences in our whole-brain analysis and each has been implicated in either the experience or the regulation of negative affect. Because we were interested in identifying how PSC changed across the various contrasts in each of these regions, we also included the initial positive recall contrast (PR1vB1) in addition to the three primary contrasts of interest.

We extracted PSC from our three ROIs [right amygdala, right OFC, and bilateral dACC (BA 32)] and analyzed these data with two-way (Group: CTL, RSK; Contrast: PR1vB1, MEvB1, PR2vB1, and PR2vPR1) repeated measures ANOVAs. We found no significant group differences for the right OFC and bilateral dACC (BA 32) ROIs, $F(3, 45) = .92$, $p > .05$ and $F(3, 42) = .12$, $p > .05$, respectively. For the right amygdala ROI, we found significant main effects for group, $F(3, 45) = 4.9$, $p < .03$, and contrast, $F(3, 45) = 3.6$, $p < .02$, that were qualified by a significant interaction of group and contrast. $F(3, 45) = 4.7$, $p < .01$ (see Figure 4). Follow-up tests indicated that the CTL and RSK girls did not differ with respect to PSC in the right amygdala ROI in the initial contrast (PR1vB1), $t(45) = -.56$, $p > .05$, but did differ significantly in the other three contrasts (MEvB1, $t(45) = -2.0$, $p < .05$; PR2vB1, $t(45) = -2.3$, $p < .03$; and PR2vPR1, $t(45) = -2.3$, $p < .02$). For all of these contrasts, the RSK girls exhibited greater activation in right amygdala than did the CTL girls.

Discussion

Despite a growing literature demonstrating that offspring of depressed mothers are at elevated risk for developing a depressive episode, we know relatively little about the factors and mechanisms that underlie this heightened risk. In this study we compared neural aspects of the instantiation of sad mood and of the use of mood-incongruent autobiographical recall to repair that mood in

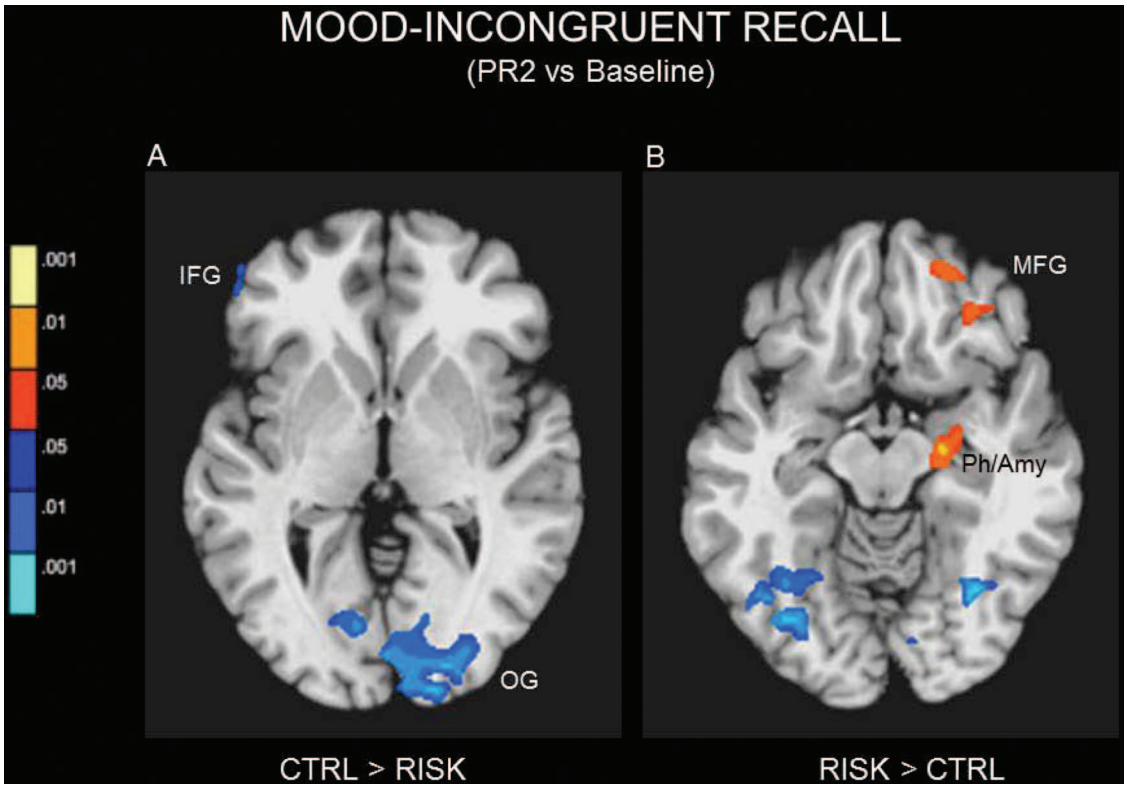

Figure 2. Mood-Incongruent Recall. Group differences in activation during mood-incongruent positive autobiographical recall after compared with baseline (Positive Recall 2 vs. Baseline). A) Activations depicted in blue (CTL > RSK): indicate greater activation in middle occipital gyrus, MOG, and lingual gyrus, LG, in CTL relative to RSK participants. B) Activations depicted in red (RISK > CTL): indicate greater activation in medial frontal gyrus, MFG, and parahippocampus/amygdala, Ph/Amy, in RSK relative to CTL participants.

carefully diagnosed never-disordered daughters of mothers with recurrent depression and never-disordered daughters of never-disordered mothers. Consistent with the results of previous behavioral studies investigating this mood regulation process in adults (Joormann & Siemer, 2004; Joormann et al., 2007), viewing a sad film clip and being instructed to elaborate the resultant sad mood significantly worsened participants' mood state; subsequently recalling a positive autobiographical memory significantly improved their sad mood.

We also found that the induction and elaboration of a sad mood state were associated with increased neural response in the amygdala, ventrolateral PFC, and subgenual ACC, regions that have been found to be active during the experience of sadness in studies using a variety of experimental paradigms to induce affect (Phan et al., 2002; Lévesque et al., 2003). These results indicate both that we successfully induced a sad mood state in participants and that this experience activated the same regions that have been identified in previous studies examining the neural correlates of sadness. Indeed, previous studies have documented similar activations in response to a sad mood induction in children and adults (Lévesque et al., 2003; Monk, 2008). In addition, instructions to repair the mood state using positive autobiographical memories led to activations in areas frequently associated with the top-down regulation of emotional states, including DLPFC and dorsal ACC (Goldin et al., 2008; Phan et al., 2005; Ochsner & Gross, 2005). Again, previous studies have found similar activations in children compared to adults when using emotion regulation strategies (Monk, 2008; Durston et al., 2006) Previous studies, however, have also reported more extensive activation in multiple structures of the PFC in children than in adults. Indeed, when comparing our findings to neural correlates of mood-incongruent recall obtained in a study with female adult participants (Cooney et al., 2007), activations in prefrontal cortex in the adult participants were more defined and limited primarily to ventromedial and ventrolateral PFC. These results replicate findings of studies that activation decreased with age in DLPFC and became more focal in the ventral PFC during a cognitive control task (Durston et al., 2006) and that children showed more extensive activation in PFC in a task that involved self-regulation of sadness (Lévesque et al., 2003; Lévesque et al., 2004).

The primary goal of this study, however, was to extend this body of research by comparing girls at high and low risk for depression. We had predicted, that compared to the low-risk girls, the high-risk girls would exhibit greater activation in limbic areas and in the ventrolateral PFC as they experienced sad mood, and less activation in dorsal areas such as the dorsal ACC and DLPFC as they recalled positive memories following the induction of a sad mood in the service of repairing negative affect. We found group differences during both mood induction and automatic mood regulation. During the mood induction

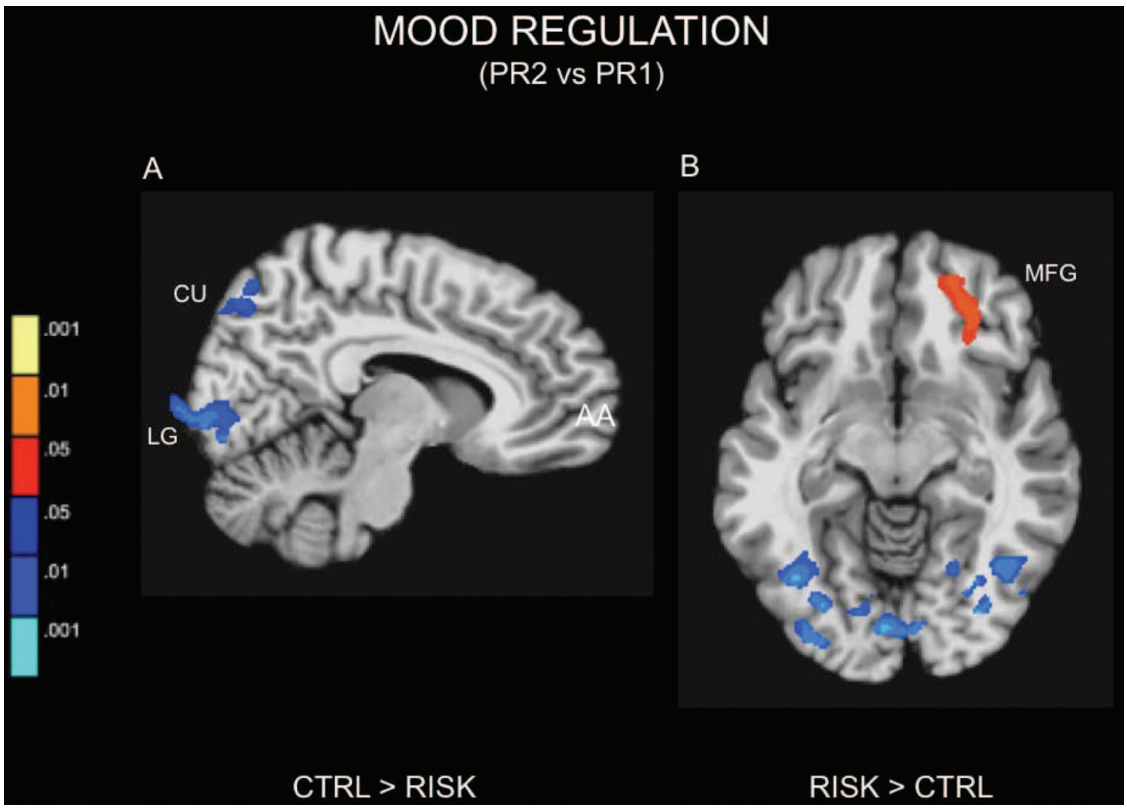

Figure 3. Mood Regulation. Group differences during mood regulation (Positive Recall 2 vs. Positive Recall 1). A) Activations depicted in blue (CTL > RSK): indicate greater activation in the cuneus, CU, and lingual gyrus, LG, in CTL relative to RSK participants. B) Activation depicted in red (RISK > CTL): indicates greater activation in medial frontal gyrus, MFG, in RSK relative to CTL participants.

scan, compared with their high-risk counterparts, the low-risk girls showed greater activation in areas frequently associated with visualization and in areas associated with autobiographical recall and self-referential processing. In contrast, the high-risk girls showed greater activation in areas frequently associated with the experience of sadness, in particular, in the ventrolateral PFC. These findings suggest that the CTL girls were more cognitively engaged with the task than were the RSK girls, possibly by trying to imagine themselves in the situation depicted in the film and trying to remember similar events from their lives. The RSK girls, however, responded with stronger activation of regions involved in the experience of sadness and with more focal ventrolateral PFC activation.

During automatic mood regulation, the RSK girls exhibited greater activation in OFC (MFG; BA 11) and the parahippocampus/amygdala complex, whereas the CTL girls exhibited greater activation in areas commonly associated with efforts to repair negative affect, such as dACC and DLPFC (BAs 32 and 9, respectively; Goldin et al., 2008; Mauss, Bunge, & Gross, 2007). These findings suggest that RSK girls had difficulties recruiting dorsal areas of the prefrontal cortex to decrease limbic activation in the service of repairing negative affect and, therefore, resulting in the observed sustained activation of the amygdala. Indeed, the ROI analysis yielded no group difference in right amygdala activation at baseline, but greater amygdala activation following the mood induction and following the mood regulation task in the RSK than in the CTL girls. These results are consistent with Johnstone et al.'s (2007) findings of changes in cortical involvement in depressed individuals during the voluntary down-regulation of negative affect. Importantly, in the present study these differences in neural processing were obtained in never-disordered girls with no past or current psychopathology who are at high risk for depression due to their mother's history of depressive episodes. These findings suggest, therefore, that changes in cortical involvement during automatic mood regulation are a trait marker of depression that is present in high-risk individuals before they experience their first depressive episode. These changes may lead to increased reactivity to negative life events or stressors (Gotlib, Joormann, Minor, & Hallmayer, 2008) and to difficulties regulating the resultant negative affect, a process that may be an important risk factor for depression.

In this study we examined automatic emotion regulation in girls at high risk for depression using a task with high ecological validity, requiring girls to recall complex emotional experiences from their own lives. The girls were not instructed to regulate affect, they were only instructed to recall mood-incongruent memories. Previous studies have shown that such recall repairs negative affect (Joormann & Siemer, 2004; Josephson et al., 1996). Designs that do not explicitly instruct

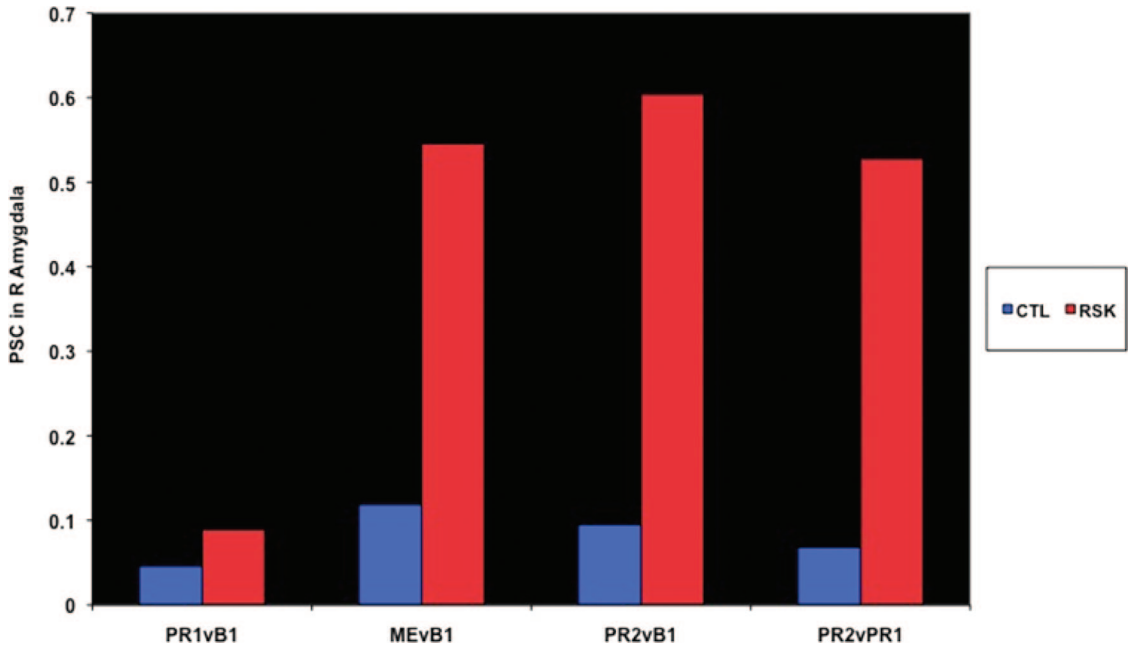

Figure 4. Percent Signal Change in Right Amygdala Across Contrasts. Percent signal change extracted from the right amygdala ROI for each contrast: positive autobiographical memory recall compared with baseline (PR1vB1), negative mood elaboration compared with baseline (MEvB1), mood-incongruent positive autobiographical memory recall (PR2vB1), and mood regulation (PR2v1).

participants to regulate affect are not as susceptible to demand effects as are tasks that ask people to use a specific strategy. Certainly, a number of diverse cognitive and emotional processes are engaged in this recall, which limits our ability to compare findings from this study directly to results of fMRI studies that have used generic stimuli and more tightly controlled experimental designs. Thus, it is possible that there is variation in the strategies people used both during the mood elaboration and during the mood regulation phase. In addition, we found no group differences for the behavioral variables, that is, self-reported mood ratings and ratings of recalled memories. In previous behavioral studies using a similar mood regulation task, no differences were found in ratings of recalled memories among depressed, formerly depressed and nondepressed adult participants (Joormann & Siemer, 2004; Joormann et al., 2007). In these studies, this finding was interpreted as suggesting that although all participants recalled equivalently positive memories, remembering these events does not repair negative affect in depressed individuals. Previous studies, however, did find group differences in self-reported affect as a function of recalling positive memories. It is possible that the lack of behavioral findings in the current study is due either to lower power given some missing data for these variables or to a lack of sensitivity of our mood measure to detect group differences in mood in children. We should point out, however, that when collapsing data across groups, mood ratings decreased after the mood elaboration and increased after the positive recall. It is also possible that the fMRI data are more sensitive, and that risk status in the absence of current or past psychopathology is not associated with behavioral group differences. Future studies are needed to examine this possibility more explicitly. In addition, the cross-sectional design of this study does not allow us to examine predictors of the onset of depression. Longitudinal follow-up studies are needed to determine whether the differences between low-risk and high-risk participants reported in this study predict which daughters will go on to develop a depressive episode. Finally, although our findings indicate that daughters of mothers with a history of depression differ from their low-risk counterparts in patterns of neural activation during mood-incongruent recall, the mechanisms underlying these group differences remain to be elucidated. The responses that enable young children to modulate emotions emerge within a complex interaction of psychosocial and biological factors (e.g., Goldsmith & Davidson, 2004). Recent studies have focused on examining genetic factors, but more research is needed examining learning processes, interactions between children and their caregivers, and the effects of these factors on depression, emotion regulation, and in the intergenerational transmission of risk for psychopathology.

Successful affect regulation is critical to maintaining both physical and mental health. This study reports differences in neural correlates of the experience of negative affect and of automatic emotion regulation between low-risk and high-risk girls, none of whom have (yet) experienced an episode of depression or any other DSM Axis-I disorder. The findings of this study suggest that sustained amygdala activation and reduced cortical involvement when repairing negative affect are not simply symptoms of depression, but instead, may be trait markers of vulnerability to this disorder. Future studies are needed to examine whether these differences between low- and

high-risk girls in neural correlates of affect regulation predict the onset of a first episode of depression.

References

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Angold, A., Costello, E. J., & Worthman, C. M. (1998). Puberty and depression: The roles of age, pubertal status and pubertal timing. *Psychological Medicine, 28,* 51–61. doi:10.1017/S003329179700593X

Beauregard, M., Lévesque, J., & Bourgouin, P. (2001). Neural correlates of conscious self-regulation of emotion. *Journal of Neuroscience, 21,* 6993–7000.

Beauregard, M., Paquette, V., & Lévesque, J. (2006). Dysfunction in the neural circuitry of emotional self-regulation in major depressive disorder. *Neuroreport, 17,* 843–846. doi:10.1097/01.wnr.0000220132.32091.9f

Burgess, N., Maguire, E. A., Spiers, H. J., & O'Keefe, J. (2001). A temporoparietal and prefrontal network for retrieving the spatial context of lifelike events, *NeuroImage 14,* 439–453. doi:10.1006/nimg.2001.0806

Campbell-Sills, L., & Barlow, D. (2007). Incorporating emotion regulation into conceptualizations and treatments of anxiety and mood disorders. In J. Gross & P. Thompson (Eds.), *Handbook of emotion regulation* (pp. 542–559). New York, NY: Guilford Press.

Campbell-Sills, L., Barlow, D., Brown, T., & Hofmann, S. (2006). Acceptability and suppression of negative emotion in anxiety and mood disorders. *Emotion, 6,* 587–595. doi:10.1037/1528-3542.6.4.587

Cole, P. M., Martin, S. E., & Dennis, T. A. (2004). Emotion regulation as a scientific construct: Methodological challenges and directions for child development research. *Child Development, 75,* 317–333. doi:10.1111/j.1467-8624.2004.00673.x

Cooney, R. E., Joormann, J., Atlas, L., Eugène, F., & Gotlib, I. (2007). Remembering the good times: Neural correlates of affect regulation. *Neuroreport, 18,* 1771–1774. doi:10.1097/WNR.0b013e3282f16db4

Cox, R. W. (1996). AFNI: Software for analysis and visualization of functional magnetic resonance neuroimages. *Computers and Biomedical Research, 29,* 162–173. doi:10.1006/cbmr.1996.0014

Durston, S., Davidson, M., Tottenham, N., Galvan, A., Spicer, J., Fossella, J. A., & Casey, B. J. (2006). A shift from diffuse to focal cortical activity with development. *Developmental Science, 9,* 1–8. doi:10.1111/j.1467-7687.2005.00454.x

Ehring, T., Fischer, S., Schnülle, J., Bösterling, A., & Tuschen-Caffier, B. (2008). Characteristics of emotion regulation in recovered depressed versus never depressed individuals. *Personality and Individual Differences, 44,* 1574–1584. doi:10.1016/j.paid.2008.01.013

Eippert, F., Veit, R., Weiskopf, N., Erb, M., Birbaumer, N., & Anders, S. (2007). Regulation of emotional responses elicited by threat-related stimuli. *Human Brain Mapping, 28,* 409–423. doi:10.1002/hbm.20291

Eisenberg, N., & Morris, A. (2002). Children's emotion-related regulation. In R. V. Kail (Ed.), *Advances in child development and behavior* (Vol. 30, pp. 189–229). San Diego, CA: Academic Press. doi:10.1016/S0065-2407(02)80042-8

First, M. B., Spitzer, M. B., Gibbon, M., & Williams, J. B. W. (1995). *Structured Clinical Interview for DSM-IV Axis I Disorders−Patient Edition (SCID-I/P, Version 2.0).* New York, NY: New York State Psychiatric Institute.

Garnefski, N., & Kraaij, V. (2006). Relationships between cognitive emotion regulation strategies and depressive symptoms: A comparative study of five specific samples. *Personality and Individual Difference, 40,* 1659–1669. doi:10.1016/j.paid.2005.12.009

Garnefski, N., & Kraaij, V. (2007). The Cognitive Emotion Regulation Questionnaire: Psychometric features and prospective relationships with depression and anxiety in adults. *European Journal of Psychological Assessment, 23,* 141–149. doi:10.1027/1015-5759.23.3.141

Glover, G. H., & Law, C. S. (2001). Spiral-in/out BOLD fMRI for increased SNR and reduced susceptibility artifacts. *Magnetic Resonance in Medicine, 46,* 515–522. doi:10.1002/mrm.1222

Goldin, P. R., McRae, K., Ramel, W., & Gross, J. (2008). The neural bases of emotion regulation: Reappraisal and suppression of negative emotion. *Biological Psychiatry, 63,* 577–586. doi:10.1016/j.biopsych.2007.05.031

Goldsmith, H. H., & Davidson, R. J. (2004). Disambiguating the Components of Emotion Regulation. *Child Development, 75,* 361–365.

Goodman, S. H. (2007). Depression in mothers. *Annual Review of Clinical Psychology, 3,* 107–135.

Goodman, S. H., & Gotlib, I. H. (1999). Risk for psychopathology in the children of depressed mothers: A developmental model for understanding mechanisms of transmission. *Psychological Review, 106,* 458–490. doi:10.1037/0033-295X.106.3.458

Gotlib, I. H., Joormann, J., Minor, K., & Hallmayer, J. (2008). HPA axis reactivity: A mechanism underlying the associations among 5-HTTLPR, stress, and depression. *Biological Psychiatry, 63,* 847–851. doi:10.1016/j.biopsych.2007.10.008

Gross, J. J., & John, O. (2003). Individual differences in two emotion regulation processes: Implications for affect, relationships, and well-being. *Journal of Personality and Social Psychology, 85,* 348–362. doi:10.1037/0022-3514.85.2.348

Hammen, C. (2009). Children of depressed parents. In I. H. Gotlib & C. Hammen (Eds.), *Handbook of depression* (pp. 275–297). New York, NY: Guilford Press.

Johnstone, T., van Reekum, C., Urry, H., Kalin, N., & Davidson, R. (2007). Failure to regulate: Counterproductive recruitment of top-down prefrontal-subcortical circuitry in major depression. *Journal of Neuroscience, 27,* 8877–8884. doi:10.1523/JNEUROSCI.2063-07.2007

Joormann, J., & Siemer, M. (2004). Memory accessibility, mood regulation, and dysphoria: Difficulties in repairing sad mood with happy memories? *Journal of Abnormal Psychology, 113,* 179–188. doi:10.1037/0021-843X.113.2.179

Joormann, J., Siemer, M., & Gotlib, I. (2007). Mood regulation in depression: Differential effects of distraction and recall of happy memories on sad mood. *Journal of Abnormal Psychology, 116,* 484–490. doi:10.1037/0021-843X.116.3.484

Joormann, J., Yoon, K., & Siemer, M. (2010). Cognition and emotion regulation. In A. Kring & D. Sloan (Eds.), *Emotion regulation and psychopathology: A transdiagnostic approach to etiology and treatment* (pp. 174–203). New York, NY: Guilford Press.

Josephson, B., Singer, J., & Salovey, P. (1996). Mood regulation and memory: Repairing sad moods with happy memories. *Cognition and Emotion, 10,* 437–444. doi:10.1080/026999396380222

Kalisch, R., Wiech, K., Critchley, H., & Dolan, R. (2006). Levels of appraisal: A medial prefrontal role in high-level appraisal of emotional material. *Neuroimage, 30,* 1458–1466. doi:10.1016/j.neuroimage.2005.11.011

Kaufman, J., Birmaher, B., Brent, D., Rao, U., Flynn, C., Moreci, P., . . . Ryan, N., (1997). Schedule for affective disorders and schizophrenia for school-age children—Present and lifetime version (K-SADS-PL): Initial reliability and validity data. *Journal of the American Academy of Child and Adolescent Psychiatry 36,* 980–988. doi:10.1097/00004583-199707000-00021

Kaufman, J., Birmaher, B., Brent, D., Ryan, N. D., & Rao, U. (2000). K-SADS-PL. *Journal of the American Academy of Child and Adolescent Psychiatry, 39,* 1208. doi:10.1097/00004583-200010000-00002

Kovacs, M. (1992). *The Children's Depression Inventory manual.* New York, NY: Multi-Health Systems.

Kovacs, M., Joormann, J., & Gotlib, I. H. (2008). Emotion (dys)regulation and links to depressive disorders. *Child Development Perspectives, 2,* 149–155. doi:10.1111/j.1750-8606.2008.00057.x

Kross, E., Davidson, M., Weber, J., & Ochsner, K. (2009). Coping with

emotions past: The neural bases of regulating affect associated with negative autobiographical memories. *Biological Psychiatry, 65,* 361–366. doi:10.1016/j.biopsych.2008.10.019

Lévesque, J., Joanette, Y., Mensour, B., Beaudoin, G., Leroux, J., Bourgouin, P., & Beauregard, M. (2003). Neural correlates of sad feelings in healthy girls. *Neuroscience, 121,* 545–551. doi:10.1016/S0306-4522(03)00528-1

Lévesque, J., Joanette, Y., Mensour, B., Beaudoin, G., Leroux, J., Bourgouin, P., & Beauregard, M. (2004). Neural basis of emotional self-regulation in childhood. *Neuroscience, 129,* 361–369. doi:10.1016/j.neuroscience.2004.07.032

Lieb, R., Isensee, B., Höfler, M., Pfister, H., & Wittchen, H. (2002). Parental major depression and the risk of depression and other mental disorders in offspring: A prospective-longitudinal community study. *Archives of General Psychiatry, 59,* 365–374. doi:10.1001/archpsyc.59.4.365

Lou, H. C., Luber, B., Crupain, M., Keenan, J. P., Nowak, M., Kjaer, T. W., . . . Lisanby, S. H. (2004). Parietal cortex and representation of the mental Self. *Proceedings of the National Academy of Sciences of the United States of America, 101,* 6827–6832. doi:10.1073/pnas.0400049101

Maguire, E. A., Frackowiak, R. S. J., & Frith, C. D. (1997). Recalling routes around London: Activation of the right hippocampus in taxi drivers, *Journal of Neuroscience, 17,* 7103–7110.

Mauss, I., Bunge, S., & Gross, J. (2007). Automatic emotion regulation. *Social and Personality Psychology Compass, 1,* 146–167. doi:10.1111/j.1751-9004.2007.00005.x

Monk, C. S. (2008). The development of emotion-related neural circuitry in health and psychopathology. *Development and Psychopathology, 20,* 1231–1250. doi:10.1017/S095457940800059X

Morris, A. S., Silk, J. S., Steinberg, L., Myers, S. S., & Robinson, L. R. (2007). The role of the family context in the development of emotion regulation. *Social Development, 16,* 361–388.

Ochsner, K. N., Bunge, S., Gross, J., & Gabrieli, J. (2002). Rethinking feelings: An FMRI study of the cognitive regulation of emotion. *Journal of Cognitive Neuroscience, 14,* 1215–1229. doi:10.1162/089892902760807212

Ochsner, K. N., & Gross, J. (2005). The cognitive control of emotion. *Trends in Cognitive Sciences, 9,* 242–249. doi:10.1016/j.tics.2005.03.010

Ochsner, K., & Gross, J. (2008). Cognitive emotion regulation: Insights from social cognitive and affective neuroscience. *Current Directions in Psychological Science, 17,* 153–158. doi:10.1111/j.1467-8721.2008.00566.x

Phan, K. L., Fitzgerald, D., Nathan, P., Moore, G., Uhde, T., & Tancer, M. (2005). Neural Substrates for Voluntary Suppression of Negative Affect: A Functional Magnetic Resonance Imaging Study. *Biological Psychiatry, 57,* 210–219. doi:10.1016/j.biopsych.2004.10.030

Phan, K. L., Wager, T., Taylor, S., & Liberzon, I. (2002). Functional neuroanatomy of emotion: A meta-analysis of emotion activation studies in PET and fMRI. *Neuroimage, 16,* 331–348. doi:10.1006/nimg.2002.1087

Phelps, E. A., & LeDoux, J. (2005). Contributions of the amygdala to emotion processing: From animal models to human behavior. *Neuron, 48,* 175–187. doi:10.1016/j.neuron.2005.09.025

Quirk, G. J., & Beer, J. (2006). Prefrontal involvement in the regulation of emotion: Convergence of rat and human studies. *Current Opinion in Neurobiology, 16,* 723–727. doi:10.1016/j.conb.2006.07.004

Tanner, J. M., & Whitehouse, R. H. (1976). Clinical longitudinal standards for height, weight, height velocity, weight velocity, and stages of puberty. *Archives of Disease in Childhood, 51,* 170–179. doi:10.1136/adc.51.3.170

Taylor, L., & Ingram, R. E. (1999). Cognitive reactivity and depressotypic information processing in children of depressed mothers. *Journal of Abnormal Psychology, 108,* 202–210. doi:10.1037/0021-843X.108.2.202

Wechsler, D. (2003). *Wechsler Intelligence Scale for Children-IV.* San Antonio, TX: The Psychological Corporation.

Received November 17, 2010
Revision received July 14, 2011
Accepted July 22, 2011 ■